THE UNIVERSITY OF MANITOBA

A STUDY OF THE INHERITANCE OF RESISTANCE TO ERGOT (*CLAVICEPS PURPUREA*) IN TWO WHEATS: *TRITICUM DURUM* DESF. CV. CARLETON AND *T. TIMOPHEEVI* ZHUK. MANITOBA ACCESSION NUMBER 4B289

by

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A THESIS

SUBMITTED TO THE FACULTY OF GRADUATE

STUDIED IN PARTIAL FULFILLMENT OF THE REQUIREMENTS

FOR THE DEGREE OF MASTER OF SCIENCE

DEPARTMENT OF PLANT SCIENCE

WINNIPEG, MANITOBA

October, 1973

ACKNOWLEDGEMENTS

The author is indebted to Dr. L. E. Evans for supervising this project and for his assistance and guidance and many helpful suggestions.

Thanks are also extended to Dr. C. C. Bernier for his support and advice, to Mr. Richard Smith for technical help, and to the staff and students in the Rosner Laboratory whose encouragement and assistance was invaluable in carrying out this project.

Financial assistance from the Department of Plant Science is also gratefully acknowledged.

ABSTRACT

An F_1 monosomic analysis was carried out on the A and B genome monosomics of Chinese Spring crossed with Triticum timopheevi, (Manitoba accession number 4B289) and T. durum desf. cv. Carleton, in an attempt to study the inheritance of the resistance of these two wheats to ergot, Claviceps purpurea.

Chromosomes 1B and 3B of Carleton were found to condition resistance to ergot. A small part of the resistance of T. timopheevi was found to be conditioned by chromosome 3B.

A major part of the T. timopheevi resistance was expressed in crosses with other tetraploids. It was thought that the suppression of the T. timopheevi resistance may be conditioned by the D genome of Chinese Spring.

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1.0 INTRODUCTION

Ergot, a disease caused by Claviceps purpurea (Fr) Tul, has been found on most native grasses and on all cereal crops in Canada (Seaman 1971). Seeds of the infected hosts are replaced by hard black bodies called sclerotia. Although ergot is best known as a disease of rye (Secale cereale), it also causes considerable economic loss in durum wheat, Triticum durum and spring wheat, Triticum aestivum.

The sclerotia of Claviceps contain toxic alkaloids which have caused epidemics throughout history. Rapid consumption of grain infected with ergot causes convulsion of smooth muscles which may result in paralysis and death. Slow continued consumption of these alkaloids causes constriction of blood vessels, resulting in gangrene. The epidemics were known in the middle ages as St. Anthony's fire and have caused thousands of deaths (Barger 1931). In animals, similar symptoms may occur which have been confused with hoof and mouth disease (Kingsbury 1964). Continuous feeding of grain with 0.07% ergot contamination has increased abortion rates and reduced growth rates in pigs and rats (Ingliss and Phillips 1971, Campbell and Burfening 1972).

Ergot in wheat has been a recurrent problem in Western Canada. Conners (1953) found ergot in 7.0% of wheat fields surveyed in Manitoba, 10.4% in Saskatchewan and 13.3% in Alberta. Current data on carlots of wheat moving to terminal ports which were downgraded or rejected on account of ergot were kindly supplied by Mr. S. Safneck of the Inspection

Division of the Winnipeg Grain Exchange. During the 22 month period from March 1970 to December 1971, a total of 124 carlots of wheat were rejected (Figure 1). However in the 11 month period from February 1, 1972 to January 1, 1973, 443 carlots of wheat were downgraded (Figure 2). (A carlot is downgraded if a 500 gram sample contains more than 7 pieces of ergot sclerotia. Rejection occurs if the sample is more than 0.25% ergot.) Durum wheat was more heavily infected than red spring wheat, with 285 and 158 carlots downgraded respectively. Heaviest losses occurred in Saskatchewan and in southwestern Manitoba.

Japan has recently announced that it will no longer accept imports of wheat containing more than .04% ergot. Countries in Europe have announced a similar policy (Anonymous 1972). Therefore wheat exports to these countries in the future will have to be virtually free of ergot.

Ergot is also a problem in the development of triticale and in male sterile wheat lines which are being developed in the hybrid wheat program at the University of Manitoba.

Platford and Bernier (1970) and Ratanopas (1973) have shown that some wheat cultivars and wheat relatives possess resistance to ergot. Two of the most resistant strains identified are the durum cultivar Carleton and a strain of Triticum timopheevi designated as 4B289.

The objectives of this study were to confirm the chromosomal location of the resistance of Carleton and to determine the mode of inheritance and the location of the resistance of Triticum timopheevi. The

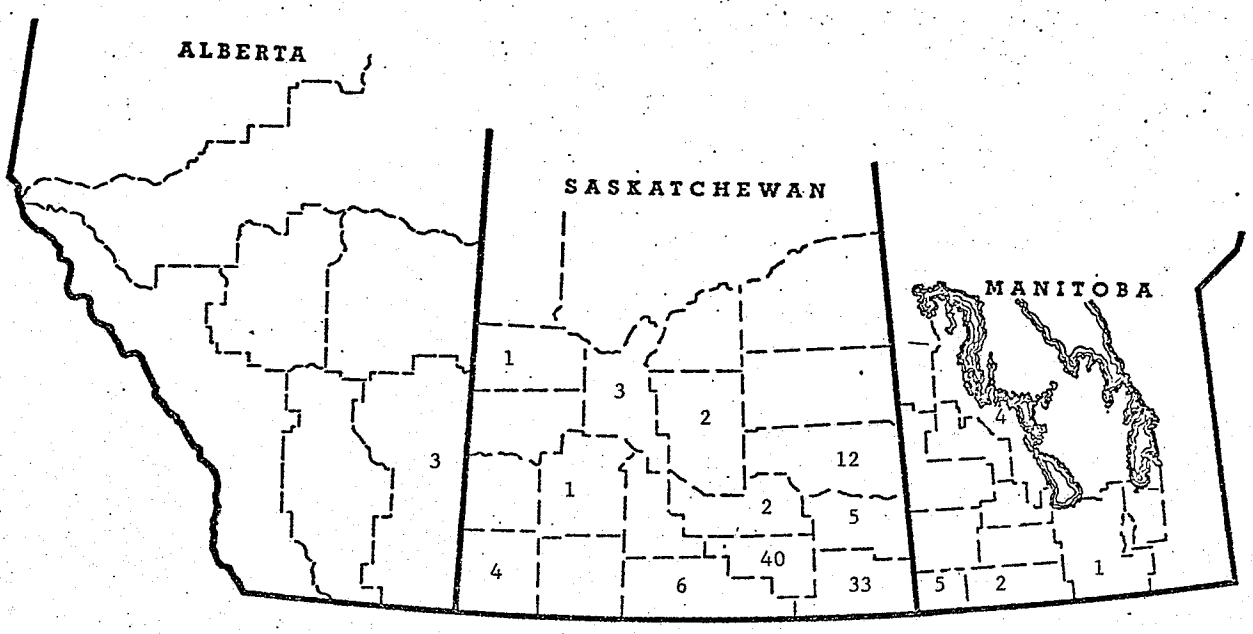


Figure 1. Carlots per Crop District of Durum and Red Spring Wheat Rejected on Account of Ergot, March 1970 to December 1971.

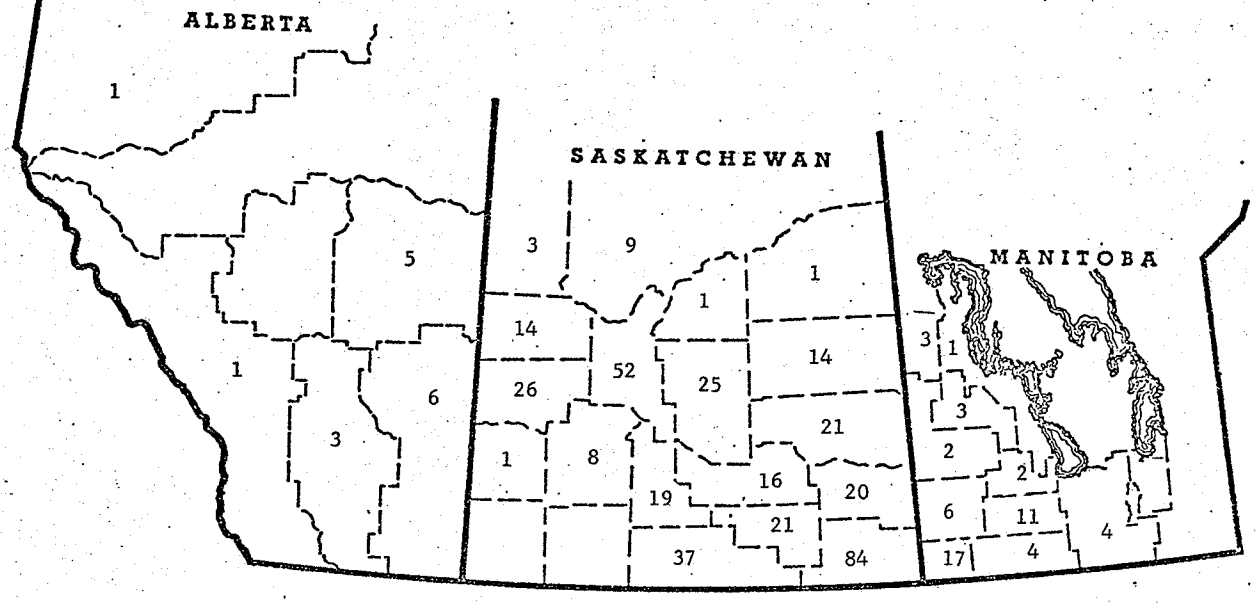


Figure 2. Carlots per Crop District of Durum and Red Spring Wheat Downgraded on Account of Ergot, Feb. 1 1972 to Jan. 1 1973.

fact that T. timopheevi has shown considerable resistance to numerous wheat pathogens prompted a comprehensive literature review of this species.

2.0 LITERATURE REVIEW

2.1 The Nature of the Pathogen

The life cycle of Claviceps purpurea has been reviewed by Dickson (1956), Heald (1933), Barger (1931) and Bove (1970). The ergot sclerotia either fall to the ground, or are planted with the grain in the spring. After a period of moist cool conditions, one to several stromata are produced. A stroma is composed of a stipe one-half to one inch long which bears a perithecium. The mature perithecium contains many long, narrow asci. Each ascus contains 8 needle-shaped ascospores which are forcibly ejected and carried by water or air currents to a floret of a susceptible host. The spore germinates in the moisture in the floral cavity within 24 hours, and penetrates the ovary wall near the base of the ovule (Kirchhoff 1929). Campbell (1958) found that the fungus grows intercellularly for two or three days, around the outer integuments of the ovary. In 4-5 days the fungus begins to grow intracellularly. It penetrates the barrier of integuments surrounding the ovule, and begins to produce conidia and exude honeydew on the surface of the ovary. Gravity and insects spread the honeydew and conidiospores to other florets. A compacted mass of large hyphae form an absorbing structure in the vascular bundles of the rachilla (Dickson 1956). As the hyphal mass advances through the ovary, it differentiates into thickened, septate hyphal filaments, which become more compact and push outwards. The production of honeydew and conidia stop. The superficial

hyphae darken and form a cortex, completing the formation of a sclerotium (Tulasne 1853, as reported in Heald, Dickson and Bove).

2.2 Host-Parasite Relationships

Claviceps purpurea is capable of parasitizing many species and genera of the Gramineae including common forage and cereal crops. However there are conflicting reports concerning host specificity. Stager (1923) believed that there were several species of Claviceps, and classified C. purpurea into three races. One race, designated P_1, was capable of infecting the cultivated cereals. Sprague (1950) suggested that seven races of C. purpurea had been found but felt that a complicated maze of races and subraces within C. purpurea probably existed.

Campbell (1957) found no indication that races of Claviceps purpurea existed that were host specific, as he was able to infect rye, wheat, and barley with all except one of 421 isolates from wild grasses. However variability in cultural colonies indicated that races in the cultural sense do exist. Campbell found that the technique used in inoculation is important, and suggested that Stager may have reported some species as being immune if he failed to observe infection.

Ratanopas (1973) found host-parasite interaction between different Claviceps isolates and different wheat varieties, which indicated that vertical resistance existed and therefore different genes for resistance were likely to exist in different wheat hosts.

2.3 Nature of Resistance to Ergot

Graminaceous hosts with tight florets, or with florets that remain open for a short time can provide resistance to infection by excluding spore entry. Abe and Kono (1957) found rye to be more susceptible than other cereals because the florets remain open longer. Campbell (1957) suggested that the morphology and development rhythm of the plant, i.e. if flowering occurs when the head is still in the boot, are of utmost importance in providing resistance to infection.

The effect that fertilization of the ovary has on conditioning resistance has been noted by several workers. In France, Rapilly (1968) noted that non-fertilized ovaries of wheat are highly susceptible. Futrell and Webster (1965) found that ergot was more likely to affect sterile heads of sorghum. They believed that the ovary becomes resistant when fertilized, and that unfertilized ovaries produce an excessive amount of flowering hormones which cause their susceptibility. Campbell (1958) found that no infection in barley occurred if inoculation was delayed until the anthers appeared, and that resistance was rapidly obtained after fertilization. Purinik and Mathre (1971) found that unfertilized male sterile barley can be infected up to 15 days after normal anthesis, while fertilized male sterile barley becomes completely resistant 10 days after fertilization.

Ratanopas (1973) found that fertilization increased resistance, which was expressed as an increase in partially infected and aborted

florets and a decrease in sclerotia and honeydew.

Resistance can also be expressed before fertilization occurs, when the ovary is most susceptible. Ratanopas (1973) carried out his inoculations two days before anthesis and found some wheat varieties more susceptible than others. Puranik and Mathre (1971) inoculated male sterile wheat with a capillary dropper into the floral cavity, and found the wheat variety Chris to be the most resistant. Chris was also most resistant when grown in the field under natural conditions of infection.

Platford and Bernier (1970) found two wheat varieties, Carleton and Kenya Farmer, which possessed a high level of resistance when inoculated two days before anthesis. Resistance was expressed by a decrease both in number of sclerotia formed and honeydew production.

2.4 Studies of the Inheritance and Transfer of Ergot Resistance

Robinson (1959) working with sugar cane in Queensland crossed a resistant cultivated variety with a susceptible, frost-resistant wild species, Sacchaum spontaneum. The F_1 plants were susceptible to a race of Claviceps purpurea called false floral smut. The wild species was found to be homozygous for susceptibility which was dominant.

Bennett and Bashaw (1960) crossed a well-adapted variety of the pasture grass Paspalum dilatatum which was susceptible to ergot (Claviceps paspali, Stevens and Hall) to an immune species, P. malacophyllum. The F_1 plants were highly resistant, indicating dominant inheritance of resistance. Highly resistant and immune plants were

obtained from an F_2 population which was segregating for ergot resistance.

Kalsdyan-Avanestan (1967) in Russia, tested the resistance of several interspecific Triticum crosses to Claviceps purpurea. He found that resistance was recessive in some F_1's but dominant in others. Dominant inheritance to resistance was found in Triticum polonicum x T. timopheevi, T. timopheevi x T. aestivum var Awnless 1, T. durum var africanum x T. timopheevi. It was concluded that ergot resistance in wheat is an unstable quantitative character controlled by more than one gene.

Ratanopas (1973) examined the resistance of the durum wheat variety Carleton and the spring wheat variety Kenya Farmer to 58 isolates of Claviceps purpurea. He found evidence that the resistance of Kenya Farmer and Carleton are genetically different and that both horizontal and vertical resistance are present.

Platford (1973) found that the resistances of the durum wheat Carleton and the hexaploid wheat Kenya Farmer were both recessive. He tested the Kenya Farmer-Chinese Spring substitution lines and the F_1's of the A and B genome monosomics of Chinese Spring by Carleton and found that some of the resistance was conditioned by chromosome 6B of Kenya Farmer and 1B of Carleton.

2.5 The Origins of Triticum Timopheevi

Triticum timopheevi Zhuk. is a cultivated tetraploid wheat found in parts of Soviet Georgia. Crosses with other tetraploid wheats produce a sterile F_1 even when chromosome pairing is almost perfect. This

has sparked interest in the origin of T. timopheevi. Lilienfeld and Kihara (1934) suggested that T. timopheevi had a different progenitor to the other tetraploids (AABB), and designated the T. timopheevi genomes as AAGG. However Kostoff (1941) believed that the T. timopheevi differs only in degree of divergence and renamed the T. timopheevi complement as AAββ . Sachs (1953) suggested that cryptic structural hybridity, i.e. many very small non-homologous chromosomal segments could account for the sterility of F_1 hybrids even when pairing was good. Wagenaar (1961, 1966) proposed an asynaptic genetic system, which could account for the poor pairing of T. timopheevi hybrids, which arose in the original tetraploid population and separated the Timopheevi group by a sterility barrier. Any structural changes would have arisen following the formation of this genetic barrier. Feldman (1966a) examined heteromorphic associations in the F_1 between Chinese Spring ditelocentrics and T. timopheevi. He suggested that five major translocations had occurred in the course of divergence between the timopheevi complex and the emmers. He proposed that the A genome was a stable "pivotal genome" and hybridization between the B genome and other diploid species may have occurred. Bozzini and Giorgi (1969) showed that the karyotypes of the T. timopheevi group and the Emmer group belong to two distinct groups. Electrophoresis of seed proteins of the two groups show strong differences at four or five loci (Johnson 1967). Most of these differences were attributed to the G(B) genome. Feldman (1966b) found the

5B diploidization mechanism to be expressed in T. timopheevi suggesting that this mechanism may have arisen in a common progenitor.

It is proposed (Zakubziner 1958, Harlan and Zohary 1966) that the T. timopheevi group arose from a non-weedy, sub-dominant group of wild wheats centred in Turkey, Iran and Iraq, while the cultivated emmers arose from a weedy, agressive race in Jordan and Palestine. Johnson (1967) suggested that T. dicoccoides may have been the wild progenitor of both groups.

2.6 Behavior of Triticum Timopheevi in Crosses

The F_1 hybrids of T. timopheevi by all other Triticum species have been found to be highly sterile (Sachs 1953) (Bell & Lupton 1955) (Allard 1949), including Triticum araracium and T. dicoccoides, which pair well in crosses with T. timopheevi (13-14 bivalents at meiosis). F_1 seed set between 35% (Fedesenko 1970) and 75% (Allard (1949) has been obtained when T. timopheevi was used as the male parent in crosses with Chinese Spring. However, when T. timopheevi is used as the female, shrivelled F_1 seed with poor germination are produced (Khodyrev 1969, Allard 1949). F_1 plants are somewhat female fertile and viable seed set of 2.2% has been reported (Allard 1949) when backcrossing the F_1 to the vulgare parent.

The pairing in the F_1 of a durum or vulgare by T. timopheevi cross is variable, with the bivalent associations ranging between 4 and 14 pairs and averaging about 9-10 bivalent and 1 trivalent association per

cell during meiosis (Sachs 1953, Love 1941, Wagenaar 1961, Allard 1949). The F_1 gametes of a vulgare by T. timopheevi cross contained 15 to 21 chromosomes (Allard 1949). In the BC_1F_1 and further backcross generations, increased pairing was observed, accompanied by greater fertility and return to the chromosome number of the recurrent parent (Allard 1949). A seed set of 56% was found after the third backcross (Allard 1949).

In crosses with T. monococcum (AA) and T. timopheevi, between 5 and 7 bivalents were found at meiosis, (Sachs 1953, Wagnaar 1961) indicating that the A genomes of T. timopheevi and the other triticums are fairly homologous.

Fedesenko (1969) found the poor fertility in vulgare x T. timopheevi hybrids manifested in low pollen germination, abnormal pollen tube growth and abnormal mitosis in the endosperm after fertilization occurred.

Meiotic instability may persist even after a return to the parental chromosome number. Semeniuk (1947) examined the F_5 and F_6 lines of Pridham's Steinweidel by T. timopheevi cross and found 4.8 to 53.8% of the cells had univalents. Chromosome instability could be recognized by abnormalities at anaphase I and II, micronuclei at interphase, and presence of aborted pollen.

2.7 Triticum timopheevi as a Source of Disease Resistance

Triticum timopheevi has been found to possess immunity or resistance to a wide range of wheat pathogens. These include P. graminis,

P. recondita, P. striiformis, Tilletia levis, T. tritici, Erysiphe graminis tritici, Ustilago tritici and Fusarium and Septoria species (Jacubziner 1958, McIntosh and Gyfaras 1971, Allard and Shands 1954).

Another indication of the wide resistance of T. timopheevi can be found in its reaction to diseases in the International Spring Wheat Rust Nursery which tests 600 to 900 accessions each year in 20 to 30 countries. An accession of Triticum timopheevi, D-357-1 from Russia has consistently been one of the most resistant entries. In the past 10 years its resistance has only been overcome by races of Puccinia graminis and P. recondita in Brazil, Bolivia, Peru, Chile, and in South Africa and it has been attacked by septoria in South Africa. Its resistance to Erysiphe graminis and Helminthosporium has apparently remained intact.

Jacubziner (1958) reported that this "complex resistance" of T. timopheevi is effective against most physiologic races of these diseases, and also is a stable resistance, maintaining resistance in the field to brown rust (P. recondita) and yellow rust (P. striiformis) for over 50 years.

The reactions to the races of diseases to which T. timopheevi had been found to be tested is summarized in Table 1.

2.8 Disease Resistant Timopheevi Derivatives

Shands (1941) produced a line of winter wheat which he reported as resistant to leaf rust, stem rust and mildew. Selections were made from

Table 1. Resistance of Triticum timopheevi to Plant Diseases.

Resistance to Stem Rust, *Puccinia graminis*

Race	9	10	11	15	15B	15B-IL	17	19	21	29	32	34	36	38	39	40	48	50	56	100	113	116	120	125	139	152	189	198	Author	Source
Reaction	I	I	I	R	MS		I	MR	R	I	I	I	R	R	I		R	I	MR R		I		I		I		R	Newton et al. (1940)	
				R			S		R			R				R				R		R		R				Bell & Lupton (1955)	
			MS	R	S.		S			MR																		Jacubziner (1969)	
						R																					S	Watson & Stewart (1956)	CI 11802
																												Garcia-Rada et al. (1942)	
																												McIntosh & Gyarfas (1971)	CI 11802

Resistance to Leaf Rust, *P. recondita*

Race	1	9	11	20	21	26	27	28	29	52	76	77	95	116	122	135	138	174	185	Author	Source
Reaction	I	I	I	I	I	R	I	I	I	I	I	I	R	I	I	R	R	I	I	Newton et al. (1940)	
																				Zacubziner (1961)	
																				Watson & Stewart (1956)	CI 11802

Resistance to Stripe Rust, *P. striiformis*

Race	2	2B	5	7	8
Reaction	R	R	R	S	S

Resistance to Powdery Mildew, *Erysiphe graminis*

3	4	5	9	10	12	13	15	18	19	Author
	I									Bell & Lupton (1955)
I		R	I	I	I	I	I	I	I	Jacubziner (1959)
										Allard & Shands (1954)

Resistance to Loose Smut, *Ustilago nuda*

Race	2	4	5	8	9	10	11	12	13	14	15
Reaction	I	I	I	I	I	I	I	I	I	I	I

Resistance to Bunt, *Tellitia tritici*

13	15	16	Author
I	I	I	Jacubziner (1969)

Reaction types:

I	Immune
R	Resistant
MR	Moderately resistant
MS	Moderately susceptible
S	Susceptible

the progeny of a single BC_1F_2 seed.

Allard and Shands (1954) produced two cytogenetically stable spring wheat lines CI 12632 and CI 12633 from a cross between a Chinese Spring by Illinois line and T. timopheevi. The two lines were selected following two backcrosses and eight generations of selfing. The lines were highly resistant to powdery mildew (Erysiphe graminis tritici) and most races of stem rust except 15B.

Moderate resistance to loose smut (Ustilago), and resistance to some races of leaf rust were also transferred, but the bunt (Tilletia) resistance was not transferred. Both the stem rust and post seedling mildew resistance (to races of mildew) were thought to be conditioned by a single dominant gene on chromosome 2B as determined by monosomic analysis (Nyquist 1957 and 1962). Seedling mildew resistance was conditioned by another independent gene (Nyquist 1963).

Pridham in Australia crossed the spring wheat variety Steinweidel with T. timopheevi which resulted in the variety Timvera. Timvera was found to possess similar stem rust resistance to that of CI 12633 and CI 12632. However not all the T. timopheevi rust resistance was transferred (Watson & Luig 1958).

Watson and Stewart (1956) found that the leaf rust resistance to four races (26, 95, 135, 138) was transferred in the Steinweidel cross, but not in the Illinois by Chinese Spring cross.

An Australian T. timopheevi derivative called CI 13005 from a cross

between a hexaploid variety Cheyenne by Shands 473 (T. timopheevi) was found to give moderate resistance to race 15B in seedling and adult stages (Atkins 1967 as reported by McIntosh and Gyarfas 1971).

Line W (W 3563) is a Steinweidel T. timopheevi reselection which is reported to be resistant to all races of stem rust in Australia (McIntosh and Gyarfas 1971).

The incorporation of mature plant stem rust resistance derived from T. timopheevi into winter wheat has been reported from the Soviet Union (Skurygina 1970).

McIntosh and Gyarfas (1971) compared the stem rust reactions of three derivatives and several accessions of T. timopheevi. By using selected North American and Australian races, they were able to differentiate a number of genes or factors for stem rust resistance. Gene SrTt 1 is present in CI 12632, Timvera and CI 13005. Gene SrTt 2 is present in line W (W3563). A third factor is present in CI 13005 which confers moderate resistance against the new subraces of the 15B complex. The nineteen T. timopheevi accessions were placed into four different reaction classes. T. araraticum was placed in a fifth class. It is believed that a great amount of resistance for stem rust which exists in T. timopheevi still remains to be exploited.

3.0 MATERIALS AND METHODS

3.1 Monosomic Analysis - Plant Materials

The materials utilized in this study included:

(I) The hexaploid Triticum aestivum L. variety Chinese Spring, and the fourteen monosomic lines of the A and B genomes of this variety. Chinese Spring has been found to be susceptible to ergot when tested at the University of Manitoba (Platford, 1973).

(II) T. durum desf. variety Carleton is a tetraploid produced from a Vernal Emmer by Mindum cross, and has been found to possess resistance to ergot (Platford & Bernier 1970).

(III) T. durum desf. variety Stewart 63 has the same parentage as Carleton, but is more susceptible to ergot (Platford 1973).

(IV) T. timopheevi Zhuk. variety Nigrum Manitoba accession number 4B289 is a tetraploid wheat obtained from Zhukovsky in Russia. This accession has maintained a high level of resistance in greenhouse and field screening (Bernier 1973).

3.2 Handling the Parents and F_1 Generation:

Root tips from ten germinated seeds of each of the fourteen Chinese Spring monosomic lines of the A and B genomes were collected and fixed. The chromosome number for each plant was determined at mitotic metaphase using the Feulgen technique for staining the chromosomes. The monosomic seedlings were grown in the growth cabinet along with T. timopheevi and

Carleton plants. In order to confirm the monosomic condition of each Chinese Spring line, the developing head of one tiller from each plant was removed and the metaphase configuration of the pollen mother cells was examined at the first division of meiosis. Twenty chromosome bivalents and one univalent was positive confirmation of the monosomic condition. The heads of two monosomic plants from each monosomic line were emasculated and crossed both with Carleton and T. timopheevi, making twenty eight different crosses. Five to ten crossed seeds were obtained from each line. It was necessary to grow and cross some of the parents a second time in order to obtain sufficient crossed seed of all lines.

The F_1 seed was germinated and the chromosome numbers were again determined at mitotic metaphase. Counts of 34 (monosomic pentaploid) and 35 (pentaploid) chromosomes were obtained for each of the crossed lines. Most of the lines were grown in the growth cabinet at 70-75^{o}F with 18 hours of light per day. Because of lack of space, three lines (Chinese Spring 1A x T. timopheevi, Chinese Spring 2B x T. timopheevi and Chinese Spring 1A x Carleton) were grown in the greenhouse August to October 1972 with natural day light supplemented with inflorescent lighting. The greenhouse temperature ranged from 65^{o}F to 85^{o}F. The 34 and 35 chromosome plants in all lines were grown adjacent to each other. Stewart 63 and Chinese Spring were included as the susceptible checks. Carleton and T. timopheevi were the resistant checks.

3.3 Inoculation:

The inoculum for this experiment originated from an isolate of Claviceps purpurea on Manitou wheat and is designated M-4. A conidiospore suspension of this isolate was maintained by the Plant Pathology Section. The inoculum was made up every second day by diluting a portion of the conidiospore suspension with distilled water to a spore density of 10^4 conidiospore per cc.

Heads were inoculated two days before anthesis according to the method outlined by Platford (1973) when the stigma on a primary floret near the centre of the head was beginning to appear feathery. The outer glumes of ten florets were removed and these florets were inoculated by inserting a hypodermic syringe through the lemma, without touching the pistil, and filling the floral cavity with approximately .02 ml. of conidial suspension. After inoculation, each head was covered with a glassine bag and fastened securely to maintain high humidity around the head. At least six heads of each monosomic line and each check variety were inoculated, except Chinese Spring 1A x Carleton, Chinese Spring 2B x Carleton and Chinese Spring 1A x T. timopheevi for which five heads were inoculated, and Chinese Spring 2A x Carleton which had 4 heads inoculated.

3.4 Rating the Infection:

The rating system was based on methods developed previously by Platford and Bernier (1970), and by Ratanopas (1973).

The honeydew produced by each inoculated head was rated visually two weeks after inoculation as follows:

1. no visible honeydew.
2. honeydew confined to glumes.
3. honeydew exuding outside glumes in small drops.
4. large drops of honeydew running down the head.

Four weeks after inoculation, the ten inoculated florets were examined. The numbers of florets were recorded which fell into each of the following reaction classes:

A sclerotium - was judged to have been produced when the ovary had been completely engulfed by mycelium.

A partial infection - occurred when the infection stopped before engulfing the entire ovary.

An abortion - occurred when no infection or seed was found in a floret.

Seed set - was the occurrence of a normal seed in the inoculated floret.

The sclerotia were subdivided into the numbers which fell into each of the following size classes:

1. the number of sclerotia smaller than a normal seed.
2. the number equal in size to a normal seed.
3. the number larger than a normal seed.

An index system was developed by Ratanopas (1973) in order to classify and quantify the overall reaction of an inoculated head. This index is based on three components, honeydew production, size of sclerotia, and number of sclerotia. The index rating system which was used in this study is presented in Table 2. The most susceptible component had the strongest influence in determining the index rating. In order to assure an unbiased rating of the reaction, the chromosome numbers were not assigned to the plants until after the rating had been carried out.

A sclerotia size index from 0 to 100 was calculated from the number of sclerotia in each of the 3 size classes by using the following formula:

$$\frac{\sum_{i=1}^{3} N_i \times R_i}{T \times 3}$$

where i = the size class

N_i = number of sclerotia in the ith size class

R_i = size class multiplier 1, 2, 3

T = total number of sclerotia on a head

3.5 Analysis of Results:

The reaction of the 34 chromosome F_1 heads were compared with the 35 chromosome plants which had arisen from the same Chinese Spring monosomic line. Therefore the plants used in each comparison were identical

Table 2. Rating System for Disease Caused by Claviceps purpurea.

(for ten inoculated florets on a head)

Reaction Index	Quantitative Index	Infection Type +	Frequency of Sclerotia	Honeydew Reaction
Immune	0	No infection	0	1
Very Resistant	1	AR	0	1
Resistant	2	AR + S_1 + S_2	4 or less	1-2
Moderately Resistant	3	Mainly S_1 and S_2 a few S_3	6 or less	1-2
Moderately Susceptible	4	S_2 and S_3	8 or less	3
Susceptible	5	Mainly S_3	more than 8	4

+ Infection types.

AR - Abortive Reaction = Abortions + partial infections

S_1 - Sclerotium smaller than seed

S_2 - Sclerotium equal in size to a seed

S_3 - Sclerotium larger than seed.

except for the absence of one chromosome.

The Mann-Whitney test was carried out on each set of comparisons. The index of each head in a comparison was ranked, and the ranked values of the 34 and 35 chromosome heads were compared. This test does not assume that the data is normally distributed. Similar tests were also carried out on the three components of the index.

3.6 Cytological Analysis:

The pairing relationships of at least 10 pollen mother cells from 2 or more F_1 plants were scored at F_1 meiotic metaphase for each of the following crosses: Chinese Spring x T. timopheevi; Chinese Spring x Carleton; Carleton x T. timopheevi; and Chinese Spring^2 x T. timopheevi.

3.7 Supplementary Crosses:

F_1 plants of the following crosses were tested for resistance to the M-4 isolate of ergot: Carleton x T. timopheevi; Stewart 63 x T. timopheevi; T. timopheevi x rye (Secale cereale L. accession OD 174); two plants of Chinese Spring^2 x T. timopheevi. Two triticale lines, 6A20 (a Carleton x rye cross) and 6A190 (Stewart x Prolific rye) were also tested. Between 6 and 20 heads were tested for each cross, except the Chinese Spring x T. timopheevi^2 cross for which 2 heads were tested. The viability of the inoculum was checked by inoculating a susceptible check variety on each date when inoculations were carried out.

4.0 RESULTS AND DISCUSSION

4.1 Monosomic Analysis - The F_1 Hybrids

Both the Chinese Spring x Carleton and Chinese Spring x T. timopheevi F_1 hybrids with 34 and 35 chromosomes were vigorous and tillered well in the growth cabinet. The 35 chromosome Carleton crosses were semi-sterile, and some 34 chromosome lines were completely sterile. All the T. timopheevi crosses were completely sterile. In both crosses, the 34 chromosome lines, 4B and 6B could be identified by their intermediate awn expression, and the 5A lines could be identified by the expression of the speltoid character (Plate 1 and 2).

4.2 Disease Reaction of Chinese Spring x Carleton Hybrids

The Chinese Spring parent was found to be susceptible, producing a large sclerotium in most inoculated florets and abundant honeydew. The resistance of Carleton was expressed by a reduction of sclerotia size and number, and a reduction of honeydew production. The Carleton reaction is shown in Plate 1 and in Table 3. The F_1 pentaploids of Chinese Spring x Carleton were as susceptible as the Chinese Spring parent.

Table 3 includes only those comparisons where a significant reduction at the 1% or 5% level was found in the disease index or in the disease components. (The complete table is presented as Appendix 1.) The 34 chromosome lines 1B and 3B were found to have a significantly lower index reaction at the 1% level when compared to their 35 chromosome counterparts. Line 1B appeared to express a greater reduction in susceptibility

Plate I. Typical reactions of 34 and 35 chromosome plants of Chinese Spring x Carleton F_1 hybrids, and their parents.

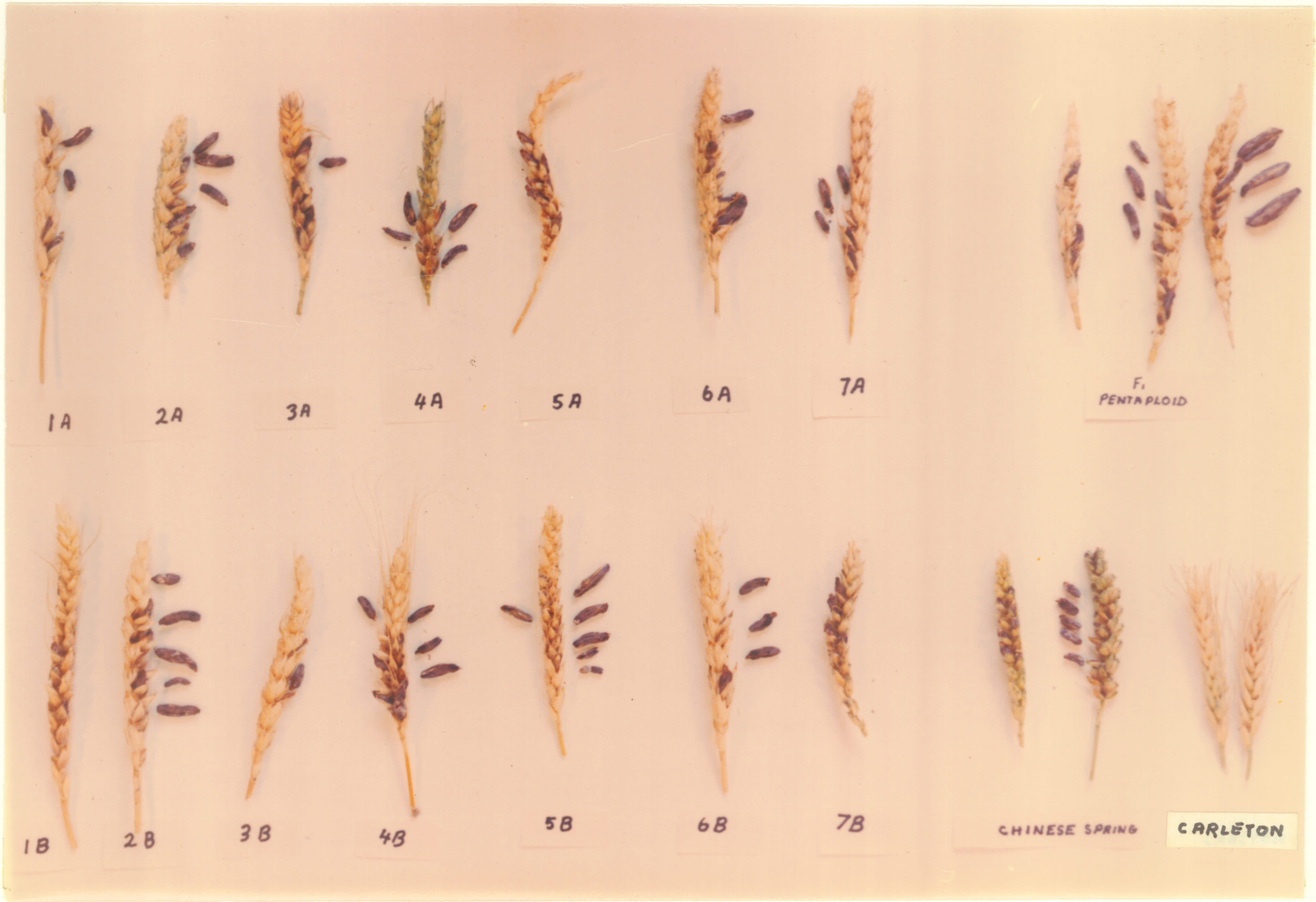

1A
2A
3A
4A
5A
6A
7A
F1
PENTAPLOID
1B
2B
3B
4B
5B
6B
7B
CHINESE SPRING
CARLETON

Table 3. Selected Comparisons of Average Disease Index and Disease Components of 34 and 35 Chromosome Plants[+] of Chinese Spring x Carleton.

Monosomic Line	Chromosome Number	Average Disease Index Rating	Sclerotia Number	Sclerotia Size Index	Honeydew Rating
6A	34	4.7	7.6	87	3.2
	35	4.7 NS	7.4 NS	92 NS	3.9 *
1B	34	2.9	4.8	60	2.3
	35	4.5 **	7.2 *	89 **	3.2 **
3B	34	3.8	1.5	78	2.9
	35	4.8 **	4.1 *	95 *	3.4 NS
Carleton	28	1.5 (Range 1-3)	0.75 (Range 1-3)	16 (Range 0-33)	1.3 (Range 1-3)
Chinese Spring	42	4.9 (Range 4-5)	7.8 (Range 3-10)	88 (Range 80-100)	3.6 (Range 3-4)

* Significant difference between comparisons at 5% level.

** Significant difference between comparisons at 1% level.

\+ Between 6 and 12 heads per line are compared.

than did line 3B. However, in neither line was the full resistance of the Carleton parent recovered. Both critical lines were capable of producing a sclerotium larger than a seed, and slight extrusion of honeydew from the florets.

The reduction in susceptibility of line 1B and 3B indicates that chromosome 1B and 3B of Chinese Spring possess genes which when crossed to Carleton, suppress the expression of resistance to ergot. Carleton and Chinese Spring are fairly homologous, and it is possible that resistance occurs in chromosomes 1B and 3B of Carleton as alleles of the dominant genes in Chinese Spring which were found to suppress the Carleton resistance. Alternately, the Carleton resistance may be located on a different chromosome, which is suppressed by the Chinese Spring genes on chromosomes 1B and 3B.

A comparison of each of the 3 disease components indicated that a reduction in sclerotia size, sclerotia number and honeydew production had all contributed to the reduction of the disease index of line 1B. The reduction in the disease index of line 3B was produced by a reduction of sclerotia size and number, but no significant reduction occurred in honeydew production. However, line 6A was found to produce a significant reduction in honeydew at the 5% level, but no significant reduction in the other 2 components. Assuming that recessive genes for ergot resistance occur on chromosome 1B, 3B and possibly 6A of Carleton, the data may indicate that the gene on 1B conditions resistance by reducing all

Plate II. Typical reactions of 34 and 35 chromosome plants of Chinese Spring x *T. timopheevi* F_1 hybrids, and their parents.


1A
2A
3A
4A
5A
6A
7A
F1
PENTAPLOID
1B
2B
3B
4B
5B
6B
7B
CHINESE SPRING
T. TIMOPHEEVI
48289

3 components of the ergot reaction; the gene on 3B reduces sclerotia size and number, and the gene on 6A seems to reduce honeydew production only. Therefore it is possible that different genes in Carleton condition resistance to different components of the ergot reaction. This supports the suggestion by Ratanopas (1973) that honeydew production and sclerotia production are not conditioned by the same genes in Carleton.

4.3 Disease Reaction of Chinese Spring x T. timopheevi Hybrids

T. timopheevi was found to possess a very consistent high resistance. No honeydew or sclerotia were produced. Infection was in all cases limited to a partial infection or an aborted reaction. Inoculated florets often exhibited immunity by producing seeds. The F_1 pentaploids were slightly less susceptible than the Chinese Spring parent, with less honeydew production and somewhat fewer sclerotia; however the reaction was much more susceptible than that of the T. timopheevi parent. Ergot resistance is therefore apparently inherited as a recessive character in F_1 crosses between Chinese Spring and T. timopheevi.

In no 34 chromosome line was the resistance of T. timopheevi recovered. Each 34 chromosome line was as susceptible or almost as susceptible as its 35 chromosome counterpart (Plate 2, Table 4, and Appendix II. However the disease index rating of line 3B was significantly lower (at the 5% probability level) than its 35 chromosome counterpart.

An equal but non-significant reduction occurred in the disease index comparison of the 1B line. Therefore, there is some indication that

Table 4. Significant Comparisons of Averaged Disease Index and Disease Components of Heads of 34 and 35 Chromosome Heads[+] of Chinese Spring x *T. timopheevi* F_1 Hybrids.

Monosomic Line	Chromosome Number	Disease Index Rating	Sclerotia Number	Sclerotia Size Index	Honeydew Rating
2A	34	4.0	4.9	88	2.4
	35	4.5 NS	6.5 NS	78 NS	3.3 **
1B	34	3.8	5.7	69	2.5
	35	4.6 NS	6.5 NS	81 NS	3.2 *
3B	34	3.8	6.8	58	2.6
	35	4.6 *	7.5 NS	83 *	2.8 NS
4B	34	4.2	4.8	86	3.2
	35	4.3 NS	7.7 **	81 NS	2.8 NS
7B	34	4.2	4.8	80	2.4
	35	4.1 NS	4.0 NS	87 NS	2.9 *
Chinese Spring	42	4.9 (Range 4-5)	7.8 (Range 3-10)	88 (Range 88-100)	3.6 (Range 3-4)
T. timopheevi	28	1 (Range 1)	0 (Range 0)	0 (Range 0)	1 (Range 1)

* Significant difference between comparisons at 5% level.

** Significant difference between comparisons at 1% level.

\+ Between 6 and 17 heads per line are compare .

genes for slight suppression of T. timopheevi resistance occur on chromosome 1B and possibly 3B of Chinese Spring. Reduction in one of the disease components, but not in the overall disease index was found in lines 2A, 4B and 7B. Genes may occur in these Chinese Spring chromosomes which suppress a slight expression of resistance in T. timopheevi. In no 34 chromosome line was a reduction in both disease index and all 3 disease components found. Because Chinese Spring and T. timopheevi chromosomes are not necessarily homologous, it cannot be assumed that a recessive gene for resistance in T. timopheevi exists as an allele to a Chinese Spring gene which is preventing the expression of resistance.

The identity of each of the monosomic lines has not yet been verified, although each line has been crossed to the ditelocentric of the chromosome in question. Therefore the possibility of univalent shift cannot be ruled out, and one or more of the A or B monosomic lines may not have been tested. Until the identity of all lines have been confirmed, there is the possibility that one or more of the A or B genome chromosomes of Chinese Spring could mask a major part of the T. timopheevi resistance.

4.4 Cytological Behavior of Hybrids

The chromosomes of the A and B genomes of Chinese Spring pair well with the chromosomes of Carleton at meiotic metaphase (Table 5 and Plate 3, Figure 1) which provides an indication of homology between the chromosome complements of the two species.

Plate III. Meiotic metaphase configurations.

Figure 1. Chinese Spring x Carleton F_1.

Figure 2. Chinese Spring x T. timopheevi F_1. Chromosomes showing good pairing.

Figure 3. Chinese Spring x T. timopheevi F_1. Chromosomes showing poor pairing.

PLATE 3

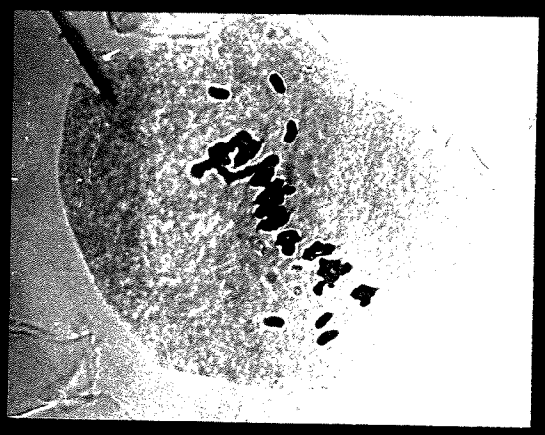


Figure 1

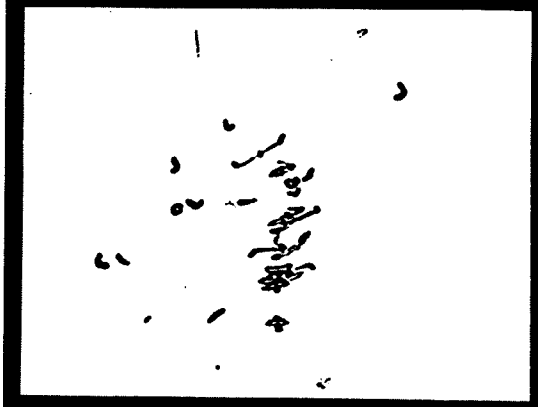


Figure 2

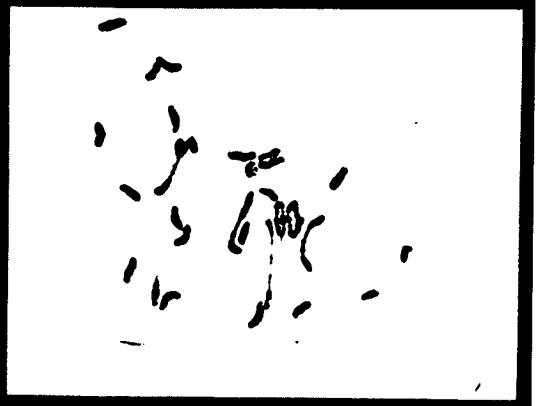


Figure 3

Table 5. A Comparison of the Pairing Behavior in F_1 Hybrids and Backcrosses.

Cross	Cytological Character (Numbers per cell)					
	Bivalents		Univalents		Trivalents	
	Avg.*	Range	Avg.	Range	Avg.	Range
Chinese Spring x *T. timopheevi* F_1	9.83	6-14	13.66	7-21	0.7	0-2
Chinese Spring x Carleton	13.5	12-14	7.7	6-11	0.12	0-1
Carleton x *T. timopheevi*	10.66	8-12	5.6	4-8	0.2	0-1
Chinese Spring^2 x *T. timopheevi* F_1	13.46	11-15	10.4	7-11	0.3	0-1

* Average of 10 or more cells from 2 or more plants.

However the pairing of T. timopheevi in F_1 crosses with Chinese Spring is variable (Table 5 and Plate 3, Figure 2 and Figure 3). The bivalents observed range from 6 to 14, with many univalents apparently arising through desynapsis (Plate 3, Figure 3). The pairing of Carleton x T. timopheevi was almost as uncertain, with a bivalent range of 8-12 in the cells counted. This seems to support Feldman's (1968) suggestion that T. timopheevi contains several chromosome segments which are not homologous to the A and B chromosomes in Chinese Spring and Carleton.

The large number of univalents (7-11) observed in a 38 chromosome plant of $(\text{Chinese Spring})^2$ x T. timopheevi indicates continued irregular meiosis after the F_1 generation.

4.5 Supplementary Crosses

Table 6 and Plate 4 present the reactions of the F_1 of the supplementary crosses and the reactions of their parents. Stewart 63 (Table 6 and Plate 4, Figure 4) was found to be susceptible, producing large narrow sclerotia and large amounts of honeydew. The F_1 of Stewart 63 x T. timopheevi (Plate 4, Figure 2) was classed as resistant. Few small sclerotia were produced, and any honeydew production was confined within the florets.

The F_1 of Carleton x T. timopheevi (Plate 4, Figure 1) was found to be very resistant, and equal to the resistance of the T. timopheevi parent.

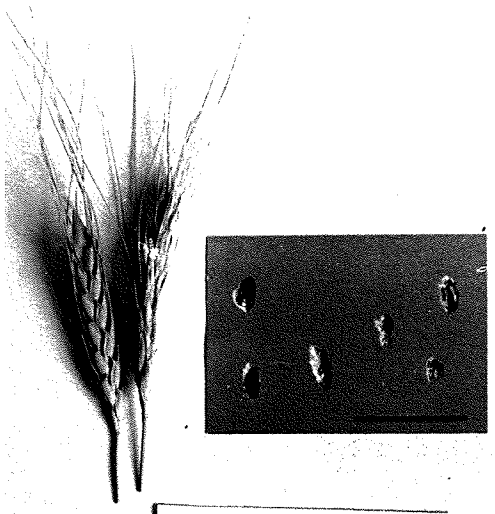
Table 6. Summary of Reactions of Parents and F_1 Crosses to Ergot.

Parent or Cross	Genome Constitution	Heads Rated	Disease Range	Sclerotia Number Range	Sclerotia Size (index) Range	Honeydew Rating Range	Reaction Type
Chinese Spring	AABBDD	10	4-5	3-10	80-100	3-4	Susceptible
Stewart 63	AABB	10	4-5	3-5	73-100	3-4	Susceptible
Carleton	AABB	10	1-3	0-1	0-33	1-3	Resistant
T. timopheevi	AAGG	15	1	0	0	1	Very Resistant
Carleton x Chinese Spring	AABBD	50	3-5	1-10	53-100	2-4	Susceptible
T. timopheevi x Chinese Spring	AABGD	50	2-5	1-10	50-100	2-4	Moderately Susceptible
Stewart 63 x *T. timopheevi*	AABG	10	1-2	0-2	0-33	1-2	Resistant
Carleton x *T. timopheevi*	AABG	25	1	0	0	1	Very Resistant

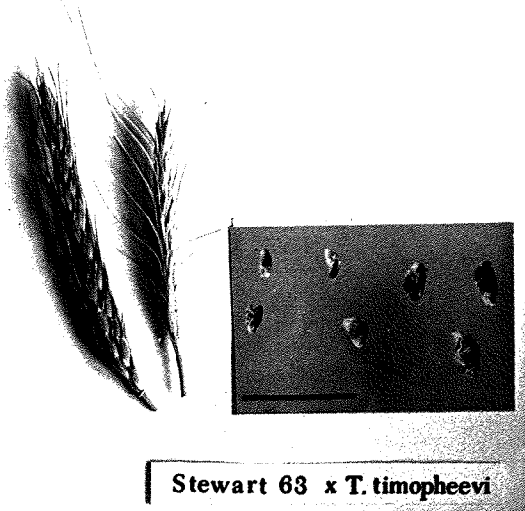
Plate IV. Resistant F_1 and Parental Reactions.

Scale of insets:

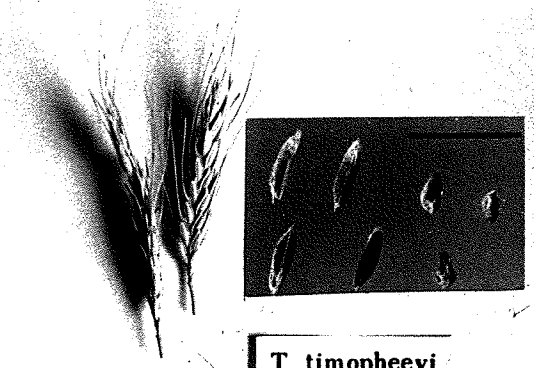
—— represents 1 cm.



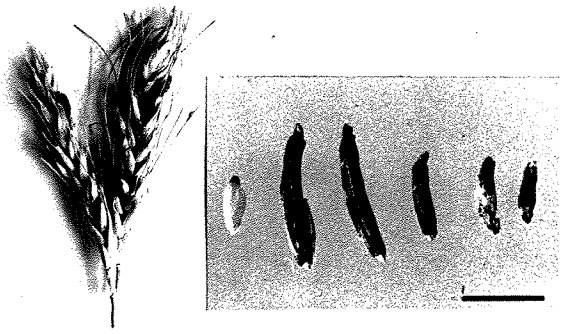
Carleton x T. timopheevi



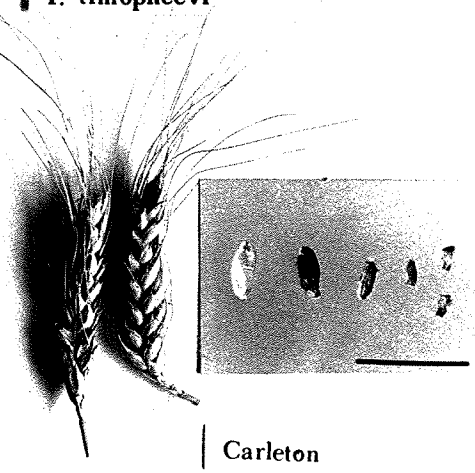
Stewart 63 x T. timopheevi



T. timopheevi



Stewart 63



Carleton

These reactions indicate that the resistance of T. timopheevi is inherited as a dominant character when crossed with the two tetraploids Carleton and Stewart 63, but as a recessive character in the hexaploid Chinese Spring. This can be explained if the hexaploid complement in Chinese Spring contains a gene or genes which suppresses a major part of the resistance of T. timopheevi. The failure of any line in the monosomic analysis to recover a major part of the T. timopheevi resistance indicates that the suppressor is not located on the A or B genomes of Chinese Spring. This suppressor gene(s), therefore is likely to be located on the D genome of Chinese Spring.

In order to attempt to confirm the existance of a repressor gene on the D genome of Chinese Spring, the D genome monosomics have been crossed with T. timopheevi. The reactions of the F_1 34 and 35 chromosome plants will be observed for the recovery of a major part of the T. timopheevi resistance.

The influence of another genome on the expression of T. timopheevi resistance was examined by crossing T. timopheevi with a susceptible rye (accession OD 271). Three F_1 triploid plants (AGR) were obtained following embryo culture. One plant was found to be susceptible. The other two plants, which had 3 heads per plant, inoculated, were found to be resistant with a disease index of 1-2. Resistance was expressed as a large number of partial infections, and one small sclerotium on

one head. This one head also produced honeydew confined within the florets. The variability of this reaction was mainly between plants and not within heads on the same plant. This suggests that the rye pollen was heterozygous for a suppressor which prevents the expression of the T. timopheevi resistance.

The resistant and susceptible plants are now undergoing colchicine treatment to produce amphidiploids. The reactions of the amphidiploids will be tested to determine if resistance is maintained when the chromosome complements are doubled.

No expression of resistance was found in the hexaploid triticale 6A20, derived from a cross between Carleton and rye, indicating that the Carleton resistance is not expressed in the presence of the particular rye genome in 6A20.

Attempts were made to obtain reciprocal backcross populations from the Chinese Spring x T. timopheevi cross, in order to determine the existance of mendelian segregation for resistance. However only 2 $(\text{Chinese Spring})^2$ x T. timopheevi plants of 38 and 40 chromosomes were obtained. Both of these were susceptible. One Chinese Spring x $(\text{T. timopheevi})^2$ plant was obtained which was very resistant. If a full scale backcrossing program were to be undertaken, the variability of chromosome pairing observed in Table 5 in the F_1 and BC_1F_1 generations of Chinese Spring x T. timopheevi would produce gametes with indefinite chromosome complements and would make the interpretation of any observed segregation difficult.

5.0 GENERAL DISCUSSION AND CONCLUSIONS

Carleton resistance is inherited principally as a recessive character when crossed either to hexaploid Chinese Spring or to tetraploid Stewart 63 (Platford 1973). This study supported Platford's (1973) results which also indicate that chromosomes 1B and 3B of Carleton condition resistance to ergot.

A small part of the T. timopheevi resistance is apparently expressed by the removal of several different chromosomes of the A and B genomes of Chinese Spring. A major portion of the T. timopheevi resistance is expressed when crossed to the tetraploids Stewart 63 and Carleton, indicating dominant resistance; however when crossed with Chinese Spring, this resistance is suppressed, possibly by a gene(s) which may be located on the D genome.

This suppressor gene may not occur in all hexaploid wheats. Bernier (1973) found that the amphidiploid of T. timopheevi x Aegilops squarrosa (AAGGDD), Accession 6A51, was very resistant to ergot isolate M-4. This indicates that the A. squarrosa parents of the amphidiploid did not contain a gene repressing the T. timopheevi resistance. It would therefore seem possible to find other hexaploid wheats which do not suppress the T. timopheevi resistance.

The possibility that the resistance may be controlled by cytoplasmic factors is not considered likely for the following reasons:

1. Manitou wheat was found to be susceptible to ergot, as was male sterile Manitou which contains T. timopheevi cytoplasm (Bernier 1973).
2. In the T. timopheevi x rye triploid where T. timopheevi was used as the female parent, one plant was found to be susceptible, even though the triploid must have contained T. timopheevi cytoplasm.
3. The expression of T. timopheevi resistance in the Stewart 63 x T. timopheevi F_1 hybrid where T. timopheevi was used as the male parent, indicates the presence of a genetic, rather than a cytoplasmic inheritance.

It is possible to speculate that resistance to ergot in cereals may be due to three possible mechanisms:

1. The functional barrier formed by the floral parts of a cereal floret which may completely enclose the ovary, thus preventing the entry of inoculum. At anthesis, the floret often opens, and this barrier ceases to be effective.
2. The resistance of the ovary to infection that was found by Ratanopas (1973) to be effective after anthesis and fertilization.
3. The resistance in T. timopheevi and Carleton to M-4 isolate of ergot which this study investigated, that was effective two days before anthesis. A major part of the T. timopheevi

resistance was found to be expressed in F_1 tetraploid hybrids even though they were completely sterile.

This third type of resistance may be useful not only in reducing the levels of ergot in spring wheat, but also in new areas of crop research where ergot is a problem. T. timopheevi resistance could be effective in reducing the susceptibility of the male sterile parent lines which are used in the production of hybrid wheat. This study has also given some indication that T. timopheevi resistance may be effective in the presence of the rye gamete. Thus it may also be possible to incorporate this resistance into Triticale.

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APPENDIX I

A COMPARISON OF AVERAGE DISEASE INDEX AND DISEASE COMPONENTS ON 34 AND 35 CHROMOSOME HEADS+ OF CHINESE SPRING x CARLETON F_1

Monosomic Line	Chromosome Number	Disease Index	Sclerotia Number (per head)	Sclerotia Size (Index)	Honeydew (Visual Rating)
1A	34	4.8	4.2	89	3.8
	35	4.3 NS	5.0 NS	96 NS	3.5 NS
2A	34	4.3	4.7	85	3.5
	35	4.6 NS	7.7 NS	87 NS	3.6 NS
3A	34	4.7	7.7	94	3.7
	35	4.6 NS	4.7 NS	85 NS	3.8 NS
4A	34	4.6	5.3	90	3.8
	35	4.7 NS	5.7 NS	85 NS	3.9 NS
5A	34	5.0	8.3	96	3.8
	35	4.8 NS	5.5 NS	89 NS	3.7 NS
6A	34	4.7	7.6	87	3.2
	35	4.7 NS	7.4 NS	92 NS	3.9 *
7A	34	4.6	5.4	84	3.6
	35	4.7 NS	7.0 NS	89 NS	3.7 NS
1B	34	2.9	4.8	60	2.3
	35	4.6 **	7.2 **	89 **	3.2 **
2B	34	4.5	7.5	90	3.2
	35	5.0 NS	8.2 NS	96 NS	3.8 NS
3B	34	3.8	1.5	78	2.9
	35	4.8 **	4.1 *	95 *	3.4 NS
4B	34	4.6	8.6	87	3.5
	35	4.9 NS	7.6 NS	93 NS	3.7 NS
5B	34	4.9	8.1	94	3.4
	35	5.0 NS	7.8 NS	97 NS	3.2 NS
6B	34	4.8	5.9	91	3.5
	35	4.7 NS	5.4 NS	89 NS	3.6 NS
7B	34	4.6	5.5	89	3.6
	35	4.4 NS	5.4 NS	83 NS	3.6 NS
Carleton check	28	1.5 Range (1-3)	0.7 Range (0-4)	16 Range (0-33)	1.3 Range (1-3)
Chinese Spring	42	4.9 (4-5)	7.8 (3-10)	88 (80-100)	3.6 (3-4)

\+ Between 4 and 20 heads compared in each comparison.

* Difference between comparisons is significant at 5% level.

** Difference between comparisons is significant at 1% level.

NS Difference between comparisons is non-significant.

APPENDIX II

A COMPARISON OF AVERAGE DISEASE INDEX AND DISEASE COMPONENTS ON 34 AND 35 CHROMOSOME HEADS+ OF CHINESE SPRING x *T. timopheevi* F_1

Monosomic Line	Chromosome Number	Reaction Index	Sclerotia Number (per head)	Sclerotia Size (Index)	Honeydew (Visual Rating)
1A	34	3.8	5.0	71	2.6
	35	3.4 NS	5.2 NS	58 NS	2.6 NS
2A	34	4.0	4.9	88	2.4
	35	4.5 NS	6.5 NS	78 NS	3.3 **
3A	34	4.5	3.3	96	3.2
	35	4.5 NS	5.9 NS	92 NS	2.9 NS
4A	34	4.0	4.0	86	3.0
	35	4.1 NS	4.6 NS	85 NS	3.0 NS
5A	34	3.8	3.8	83	2.8
	35	3.6 NS	2.0 NS	82 NS	2.8 NS
6A	34	4.2	3.7	91	3.0
	35	4.1 NS	7.0 NS	82 NS	2.6 NS
7A	34	4.2	6.5	82	3.1
	35	3.8 NS	4.2 NS	85 NS	2.7 NS
1B	34	3.8	5.7	69	2.5
	35	4.6 NS	6.5 NS	81 NS	3.2 *
2B	34	4.3	6.9	81	3.0
	35	4.4 NS	6.3 NS	87 NS	3.1
3B	34	3.8	6.8	58	2.6
	35	4.6 *	7.5 NS	83 *	2.8 NS
4B	34	4.2	4.7	86	3.2
	35	4.3 NS	7.7 **	81 NS	2.8 NS
5B	34	4.3	5.1	85	2.9
	35	3.7 NS	3.7 NS	76 NS	2.4 NS
6B	34	4.4	6.4	89	3.2
	35	4.4 NS	5.8 NS	92 NS	2.9 NS
7B	34	4.2	4.8	80	2.4
	35	4.1 NS	4.0 NS	87 NS	2.9 *
T. timopheevi	28	1 Range (1)	0 Range (0)	0 Range (0)	1 Range (1-)
Chinese Spring	42	4.9 (4-5)	7.8 (3-10)	88 (80-100)	3.6 (3-4)

\+ Between 6 and 20 heads compared in each comparison.

* Difference between comparisons is significant at 5% level.

** Difference between comparisons is significant at 1% level.

NS Difference between comparisons is non-significant.