THE UNIVERSITY OF MANITOBA

THE GENETICS OF RESISTANCE TO *PUCCINIA GRAMINIS TRITICI* IN EIGHT WHEAT VARIETIES FROM KENYA

by

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In searching for rust resistant wheats for breeding purposes in Kenya, one of the major impediments has been to identify the factors for resistance carried by the major sources of resistance. Some progress has been made in this direction in the present investigations.

The inheritance of seedling resistance to races EA4(295), EA5(34), EA7(40), EA8(40), C10(15B-1) and C17(56) of stem rust was studied in the wheat cultivars and strains: Kenya Hunter, Kenya Leopard, C.I.8154-Frocor^{2}, Tobari 66, Trophy, Minnesota 3654/60, Conley, and Wisconsin 245-II-50-17 from the Kenya Stem Rust Parental Collection nursery. Each variety was analysed genetically by determining the ratios obtained from F_2 populations and backcrosses to the rust susceptible variety Hindi 62. The interrelationships of the genes in the varieties were determined by: crossing with the Sr tester stocks, inter-varietal single gene line crosses, gene expressions, host-pathogen infection types and the pedigree relationship. Three backcrosses were made and 25 F_3 homozygous single gene lines were isolated. By genetic analytical methods, these 25 inter- and intra-varietal lines were found to possess ten different genes. Five of these were identified as: Sr1, Sr6, Sr7, Sr11 and sr17. While the remaining five genes have not been described previously.

Additional races: EA11(40), EA12(40), EA13(34), EA14(11), EA15(11), EA16(40); C1(17), C2(17A), C18(15B-1L), C20(11), C22(32), C25(38) and C41(32-113) were used to determine the spectrum of effectiveness of the five new isolated single genes. Excellent levels of resistance to all

races tested are conferred by one or more of these genes.

The mode of inheritance was either single or duplicate for any one particular host-pathogen relationship. Recessive, dominant and partial dominant genotypes were predicted. In certain cases the mode of inheritance could be explained most precisely when modifiers or linkages were assumed. For instance, of the two genes in Kenya Leopard: one conditioned resistance to both EA5 and C17(56) while the other controlled resistance against EA7. These genes were found to be linked with 28.3 per cent recombination value. Varieties such as Trophy, Tobari 66 and C.I.8154-Frocor^{2} exhibit a moderate resistance reaction ranging between infection type 2^{+}_{-} and x^{-}. The reactions were usually temperature sensitive and were apparently conditioned by several minor factors. These were difficult to retrieve in a backcross programme. Resistance to race EA8 was characterized by being conditioned by recessive genes in addition to a number of modifiers which generally enhanced the major gene effect. Other races did not demonstrate any particular pattern for gene expression.

In the absence of inter-allelic gene interaction it was difficult to explain the broad resistance of the monogenic lines by assuming the gene-for-gene hypothesis alone. More information regarding allelism, linkage between the host resistance genes or an alternate hypothesis is needed to explain the host-pathogen relationship.

INTRODUCTION

Stem rust, one of the most devastating diseases of wheat Triticum aestivum L., is probably more severe in Kenya than in any other wheat producing area. This is due to a combination of ideal climate, year round wheat production and highly virulent strains of the causal organism, Puccinia graminis Pers. f. sp. tritici Eriks. and E. Henn.

Researchers in Kenya have aimed at producing rust resistant varieties since 1908 and although many varieties have been released their average period of effective resistance has been only 4.1 years. Periodically, rust attacks of epidemic proportion have occurred resulting in erratic production levels.

To prolong the effective life and reduce the number of varieties, more effective breeding procedures are required. One way is to systematically incorporate several genes for resistance to each of the prevalent races into a single cultivar thus providing protection in depth so that the organism must pass through a number of mutations to overcome the resistance. However, to produce varieties with the adequate depth and wide spectrum of resistance the plant breeder must be aware of the genetic identity and level of resistance which he can extract and easily transfer from his sources of resistance. Another essential requirement to breeding for such resistance is the prompt identification of new and potentially dangerous races of rust. Perhaps, even more critical are surveys of currently resistant varieties and the identification of rust that appears on them. In this way the breeder obtains advance informa-

tion regarding the new races and new sources of germplasm for a systematic breeding programme against rust.

In Kenya, despite the extensive research on rust resistance in wheat and the notable, frequent successes in producing ephemerally resistant varieties, no genetic studies of host-parasite relationships have been carried out on any of the current sources of resistance.

Investigators in Australia, Canada, the United States and other countries have established the presence in common wheat of a number of major loci which condition seedling reactions to various stem rust cultures on the gene-for-gene model. Many of these genes have been isolated in monogenic lines with a common background. Most of these monogenic stem rust (Sr) lines being used in other countries, are ineffective against most of the prevalent races in Kenya as reported by Green (1970) and Harder (1972). However, adequate sources of resistance are available to the races of rust now prevalent in East Africa but information regarding the mode of inheritance, the number of loci involved and the relationship with identified genes for resistance are largely unknown.

Within this context the following eight varieties or strains of T. aestivum L. were selected for study from the Kenya Stem Rust Parental Collections (SRPC): Kenya Hunter, Kenya Leopard, C.I. 8154-Frocor^{2}, Tobari 66, Minnesota 3654/60, Conley, Trophy and Wisconsin 245-II-50-17. These cultivars are of diverse parentage, origin, have a wide range of seedling resistance and in most cases have a very high level of field resistance in Kenya. They are grown commercially or are used extensively

in the Kenya breeding programme as sources of rust resistance. Attempts are also being made to find resistance genes of value to plant breeding programmes in North America. The primary tester races are East African races EA4(295), EA5(34), EA7(40) and EA8(40) and the important North American races C10(15B-1) and C17(56).

The aims of the present investigations were to:

1. Determine the mode of inheritance and number of factors for resistance to stem rust races EA4(295), EA5(34), EA7(40), EA8(40), C10(15B-1) and C17(56) in the eight cultivars.

2. Isolate monogenic resistant lines. Stem rust resistant monogenic lines were established by placing each effective identified gene into a common genetic background of Hindi 62 by backcrossing.

3. Identify the isolated genes. Each monogenic line will be defined in relation to:

   a) the established stem rust (Sr) genes.

   b) Intra-varietal genes.

   c) Inter-varietal monogenic lines conferring identical resistance reaction to the same race.

4. Determine the effective range of resistance of the isolated genes to races other than the primary tester races.

5. Evaluate the genes revealed in relation to the Kenya breeding programme.

2. REVIEW OF LITERATURE

2.1 STEM RUST AND SOURCES OF RESISTANCE IN KENYA

Wheat (Triticum aestivum L.) was introduced into Kenya at the end of the nineteenth century, and by 1908 black stem rust, a disease caused by the basidiomycetous fungus Puccinia graminis Pers. f. sp. tritici Eriks. and E. Henn. had appeared. By 1910, when a breeding programme for resistance was initiated, the disease had reached epidemic proportions. Reviews of the efforts to protect the wheat industry against rust have been published by Lathbury (1947), Thorpe (1958), Dixon (1960), Guthrie (1966), Hurd et al. (1969), Pinto et al. (1970) and Harder et al. (1972). In order to comprehend the significance of the present genetic study a brief review of the wheat improvement programme in Kenya is necessary.

2.1.1 THE PERIOD 1900-1952

Prior to 1925 the majority of the wheat grown was introduced varieties from Australia, Egypt, Italy and Canada. From 1925-1951 there was an increase in the number of locally bred varieties derived from crosses between Kenya varieties and a few significant introductions (Dixon 1960 and Knott 1962). Consequently, the majority of the commercial varieties possessed a narrow genetic base for resistance. During this period, a succession of new stem rust races occurred, but the frequency with which they appeared and their virulence was not severe thus enabling the breeding programme to keep pace. It is most probable that initially the host-resistance genes surpassed the pathogenicity genes of the contem-

porary races. Therefore, in spite of new races, Kenya was well supplied with resistant germplasm of local origin. In 1951, eighty per cent of the Kenya wheat acreage was sown with local varieties (Anonymous 1969).

Many of these varieties have been used as sources of stem rust resistance in breeding programmes throughout the world (Macindoe 1937 and Watson et al. 1963). Those most widely used have been K58, K117A, K321, K324, K338AC named "Kenya Farmer", K340, K360 and Kenya Governor also known as Egypt Na 101. Knott (1962) showed most of these varieties to contain important genes conferring resistance to the North American races. These cultivars are now susceptible to the prevalent stem rust races in East Africa and additional sources of resistance had to be introduced to provide adequate protection.

2.1.2 THE PERIOD 1952-1959

During this era a large number of highly virulent rust races emerged which were capable of attacking all the Kenya commercial varieties (Dixon 1960 and Guthrie 1966).

To counteract this problem, Kenya first took part in the international nurseries of organized wheat collections in 1953. The best selections from this cooperative programme were put into the Stem Rust Parental Collection (SRPC). Since many of the exotic varieties had relied on genes for resistance from Kenya varieties, they too succumbed. While the breeder thus responded by broadening the genetic diversity of the breeding material, the pathologist sought for a differential set of varieties with a wider genetic background to enable him to relate more precisely

the work in Kenya with that in other parts of the world (Guthrie 1964 and 1966). Subsequently, McDonald's (1931) system of local differentials was abandoned in favour of Stakman's differential system (1962). With this system, Guthrie (1966), isolated and reclassified sixteen races and six biotypes. TABLE 2:1 shows the races identified to 1970, their time of occurrence and the systems adopted to describe them.

TABLE 2:1 THE STEM RUST RACES OF PUCCINIA GRAMINIS TRITICI IN KENYA DESCRIBED SINCE 1928.

YEAR IDENTIFIED	RACE
McDonald's System	
1928	K1(21), K2(17)*
1930	K3(34)
1931	K4(116)
1936	K5(21)
1938	K6(107)
1943	K7
1948	K8(24)
1950	K9(122), K10(24), K11(24)
1951	K12(21)
1953	K13(42), K14(143)
1954	K15
1955	K16(40)
1957	K17(40), K18(40), K19(189)
Guthrie's System	
1959	11, 14, 184, 295
1960	296, 40T
1961	297, 4 Sub-races of 40
1963	117, 15
Green's System	
1968	EA1(297), EA2(21), EA3(295), EA4(295), EA5(34), EA6(40), EA7(40), EA8(40), EA9(11)
1969	EA10(15), EA11(40), EA12(40)
1970	EA13(34), EA14(11), EA15(11), EA16(34), EA17(143), EA18(83), EA19(40)

* Numerals in parentheses indicate the "standard" race number.

K = Kenya races, EA = East African races.

(Compiled from Records of the Plant Breeding Station, Njoro, Kenya).

2.1.3 THE PERIOD 1960-1966

The disease resistance programme depended heavily on foreign introductions as stated by Dixon et al. (1965). By 1967 foreign varieties accounted for 66 per cent of the Kenya wheat acreage. Although the stem rust races showed a wider range of pathogenicity, the germplasm in the commercial varieties was greatly diversified and broadened. During this period the main source of resistance stemmed from the Mexican Yaqui group of varieties, the Triticum timopheevi Zhuk. derivatives C.I.12632 and C.I.12633, the S.A. No. 43 complex which incorporated T. timopheevi and Agropyron elongatum (Pinto et al. 1970), the Frontana-Kenya 58-Newthatch group and Mida-McMurachy-Exchange from Minnesota. Numerous crosses were made involving these cultivars with the hope that highly resistant recombinants might be identified. In spite of these efforts, the cultivars released to the farmers usually became susceptible in a few years, but losses were kept at a minimum by requiring each farmer to grow several cultivars with different kinds of resistance. Growing two crops in a year on the same land or farm was prohibited in most of the country. Production during this period improved tremendously, and by 1967 there was a problem of disposal of the surplus wheat.

2.1.4 THE PERIOD 1967-1972

This is the period when the Canadian International Development Agency (CIDA) team became actively involved in the wheat improvement research in Kenya. The current programme has been reviewed by Hurd et al. (1969), Evans et al. (1969), Green et al. (1970), Pinto et al. (1970)

and Oggema et al. (1971).

During this period the previously used methods were re-evaluated with a view to improving the wheat research efficiency. The theme of the period is to produce agronomically suitable cultivars with lasting resistance. To achieve this, the main sources of resistance are being utilized more systematically as outlined by Evans (1969). Close co-operation between the plant breeder and the pathologist is imperative in order to screen rapidly the breeding material and the genetic stocks both in the greenhouse and in the field. Race surveys and identification are conducted throughout the year to determine the epidemeological cycle, virulence of the races and race identity in the manner described by Green et al. (1970) and Harder et al. (1972). Whenever new virulent races are identified, new kinds of resistance are located and transferred to adapted Kenya varieties promptly by a backcrossing or a convergent backcrossing programme (Hurd et al. 1969, Evans 1969 and Pinto et al. 1970). When the genetics of resistance of the SRPC material is adequately established, a gene management programme to utilize the identified genes most efficiently will be planned (Anonymous 1971).

The change in the breeding approach led Green (1970) to alter the race differential varieties and the nomenclature of the races. TABLES 2:1 and 3:5 show the current race nomenclature.

Although much emphasis is placed on seedling resistance as a criterion of selection, post-seedling resistance is also being incorporated simultaneously wherever possible (Anonymous 1971 and Harder et al. 1972).

2.2 DISTRIBUTION AND ORIGIN OF STEM RUST EPIPHYTOTICS IN KENYA

One of the subjects which has attracted great interest in Kenya is the mode of origin of new rust races. McDonald (1931) examined local species of barberry (Barberis holstii) and concluded that the rust that was found on this species was not Puccinia graminis tritici and that the barberry did not function as an alternate host. Guthrie (1964) confirmed this finding. Guthrie (1964) stated that large scale movement of spores from Ethiopia by the North East monsoon wind which blows during the months of November to March probably influences the distribution of the races in Kenya. The minimum distance between wheat growing areas in the two countries is approximately 400 miles. In an attempt to get more evidence on rust movement (Guthrie 1964) a Uniform Rust Nursery was organized which included several African countries. The results indicated that virulence in Ethiopia was similar to Kenya. Preliminary studies reported by Oggema et al. (1971) tend to support this view. Ethiopian races identified by Sibilia (1939) were compared by Nattrass (1949) and proved to be similar to the Kenya races prevailing at that time.

Another factor is that wheat is planted in Kenya at times to maximize use of rainfall while facilitating harvest during a dry period. Due to major seasonal and altitude differences wheat is growing continuously somewhere in Kenya throughout the year so that the uredial stage of the rust is perpetual. The epidemeological data reported by Green et al. (1970) and Harder et al. (1972) revealed that there are airborne urediospores throughout the year. These authors suspect this to be the most

probable source of initial inoculum. Preliminary studies of wild grass as a possible second source of inoculum did not give clear evidence that grasses growing in the areas in which the collections were made were major sources of inoculum.

2.3 THE GENETICS OF RESISTANCE

2.3.1 HISTORICAL REVIEW

A number of workers have classified rust resistance into morphological, functional and physiological resistance (Goulden et al. 1930). However the two major types of resistance to stem rust which have been differentiated on a genetic basis (Hooker 1967) are seedling and adult plant resistance. Seedling resistance conditions resistance throughout the life of the plant to a particular race or races of stem rust. The resistance is usually race-specific. A variety carrying adult plant resistance may be resistant to all prevalent races under field conditions even though it is susceptible to certain of these races in the seedling stage (Stakman et al. 1925, Koo et al. 1951, Van der Plank 1963, MacKey 1965 and Hooker 1967).

In the early history of rust work there was doubt regarding the use of seedling tests as a measure of resistance in the after-heading stages (Goulden et al. 1930). However, the following advantages have been cited by MacKey (1965), Hooker (1967) and Johnson et al. (1967) which point to the usefulness of seedling resistance: a) In almost all cases plants resistant in the seedling stage are also resistant at maturity (Goulden 1930); b) Under greenhouse conditions the environment can be manipulated

to suit the purpose of the test; c) Dangerous rust forms of rare occurrence which are to be restricted may be used for inoculations in the greenhouse and therefore it is possible to get advance performance on the prevailing breeding material (MacKey 1965); d) A large number of plant populations can be screened in a restricted space within a short time. e) Host-gene : parasite-gene relationships can conveniently be analysed from seedlings as noted by Loegering et al. (1962 and 1969), Rowell et al. (1963), Green et al. (1966) and Williams et al. (1966).

Nevertheless, physiologic resistance determinations to be of any real benefit towards elucidating the problem of the plant breeder, should be established at that stage in which the plants become most vulnerable (Watson 1968, Anonymous 1970).

2.3.2 THE HOST : PARASITE RELATIONSHIP

It is evident that the infection type produced by any combination of rust race and host variety must be the result of an interaction between the two genetic systems.

The gene-for-gene hypothesis postulated by Flor (1942, 1946, 1956 and Flor et al. 1971) for flax (Linum usitatissimum Ehrenb.) and flax rust (Melampsora lini Desm.) has found wide recognition by many workers including Person (1959), Loegering et al. (1962 and 1966) and Johnson et al. (1967). Mayo (1956) and Green (1965) have encountered certain situations which cannot be simply explained by the gene-for-gene model.

Loegering and Powers (1962) detected eight genes for pathogenicity in the F_2 progeny of a cross between races 111 and 36 of wheat stem rust.

The genes were identified on the basis of the differential pathogenicity of the F_2 cultures on wheat varieties Hope XVII, Marquis, Reliance, Chinese Spring, Acme and Einkorn. On the basis of the gene-for-gene hypothesis they postulated that these varieties carried eight resistance genes.

Green (1964) using a rust culture with a colour mutant indicated that a gene-for-gene relation may exist between Puccinia graminis Pers. f. sp. tritici and T. aestivum L.

The results presented by Williams et al. (1966) strongly supported the gene-for-gene hypothesis for the wheat-rust genetic relationship. In addition to a one-to-one genic relationship, they also observed that phenotypes (agricorpus) resulting from highly specific gene-for-gene interactions were sometimes modified slightly by other resistance genes in the host.

2.3.3 MODES OF INHERITANCE

The first demonstration that resistance to any plant disease may be inherited in a Mendelian manner involved a plant rust. This important finding was reported by Biffen in 1905. Consequently, the literature on inheritance of resistance to physiologic rust races in wheat is voluminous. The examples presented in this review only show its diversity. Studies on physiologic specialization in rust resistance have involved chiefly the hypersensitive type of resistance. Segregations for physiologic resistance (specific resistance) are usually discrete and frequently fit simple genetic ratios. Additional information on inheritance and in-

formation on the chromosomal locations of genes for resistance have been obtained through the use of chromosome translocation by Sears (1956), and Russell et al. (1962); chromosome substitution lines by Sears et al. (1957), Kuspira (1959), Knott (1959), Green et al. (1960), Sheen et al. (1965) and by the use of other aneuploidy analysis Nyquist (1957), Loegering et al. (1966) and Sears et al. (1968).

The following modes of inheritance have been postulated from a number of genetic studies:

2.3.3.a MONOGENIC INHERITANCE

Resistance that is due to a single completely dominant gene is commonly observed in rust inheritance studies, (Macindoe 1948 and Knott et al. 1957). Resistance that is due to a single incompletely dominant gene has been reported by Ausemus (1943), Green et al. (1960) and Loegering et al. (1969). Berg et al. (1963) found a recessive gene to condition resistance in a cross between Marquis and Little Club. Watson et al. (1968) have transferred a recessive gene sr17 from "Yaroslav Emmer" into the Marquis background. In classical genetics (Strickberger 1969) the gene is equated to the expression of a certain character and exists in at least two separate functional forms (alleles or allelomorphs) which segregate at meiosis and recombine at fertilization. When studied in detail, a gene is frequently found to exist in several allelic forms. This is apparently also true for genes conditioning rust resistance. Pathological and genetic investigations involving Chinese Spring substitution lines, conducted by Loegering and Sears (1966), showed that the variety

Kenya Farmer carries the Sr7a gene for resistance on chromosome 4B. Hope carries a different allele Sr7b. They also confirmed the existence on chromosome 2B of gene Sr9a in Red Egyptian and Sr9b in Kenya Farmer and Kenya 117A.

These genes are regarded as alleles for two reasons: a) they show no crossing over in the usual genetic studies where only a few hundred products of meiosis are examined and b) each gene conditions a different spectrum of resistance to a series of differential biotypes of the pathogen. However, with the possible discovery of closely linked genes, interpretations of allelic relationships may be revised.

2.3.3.b TWO OR MORE GENES ACTING INDEPENDENTLY

Numerous genetic studies have revealed several genes for resistance in a variety. This is to be expected since present day plant breeders incorporate several genes for resistance into a single variety. The genes may be dominant, recessive, or a combination of dominants and recessives and act independently or through a form of gene-interaction. Athwal *et al.* (1956) tested F_2 and F_3, backcross F_2 and families from F_4 and backcross F_3 of *Triticum dicoccum* Schübl. "Khapli" x *Triticum durum* Desf. Marrocos 9623. Their results indicated that at least four genes control the seedling reaction of Khapli to *P. graminis tritici*. In a cross between Minnesota II-50-18 and Kenya Farmer, Ausemus *et al.* (1951) obtained a segregation of fifteen resistant to one susceptible indicating that II-50-18 and Kenya Farmer differ by two pairs of dominant factors in their reaction to race 15B.

Knott (1962) concluded that resistance to race 56 of Khapstein (T. dioccum "Khapli" x T. aestivum "Steinwedel") is conditioned by two dominant genes which he designated Sr13 and Sr14. While resistance to race 15B was conditioned by Sr13 and Sr7. In the latter case Sr13 acted as a recessive for resistance.

Gough et al. (1963) crossed and backcrossed two durum varieties "Acme" and "Mindum" to a susceptible variety "Marrocos 9623". Tests of F_2 and F_3 generations from the crosses and F_1 and F_2 generations from the backcrosses indicated that the resistance of each of the varieties was conditioned by at least three incompletely dominant genes. Resistance has also been noted by Sears (1957) to be due to two recessive genes.

2.3.3.c LINKAGE AND INDEPENDENT ASSORTMENT

A gene can obey Mendel's second law only if the two genes occupy sites on different chromosomes or are located 50 or more cross-over units apart on the same chromosome. If two genes segregate together more often than they do apart, the two genes are regarded as being linked. Loose linkage of rust resistance genes has been noted in several genetic and aneuploidy analyses (Loegering et al. 1966, Sears et al. 1968, Shuh-Ji et al. 1968). Knott (1959) reported that Sr11 and Sr12 were located on chromosome 6B with a 28 per cent recombination. Later Green et al. (1960) detected only one gene which has been designated as Sr11.

Some of the so-called alleles might be tightly linked genes which would require a very large population for resolution (Hanson 1959).

2.3.3.d GENE INTERACTIONS

It has been frequently observed that many resistance genes are modified by interaction with non-allelic genes.

Complementary gene action is commonly used to describe the interdependence of two or more genes, all of which are essential for the ultimate expression of a character. Ausemus (1943), Macindoe (1948) and Koo et al. (1951) found two recessive complementary genes; while Bahl et al. (1961) detected three recessive complementary factors for resistance. In the cross between Ceres and Kenya Farmer, (Ausemus et al. 1957) the F_2 population segregated into a ratio of 9 resistant to 7 susceptible indicating a digenic complementation. Others who have observed two-gene complementary factors in their material include Knott et al. (1956), Aslam et al. (1958), Omar et al. (1956) and Rajaram et al. (1970). Rao 1970 reported one dominant and one recessive complementary gene interaction.

Epistatic gene action has also been reported. Ausemus (1943) noted that the expression of a gene for rust resistance may be inhibited by a gene at another locus. Berg et al. (1963) and Sunderman et al. (1963) observed that genes that condition a higher level of rust resistance are commonly epistatic to those conditioning a lesser reaction.

Genes which modify the action of other major genes have also been reported. Green et al. (1960) observed that genes Sr9, Sr10 and Sr11 modify the action of Sr7 and increase its resistance to race 15B. The effects of modifier genes have clearly been demonstrated when major genes are transferred individually to a common background by backcrossing.

Green et al. (1960) and later Knott (1964) noted that with extended backcrossing in the isolation of monogenic lines the level of resistance retained was less than the original parent. For instance, Marquis lines backcrossed ten times to isolate Sr11 from Lee were less resistant than lines with six doses of Marquis and less than the donor parent variety Lee. Presumably Lee carries modifier genes for resistance to stem rust which were not retained. Sears et al. (1957), by the use of monosomic analysis obtained evidence for the existence of several modifiers of resistance to certain races.

Transgressive segregation for rust reaction refers to the appearance of plants in the F_2 or later generations that are either more resistant or more susceptible than either of the two original parents. This has been reported by Allan (1966) for wheat stripe rust (Puccinia striiformis West.). Shuh-Ji et al. (1968) found that the varieties Conley and CT231 have major genes for 15B stem rust which were different and additive in their effects.

Reversal of dominance of a gene for rust resistance is unusual. In some instances a gene may be expressed as a dominant to some biotypes of the fungus and as a recessive to others. Knott et al. (1956) reported that Sr6 controls resistance to both races 56 and 15B, being dominant to race 56, but recessive to race 15B. Sunderman et al. (1963) also observed that in some genetic backgrounds a gene may be expressed as a recessive and dominant in others. Chester (1946) reported that a gene may behave as a recessive in the seedling stage, but behave as a dominant as the

plant becomes older.

Samborski (1963) cautioned that recessiveness or dominance of certain resistance genes may depend upon the homozygosity or heterozygosity of the corresponding locus controlling avirulence in the parasite, in some cases the apparent reversal of genes may be attributed to differences between cultures of races used in the studies.

2.4 RUST RESISTANCE GENES AND ENVIRONMENT

Susceptibility or resistance reaction phenotypes are the disease symptoms which result from a complicated interaction. The interactions result from combinations of several factors: the host, the rust pathogen and the ambient environment.

Most of the classical investigations pertaining to environmental effect on resistance have been concerned mainly with temperature and humidity and to a very limited extent with light or soil factors (Johnson et al. 1940, Forsyth 1956, Sheen and Snyder 1965). Genes for rust resistance may respond differentially to environmental conditions (Knott 1957, Watson et al. 1963 and 1968). Two genes which condition resistance to the same rust biotype may differ in temperature sensitivity. This trend has been discussed by Knott (1957) for Sr6 and Loegering et al. (1969) for Sr16. Loegering et al. (1969) also observed that Sr8 confers moderate resistance under normal circumstances but at temperatures below 20°C the characteristic chlorosis becomes indistinct, and the homozygous isogenic pair may be difficult to differentiate from the heterozygote. Thus the dominance is variable in response to temperature effect.

Mohamed (1960) found that pre-inoculation temperatures affect the number of infection centres and stem rust infection. More infection centres were produced and infection types were higher if the pre-inoculation temperature was 24°C.

2.5 THE ESTABLISHED STEM RUST (Sr) GENES

Most of the identified stem rust (Sr) genes have been isolated and named by Dr. D. R. Knott and collaborators, (Knott 1957, 1959 and 1966) and (Knott et al. 1956 and 1960). The system of allocating gene symbols is basically the same as that recommended by Ausemus et al. (1946). The symbol for stem rust resistance "Sr" is followed by the Arabic numeral to indicate the locus in the variety.

2.5.1 THE GENES Sr1 and Sr2

Ausemus et al. (1946) recommended that the two genes conferring adult plant resistance in Hope and H-44 be named Sr1 and Sr2. Since much of the previous work was based on field reactions, it is impossible to be sure of the identity of these two genes. Knott (1968) identified one gene from Hope from seedling studies as being located on chromosome 2B, and this he designated Sr1. While the gene giving adult plant resistance he named Sr2.

The second gene (Sr2) has not been isolated probably because of the difficulty of working with adult plant resistance. Furthermore the gene Sr7 in Khapstein is likely to be the gene previously named Sr2 by Heermann et al. (1957) and Srd2 by Kenaschuk et al. (1959).

2.5.2 THE GENES Sr3 and Sr4

The naming of all the genes identified prior to 1946 as Sr1 to Sr4 was recommended by Ausemus et al. (1946). The genes Sr3 and Sr4 are known to be complementary genes in the variety Marquillo (Hayes et al. 1925). Till now, it has not been confirmed whether any of these genes are identical with any of the current Sr genes. These two genes have neither been isolated into any common background nor have they been properly described. However, they are known to confer mature plant resistance.

2.5.3 THE GENE Sr5

Sr5 is the "Kanred" immunity gene. It is located on chromosome 6D of the variety Thatcher (Watson et al. 1963). Sears et al. (1957) and Sheen and Snyder (1964) observed that this gene provides a high level of resistance to numerous cultures.

2.5.4 THE GENE Sr6

Sr6 is often referred to as the McMurachy gene. It occurs on chromosome 2D. It conditions a hypersensitive reaction to races 15B and 56, but is recessive to 15B and dominant to 56. It is very temperature sensitive (Knott 1957 and Watson et al. 1963). Watson et al. (1963) listed 55 wheat sources of Sr6.

2.5.5 THE GENE Sr7

Sr7 conditions a type $1^{\pm}$ infection type to race 15B and is partially dominant. Loegering et al. (1966) located it on chromosome 4B of Kenya

Farmer and Hope. Kenya Farmer and Sapporo No. 1 both carry the Sr7a allele while Hope carries the Sr7b allele. A characteristic of Sr7 resistance is the diffuse necrosis of the seedling leaves particularly at the leaf tip and margins. With other races, various infection types and degrees of yellow chlorosis occur. Pustule size may vary from infection type 1 to 3. This gene came from the Emmer variety Khapli (Knott 1966). Sr7 is likely the gene named Sr2 by Heermann et al. (1957) and Srd2 by Kenaschuk et al. (1959). The gene is known to be extensively modified by other major Sr genes (Green et al. 1960).

2.5.6 THE GENE Sr8

This gene conditions a 2^{+}_{-} seedling infection to races 15B and 56 and is partially dominant. It is temperature labile. It occurs on chromosome 6A. It has been transferred to Marquis from Red Egyptian. Knott (1962) was not able to detect the presence of Sr8 in the Kenya varieties.

2.5.7 THE GENE Sr9

Sr9 conditions a moderately resistant reaction to race 56 and is partially dominant. Three alleles Sr9a, Sr9b and Sr9c have been reported (Green et al. 1960, Watson et al. 1968 and Sears 1969). Sr9a was detected in Red Egyptian and Sr9b in Kenya 117A and Kenya Farmer. Three Australian varieties; Timvera, Mengavi and Mendos, and the Wisconsin lines C.I.12632 and C.I.12633 possess the Sr9c allele. Sr9 is located on chromosome 2B of Kenya Farmer and Red Egyptian (Sears et al. 1957).

2.5.8 THE GENE Sr10

Sr10 is a partially dominant gene. It conditions moderate resistance to race 56 but is susceptible to race 15B. It was transferred to Marquis from Egyptian Na 95 (Green et al. 1960). Sr10 appears to be a major modifier of the resistance to race 15B conditioned by Sr7. Dyck et al. (1969) state that Sr10 does not provide resistance to races currently prevalent in Western Canada, and as far as they know, Sr10 is not present in the modern cultivars of North America.

2.5.9 THE GENE Sr11

This gene occurs in the varieties; Gabo, Timstein and Kenya Farmer. It conditions a moderate resistant reaction to race 56. The gene is located on chromosome 6B in Timstein and Kenya Farmer (Loegering et al. 1966). Sr11 is closely linked to the killer gene (ki) which reduces the male transmission and results in a distorted fit of a 3:1 resistant: susceptible plant ratio (Luig 1960 and Loegering et al. 1966). The gene Sr11 has been used extensively in plant breeding (Anonymous 1970). The gene gives clear infection types with most rust cultures.

2.5.10 THE GENE Sr12

Sr12 has not been properly identified (Green et al. 1960). It was originally shown to complement with Sr11 and had been located on chromosome 3B of Thatcher. Subsequently, Sheen et al. (1964) proposed the symbol Sr12 be used to designate the locus on chromosome 3B of Thatcher conferring high resistance to the 111 race culture.

2.5.11 THE GENE Sr13

Sr13 was transferred to "Marquis" background from "Khapstein". It conditions a moderate resistance reaction to races 56 and 15B. It is dominant to race 56 but, Knott (1962 and 1966), has found it to be recessive to race 15B. This gene together with Sr14, Sr15 and Sr16 was used for the first time in Canada in 1968 (Green 1969). Sr13 gives clear infection types with most race cultures.

2.5.12 THE GENE Sr14

The gene Sr14 was identified simultaneously with Sr13 by Knott (1962) and has since been transferred to Marquis from Khapstein (Knott 1966). It has no effect on race 15B and when alone it has only a slight effect on race 56. Knott's (1966) preliminary data indicated that Sr14 was located on chromosome 1B of Khapstein.

2.5.13 THE GENE Sr15

Sr15 has been identified in the varieties "Norka" and "Thew". It occurs on chromosome 7A. Green (1969) reported that this gene gave variable infection types that were often difficult to interpret. The variety Norka is used to test for the effectiveness of Sr15. It is mesothetic to a number of North American races including C9 and C18 and susceptible to all other North American races. The mesothetic reaction x^{-} is interpreted as a moderately resistant reaction. Sr15 is ineffective against all East African races, (Green et al. 1970).

2.5.14 THE GENE Sr16

Sr16 is located on the long arm of chromosome 2B, 5D or more cross-over units from the centromere, and is inherited independent of Sr9. The host-reaction is the infection type 2^{+}_{-}. Sr16 is incompletely dominant. Loegering et al. (1966 and 1969) and Green (1969) reported that Sr16 presents problems for genetic studies because it is affected by the environment and gives indefinite infection types. Sr16 has been isolated from Thatcher (Bârtos et al. 1970). Race C17 (56) is virulent on Sr16.

2.5.15 THE GENE sr17

This recessive gene has been transferred to Marquis from Yaroslav Emmer. Seedlings having this gene are highly resistant against specific strains in Australia. So far no rust culture from North America has been found which possess a gene interacting with sr17 (McIntosh et al. 1967 and Watson et al. 1968). It is present in the variety Mendos. McIntosh et al. (1967) located sr17 on chromosome 7B linked with a recessive gene for resistance to mildew and with an incompletely dominant gene for leaf rust resistance.

The Sr monogenic lines can be used to determine the races to which a particular gene gives resistance; they are being used as sources of resistance in breeding programmes and Watson et al. (1963) and Green (1965) have employed them as supplementary differentials to identify rust races. Sr lines are prerequisite in any successful regional gene-deployment programme or as a logical source of multiline varieties (Knott 1967,

Browning et al. 1969a and 1969b).

Many studies indicate that genes effective against races in one country are not necessarily useful in another country with a different rust pattern; or genes isolated in one country cannot often be differentiated in another country during the same period of time. For instance, the discovery of sr17 in Australia and not in North America.

Evans et al. (1969), Green et al. (1970) and Pinto et al. (1970) pointed out that the current Sr lines identified in North America are ineffective against most of the East African races in Kenya. Consequently, genetic investigations and the genes isolated from other regional programmes are of limited international application unless all the rust races are taken into consideration (MacKey 1965). Presently, for many countries, it is still unpopular to import rust races. Therefore, for rust-resistance genetics results to apply wholly to Kenyan rust conditions, the investigations have to be conducted in Kenya.

3. MATERIALS AND METHODS

3.1 SELECTION OF PARENTAL VARIETIES AND LINES

Over 3,500 varieties and homozygous lines of Triticum aestivum L. from the World Spring Wheat Collection Nursery sent to the Plant Breeding Station at Njoro (PBS) were screened for field resistance in 1967. Natural epiphytotics were good to excellent at both the Njoro and Molo trial sites. Nevertheless, to ensure heavy epidemics, the nurseries were artificially inoculated with a mixture of virulent stem rust races. Thirty-eight of the original cultivars and lines appeared highly resistant or heterogeneous for resistance under Kenya conditions (Evans et al. 1969). Out of these, eight have been extracted as donor parents in the present studies. TABLE 3:2 presents the names, abbreviations and pedigrees along with their geographic origin.

The cultivars and strains listed in TABLE 3:2 were selected for the following reasons:

a. Each strain has an established recognizable infection response to the main test races (EA4, EA5, EA7, EA8, C10 and C17). Most of these varieties possess genes which provide an effective resistance to at least two of the test races. TABLE 3:3 shows the infection types of the eight parental sources of resistance and of Hindi 62. The latter is susceptible to the current virulent East African races and to most of the North American races.

b. The strains are either being grown commercially in Kenya or are important sources of rust resistance in the improvement programme.

TABLE 3:2 THE PEDIGREES AND ORIGIN OF THE NINE PARENTAL WHEAT CULTIVARS AND STRAINS.

Abbre-viations	Cultivar of Strain	Pedigree	Origin
KHR*	Kenya Hunter	(Equator II x K318^2) x (Hope-Timstein x Regent) 869, B.4.C.1	Kenya
KLD	Kenya Leopard	(Lagaedinho x K354P^3) x (C.I.12632 x K354P^3) 1346 A.2.A.1	Kenya
TPY	Trophy	(Timstein-Kenya^2) x Yaqui 50 VI-42-17-1r-1m	Mexico
TBR	Tobari 66	Tzpp x Son 64A., 19021-4m-3y-102m-100y-101c	Mexico
CLY	Conley	Thatcher (McMurachy-Exchange x Redman^2) x Lee	U.S.A.
CIF	C.I.8154 x Frocor^2	III-1009-6-2t-3b-1t-2b-1t	Colombia
WIS	Wisconsin 245-II-50-17	C.I.12633 x (Frontana-Kenya 58-Newthatch)	U.S.A.
MIN	Minnesota 3654/60	(Frontana-Kenya 58-Newthatch) x Pilot	U.S.A.
H62	Hindi 62	Unknown	India

* These abbreviations will be used often in the paper.

TABLE 3:3 THE SEEDLING INFECTION TYPES OF THE NINE PARENTS TO THE TESTER SIX RACES.

VARIETY	INFECTION TYPES					
	EAST AFRICAN				NORTH AMERICAN	
	EA4(295) *1	EA5(34)	EA7(40)	EA8(40)	C10(15B-1)	C17(56)
KHR	;1 *2	23	3+	3+	3+	0;
KLD	3±	2-	;1	3+	3+	0;
TPY	1±	1±	2±	2+	3-4	0;
TBR	2±	;1	12	3+	x+	0;
CLY	2±	;1	12	3	23cn	0;
CIF	;1-	;1	;2	;2	x±	0;
WIS	23+	0;	;1-	23+	0;	0;
MIN	;2	;1-	2±	2±	0;	0;
H62	4	4	4	4	4	4

*1 Numerals in parenthesis refer to the international standard races (Stakman et al. 1962).

*2 The infection type symbols are explained in the text.

c. They are of very diverse origin and probably possess genes for rust resistance not previously identified.

Hindi 62 was selected as the susceptible parent because: it crosses easily and sets good seed with all the varieties being studied; it is photo-insensitive; has consistently exhibited a 4^{+} infection type to the six rust races being studied and displays very little environmental reaction variation.

3.1.1 THE PURITY OF THE HOST PARENTS

Prior to the investigations, the experimental material was grown in the PBS nurseries for at least five years. During this period, it was continually rogued. The year before the crosses were made a number of heads were bagged individually from random selected plants. Progenies of these selfed parental lines were inoculated several times to ascertain the infection types presented in TABLE 3:3.

3.2 THE OUTLINE OF THE ANALYTICAL PROCEDURE

The basic procedure followed throughout the investigations is illustrated in TABLE 3:4.

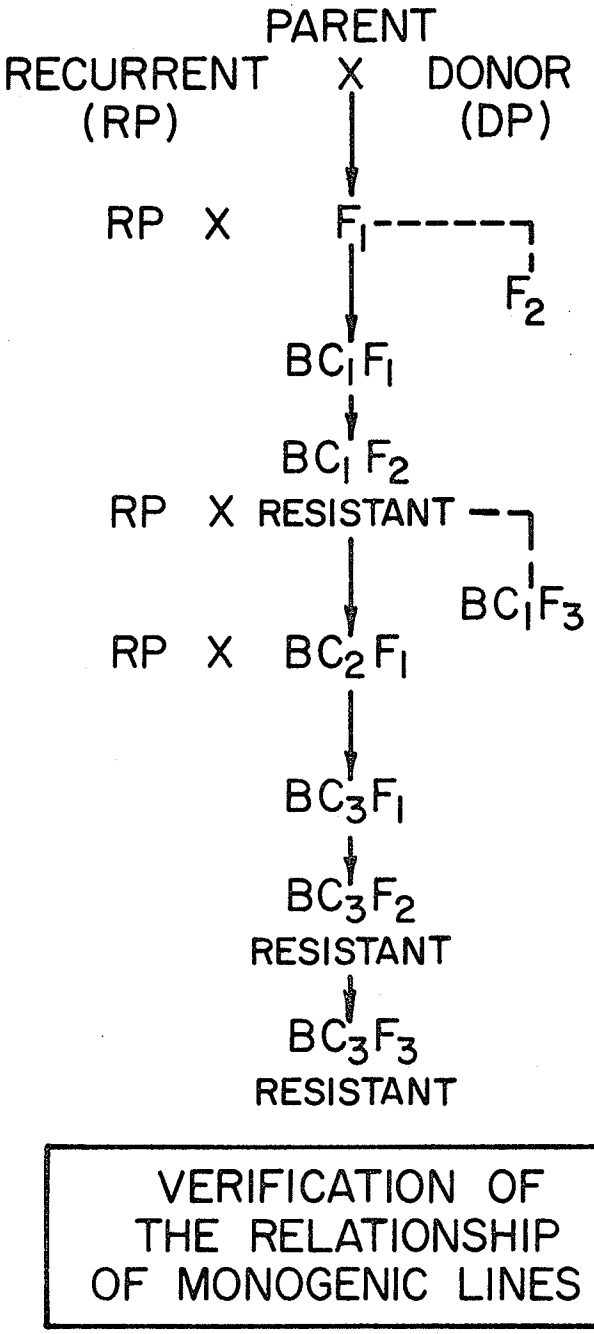
At PBS the crosses were usually made in the bird-proof wire cages or in the growth chambers. At the University of Manitoba all the crosses were done during the winter in heated greenhouses.

3.3. PATHOLOGICAL

3.3.1 STEM RUST INOCULA USED

The pure cultures of races EA4(295/1/3/4), EA5(34/2/5/7/8),

Table 3:4 The Working Schema Followed to Study the Mode of Inheritance, to Isolate and Identify Monogenic Lines



MODE OF INHERITANCE STUDIES

MODE OF INHERITANCE STUDIES AND START TO ISOLATE MONOGENIC LINES

PROGENY TESTS

ISOLATION OF MONOGENIC LINES CONTINUED

ISOLATION OF MONOGENIC LINES CONTINUED

ISOLATION OF MONOGENIC LINES CONTINUED

CROSSED HOMOZYGOUS MONOGENIC LINES AMONG THEMSELVES, TO H62 AND TO ALL THE Sr TESTER STOCKS

Recurrent Parent – H62

Donor Parents – KHR, KLD, TPY, TBR, CIF, CLY WIS and MIN

EA7(40/1/5/6), EA8(40/1/3/4/5/6) were originally provided by PBS. Races C10(15B-1) and C17(56) cultures were obtained from Canada Department of Agriculture Research Station, Winnipeg (CDA), through the courtesy of Dr. G. J. Green. These races were increased on seedlings and on adult plants of the varieties Florence Aurore and Little Club in isolated greenhouse compartments, at PBS and CDA respectively. TABLE 3:5 lists the East African and Canadian races used and their virulence formulae. Their "International" race equivalents are indicated.

Referring to TABLE 3:5 the races are given East African (EA) or Canadian (C) designations after the system developed by Green (1969) for wheat stem rust. The resistant varieties in the virulence formula are given in the numerator while those in the denominator are susceptible.

Only races with a broad virulence range or prevalent in the fields when this project was started were included in the present studies.

3.3.2 THE PURITY OF THE PRIMARY INOCULA

The original inocula were tested on the differential set and were found pure. The four EA races were maintained on Florence Aurore at PBS while the two Canadian races were maintained on Little Club at CDA Research Station. Pots of the susceptible host plants were grown and inoculated in different greenhouse chambers to prevent contamination. Strict sanitary precautions were adhered to at the time of inoculation, collection and storage of the inocula. The purity for each race was ascertained by inoculating the differential set of varieties and the parental varieties

TABLE 3:5 KEY, RACE NUMBERS AND FORMULA NUMBERS FOR RACES OF STEM RUST IN EAST AFRICA AND CANADA.

KEY	Virulence formula	Formula number	Standard race
EAST AFRICA *1			
Reliance susceptible			
Kota resistant	2,4,6,8,9	EA4	295/1/3/4
H441 susceptible	1,3,5,7		
Kota susceptible			
Einkorn resistant	3,4,5,7,8,9	EA5	34/2/5/7/8
Vernal resistant	1,2,6		
Vernal susceptible			
H441 resistant	3,5	EA7	40/1/5/6
Gala susceptible	1,2,4,6,7,8,9		
H441 susceptible	3	EA8	40/1/3/4/5/6
	1,2,4,5,6,7,8,9		

continued .....

TABLE 3:5 continued

KEY	Virulence formula	Formula number	Standard race
CANADA *2			
Little Club susceptible Marquis susceptible Reliance susceptible Arnautka resistant	1,6,8,9a,9b,11,13 5, 7, 10, 15, 16	C17	56
Arnautka susceptible Mindum susceptible Vernal susceptible Lee susceptible Golden Ball resistant Selkirk resistant	6,7,8,GB 1,5,9a,9b,10,11,13,14,15,16	C10	15B-1

Differential cultivars

*1 East Africa: 1 = Reliance; 2 = Kota; 3 = Einkorn; 4 = Vernal; 5 = H441; 6 = Gala; 7 = Marquis-Sr11; 8 = Giza; 9 = 501/67.

*2 Canada: 1 = Sr1; 5 = Sr5; 6 = Sr6; 7 = Sr7; 8 = Sr8; 9a = Sr9a; 9b = Sr9b; 10 = Sr10; 11 = Sr11; 13 = Sr13; 14 = Sr14; 15 = Sr15; 16 = Sr16; GB = Golden Ball.

Sources: 1. Extracted from PBS Annual Report 1968 for EA races.
2. Key to the physiologic races of wheat stem rust isolated in Canada from 1944 to 1954.
3. Canadian Plant Disease Survey 1971.

(TABLE 3:3). The scores were always checked against the Key prepared by Green (1970).

The above procedure was repeated for all the tests conducted during the course of these investigations. The same race cultures were employed throughout the period of this study. This was possible because of the excellent storage facilities available. The inoculum was vacuum sealed in glass vials and stored either in liquid nitrogen or refrigerated at 2° to 4°C. Maximum storage periods never exceeded six months. There was no noticeable reduction in viability of the cultures nor was there any evidence of deviation from the expected reaction types on the parental varieties or on the differential set of varieties.

3.3.3 METHODS OF SEEDLING INOCULATION

Seedlings were inoculated, when the primary leaves were 7 to 10 centimeters long, by spraying with urediospores of pure cultures suspended in Bayol-D oil or Mobilsol-100 oil. They were then exposed to atmosphere for approximately twenty minutes to dry the excess oil. The pots were incubated in dew chambers for 20 to 24 hours where they were fogged with a fine spray of water and maintained in a saturated atmosphere. Following incubation, the pots were placed on the benches in the greenhouse where they remained until notes were taken on their rust reactions thirteen to fifteen days after inoculation. The benches had overhead, timed, fluorescent, supplementary lights. Night temperatures averaged 18°C and day temperatures ranged from 21°C to 24°C.

3.3.4 RECORDING OF THE PATHOLOGICAL OBSERVATIONS

Classification of infection types was in accordance with the international usage described and revised by Stakman et al. (1962). The actual categories used in these experiments are listed in TABLE 3:6.

TABLE 3:6 CLASSES OF HOST REACTION AND TYPES OF RUST INFECTION.

Symbols for infection type*	Valuation in terms of pathogen-host reaction	Pathogen-host reaction abbreviation
0;, 1^-	Highly resistant	VR
1, 1^+, 2^-	Resistant	R
2, 2^+, 3^-, 3^{cn}, x^-	Moderately resistant	MR
x^+, 3	Moderately susceptible	MS
3^+, 4^-, 4, 4^+	Susceptible	S

* 0; = no uredia are developed, hypersensitive flecks occur;

1 = uredia are very small and surrounded by sharp, hypersensitive, necrotic areas;

2 = the uredia are small to medium in size; hypersensitive areas present in the form of necrotic halos, surrounding green islands in the centre of which the uredia are usually located;

3 = uredia are of medium size and usually separate. Necrosis and hypersensitiveness are absent but chlorotic areas may surround the uredia, especially under unfavourable conditions.

4 = uredia are large and usually coalesce to form large irregular pustules.

x = plants are heterogeneous in their reaction.

The symbols $^-$, $^{\pm}$, $^+$ indicate quantitative variations in types of uredial infection.

The divisions between the different classes in the scale given in TABLE 3:6 are quite arbitrary. The justification of this procedure is that it facilitates a comparison between the relative resistance of two or more varieties. Except where otherwise specified plants with infection types less than the standard 3 type including 3^- and x^- type pustules were considered as resistant and others as susceptible.

3.4 TESTS FOR GOODNESS OF FIT

The Karl Pearson's Chi-square method was extensively used to test the validity of hypothetical population ratios. Yates correction factor was used whenever class populations were less than 30 plants.

4. EXPERIMENTAL PROCEDURE FOR INDIVIDUAL EXPERIMENTS

4.1 MODE OF INHERITANCE

In this experiment the parental varieties were examined for the number of genes each possess which confer resistance against the races to which each variety is resistant.

4.1.1 THE METHOD OF STUDY

The eight donor parents were each crossed to the susceptible recurrent parent Hindi 62. One-third of the F_1 seeds from each cross were space planted in the bird-proof wire cage to raise adequate F_2 seeds for a reliable inheritance analysis. These plants were also used to backcross to Hindi 62 to produce the BC_1F_1. The remaining two-thirds of the F_1 seeds were used for testing to the six tester races (approximately 10 to 17 F_1 seeds per race).

4.1.1.a Handling F_2 generations

In order to test large F_2 progenies at the PBS, plants were grown in beds 3.1 by 1.1 metres. Rows were spaced 10 centimetres apart. 100 to 120 seeds were planted in each row. All the seedlings were inoculated at the one leaf stage using a slight modification of the standard procedure described in section 3.3.3. The beds were covered for 24 hours with a polyethylene sheet to serve as an incubation chamber. During this period the polyethylene and the seedlings were given a fine spray of water periodically to maintain high relative humidity. At the CDA Research Station, the plants were treated as described in section 3.3.3.

4.1.1.b Handling the BC_1F_1 plants.

The backcross F_1 plants were space planted in the greenhouse of the University of Manitoba during the winter 1969. The minimum number of BC_1F_1 plants which were raised were calculated according to the method described by Hanson (1959) with five to one per cent probabilities for failing to observe a genotype segregating for at least two gene difference. Usually many more plants were planted than required from the calculations. TABLE 4:7 shows the cross, generations and the plant or family numbers used for each race-test.

4.1.1.c Planting and inoculating of F_2 backcross families (BC_1F_2).

Progenies of individual BC_1F_1 plants were planted in pots to test seedling reactions. The BC_1F_2 seeds from each family were divided into as many portions as the number of races to which the donor parent was originally resistant. Whenever the quantity of seed was inadequate, only the most important races were given priority. The identity of each family was kept and the ratio of resistant to susceptible plants in each progeny was determined.

In some cases, in order to save time, space, seed and to isolate the genes most effectively, the same plants were tested with two races of rust. At the first leaf-stage, the seedlings were inoculated with the first race and seven to eight days later, at the time flecking began, the same seedlings were inoculated with the second race. After thirteen to fifteen days the first infection was read and the rusted leaves removed before the second infection developed. A week later the second infection

TABLE 4:7 CROSSES, GENERATION, POPULATIONS AND TEST RACES.

CROSS	GENERATION	POPULATION					
		Test Races					
		EA4	EA5	EA7	EA8	C10	C17
H62 x KHR	F_1	17	-	-	-	-	17
	F_2	3248	-	-	-	-	875
$H62^2$ x KHR	BC_1F_2 Families	65*	-	-	-	-	71
H62 x KLD	F_1	-	16	18	-	-	17
	F_2	-	727	596	-	-	1288
$H62^2$ x KLD	BC_1F_2 Families	-	52	65	-	-	40
H62 x TPY	F_1	-	16	-	17	-	16
	F_2	-	637	-	581	-	896
$H62^2$ x TPY	BC_1F_2 Families	-	53	-	52	-	65
H62 x TBR	F_1	-	15	16	-	-	16
	F_2	-	637	970	-	-	554
$H62^2$ x TBR	BC_1F_2 Families	-	50	46	-	-	32
H62 x CLY	F_1	-	18	17	-	-	16
	F_2	-	712	1109	-	-	486
$H62^2$ x CLY	BC_1F_2 Families	-	61	56	-	-	37
H62 x CIF	F_1	17	16	-	-	17	17
	F_2	3248	870	-	-	487	1144
$H62^2$ x CIF	BC_1F_2 Families	54	53	-	-	48	60
H62 x MIN	F_1	14	-	-	-	-	16
	F_2	1120	-	-	-	-	570
$H62^2$ x MIN	BC_1F_2 Families	47	-	-	-	-	46
H62 x WIS	F_1	-	-	18	-	-	-
	F_2	-	-	1040	-	-	-
$H62^2$ x WIS	BC_1F_2 Families	-	-	46	-	-	-

* The average population within each BC_1F_2 family was 38 plants.

was read. No evidence was found to indicate that the second infection interferred adversely with the first infection. The differential varieties and the parental varieties produced their typical reactions to the two races when inoculated in the same way. A method similar to this has been used by Knott et al. (1956) and Loegering et al. (1966).

In certain cases reciprocal inoculations were performed. Reciprocal tests involved dividing the BC_1F_1 progenies into at least two lots of seed. Then inoculating the first leaves of the first lot with one of the races and the second leaves with the other race. The race priority was reversed in the second batch of seed.

In general, very uniform rust infections were obtained. In the few cases where an '0' reaction type was recorded, the plants showing no infection were either reinoculated or raised to maturity and their progenies were re-evaluated as BC_1F_3 lines.

4.2 THE ISOLATION OF THE MONOGENIC LINES

4.2.1 MATERIAL AND METHOD

The material for this experiment was advanced from the BC_1F_2 families described in the previous section. A careful examination of the results from the doubly inoculated plants was conducted. Several plants from a single BC_1F_2 family which segregated for one race but was susceptible to the other were saved. Only those BC_1F_2 families which segregated in a 3:1 or 1:3 monogenic ratio were kept for the isolation exercise. These selected resistant BC_1F_2 plants were grown to maturity. In cases where

apparently a single gene conferred resistance to a number of test races the same procedure was followed. In all cases susceptible families were discarded.

In order to obtain monogenic lines which are essentially similar to the recurrent parent (H62) a number of backcrosses are essential. Consequently, resistant BC_1F_2 plants which resembled H62 were used to backcross to Hindi 62. The individual pollinators were selected from different BC_1F_2 families and pollen from a single plant was used to make each cross. The BC_1F_3 (non-crossed seeds) were used for a progeny test for resistance against the race or races involved to confirm the genetic ratios obtained from the F_2 backcross families. The BC_1F_2 plants which segregated in the BC_1F_3 together with their BC_2F_1 were not used for the next backcross but were kept as reserve seed stock. The BC_2F_1 seed from the homozygous BC_1F_2 plants were grown and a fourth cross to Hindi 62 (BC_3F_1) was made. Hindi 62 was always used as female parent and only the backcross progenies which resembled Hindi 62 were used in subsequent backcrosses.

The BC_3F_1 from at least seven different BC_1F_2 progenitors were tested. The resistant plants were raised to maturity. The BC_3F_2 seedlings were subsequently tested and the resistant plants raised to maturity. The resulting BC_3F_3 progenies were tested in the greenhouse and the resistant plants transplanted out in the field during the September-February out-of-season period. Three resistant BC_3F_3 from each of 6 to 17 BC_3F_2 homozygous resistant progenitors were retained.

On these apparently homozygous lines, a series of verification crosses were performed. An extra cross to H62 was conducted to ratify the monogeneity of the isolated genetic lines and to further reconstitute the H62 background. Eventually one plant progeny resembling Hindi 62 in most respects was retained to represent each isolated resistant gene.

The schedule for the selections performed at University of Manitoba was slightly different from the PBS programme. For the U of M material, the BC_3F_2 seed from every family was divided into two lots. The first lot was field planted at PBS where the plants resembling Hindi 62 were crossed to H62, to the Sr series, within themselves and between lines from other varieties which had identical resistance. The second lot of BC_3F_2 seed was tested with races C10 and C17 at the CDA, Research Station. From the results with the BC_3F_2 seedlings, all the BC_3F_3 generations including their crossed seed originating from segregating BC_3F_2 progenitors were dropped from further tests. The BC_3F_3 progeny plus the crossed (F_1) seeds emanating from homozygous BC_3F_2 plants were again grown at Njoro to obtain BC_3F_4 and F_2 seed stocks for monogenic line comparisons. In the PBS fields the BC_3F_3 were selected for resemblance to Hindi 62. The U of M selected lines were tested against East African races. During the winter months, February and March 1972, the BC_3F_4 lines together with the verification F_2 progenies were tested in the greenhouse at CDA Research Station. TABLE 4:8 illustrates the calendar of operations followed to isolate and to identify the monogenic lines.

Most of the monogenic lines were expected to be highly susceptible

TABLE 4:8 THE CALENDAR FOR MONOGENIC ISOLATION OPERATIONS.

Generation	Year	Season*	Operation
A. NJORO PROGRAMME:			
BC_1F_2	1968	Oct.-Feb. (C)	Mode of inheritance; Gene isolation; BC_2F_1 produced.
BC_2F_1	1969	Mar.-Jul. (C)	No seedling test; BC_3F_1 produced.
BC_3F_1	1969	Aug.-Feb. (C)	Seedling test; transplant resistant.
BC_3F_2	1970	May -Jul. (GC)	Seedling test; transplant resistant.
BC_3F_3	1970	Aug.-Feb. (C)	Seedling test; transplant resistant; cross homo. to H62, Sr lines, intragenic; intergenic lines.
	1971	Feb.-Aug. (C)	Verification crosses advanced to F_2.
	1971	Aug.-Dec.	Completed isolation; checked the identity; Sr relationship; studied effect of isolated genes to other races; tested U of M selected lines to EA. races.
B. U of M and CDA PROGRAMME:			
BC_1F_2	1969	Sep.-Dec. (G)	Seedling test; transplant resistant; BC_2F_1 produced.
BC_2F_1	1970	Feb.-Jun.	Planted at PBS; No test; BC_3F_1 produced.
BC_3F_1	1970	Sep.-Dec. (G)	Seedling test; transplant resistant at U of M.
BC_3F_2	1971	Jan.-May (C)	Planted at PBS; seedling test; crossed to H62, Sr lines, intragenic and intergenic.
BC_3F_3	1971	Jun.-Nov.	Planted at PBS, seedling test to EA races; advanced verification crosses to F_2.
BC_3F_4	1972	Feb.-Mar. (G)	Completed isolation; checked the identity; Sr relationship; studied effect of isolated genes to other races; tested Kenya selected lines to Canadian races.

* Letters in parenthesis indicate where the generation was raised to maturity.

(C) = Bird-proof Cage; (G) = Greenhouse; (GC) = Growth Cabinet.

to a number of the races in the field. Therefore, fortnightly sprayings with "Dithane" fungicide was commenced after the tillering stage.

4.3. IDENTIFICATION OF THE ISOLATED MONOGENIC LINES

4.3.1 RELATIONSHIP WITH THE IDENTIFIED STEM RUST GENES

The established stem rust (Sr) genes designated Sr1, Sr5, Sr6, Sr7, Sr8, Sr9a, Sr9b, Sr10, Sr11, Sr13, Sr14, Sr15, Sr16 and sr17 were obtained from Dr. G. J. Green's differential seed stock. These lines were included in all the tests as a check on the purity of the races. Their infection to the six races used are tabulated in TABLE 4:9.

When the relationship studies were conducted, the Sr infection types were carefully compared with the infection types recorded from the parents given in TABLE 3:3. Only those Sr gene lines that exhibited resistant reactions similar to the parent variety or comparable to the isolated monogenic lines were used in the crosses for the present investigations.

The technique employed to obtain closely related and comparable hybrids was to select three well tillered plants of the homozygous lines (BC_3F_3 lines for PBS material and BC_3F_2 lines for the U of M material). As many heads as there were numbers of Sr lines showing resistance to the same race were emasculated. The pollen was obtained from a single plant in most cases.

4.3.2. COMPARISON OF MONOGENIC LINES FROM THE SAME VARIETY

The identity of intra-varietal genes was initially done with the BC_1F_2 families. The plants of each family were inoculated with at least

TABLE 4:9 INFECTION TYPES PRODUCED ON THE 14 Sr LINES BY 6 STEM RUST RACES.

Sr Line	Infection Type*1					
	EA4(295)	EA5(34)	EA7(40)	EA8(40)	C10(15B-1)	C17(56)
1	4	3	4	4	4	2
5	3^{+}	4^{-}	4	4	4	4
6	3^{+}	3^{+}	4	3^{+}	;1	;1
7	3^{+}	4	4	4	;1	4
8*2	3^{+}	3^{+}	3	4	3^{+}	2^{+}
9a	4^{-}	4	4	4	4	2
9b	4	4	3	4	4^{-}	2
10	3^{+}	4^{-}	4	4	3^{+}	4
11	3^{+}	0;	4	4	3^{+}	;1
13	$2^{\pm}$	2	3^{+}	3^{+}	4	2^{+}
14	3^{+}	4	3^{+}	3^{+}	4	$2^{+}-3$
15^{x}	4	x^{-}	4	4	4	4
16	3^{+}	3^{+}	4	4	4	4
17^{x}	4	;1	4	4	4	;1

*1 Figures in parenthesis denote the International "standard" races.

x Not in Marquis background.

*2 Normal infection type for Sr8 is 2^{+} to race C10(15B-1).

two different races as described in section 4.1.1.c. If the gene(s) for resistance to one race segregated independently of the gene(s) for the other race, then the two genes were different from one another. But in cases where a single gene conferred resistance to two or more races, families which segregated for one, also segregated for the other, and individual plants reacted similarly to both races. This was conclusive evidence that the same gene(s) gave resistance to both test races.

In the cases where BC_1F_2 families were not conclusive, the BC_3F_3 homozygous lines were compared with the Sr lines as described in the previous section 4.3.1. F_3 lines which did not segregate when crossed to the same Sr gene line when inoculated with the same race were classified as identical genes. Then there were others (BC_3F_3 lines) which defied all the above tests. These were intercrossed. The resulting F_2 progenies were inoculated with the race(s) to which the parental lines were resistant. Those BC_3F_3 lines which segregated were classified as being different while those which bred true for resistance were classified as being identical genes.

Another criterion to the identity of intra-varietal genes was the mode of inheritance. Those genes which displayed a recessive mode of inheritance were usually classified as being different from those showing a dominant resistance gene.

4.3.3 COMPARISON BETWEEN INTERVARIETAL GENES

The remaining BC_3F_3 lines, after identifying those which were identical to the established Sr gene lines and after regrouping the intra-

varietal gene lines, were crossed. TABLE 5:39 shows the lines which were compared. Here again recessiveness and dominance of the resistant genes were considered when making the crosses. A monogenic line derived from a parent which was susceptible to a certain race was not crossed to a monogenic line whose progenitor was resistant to the same race. Only those monogenic lines derived from parents conveying resistance to the same race or races were crossed for comparison. Nevertheless, all the BC_3F_3 lines from all sources were tested to all the tester races.

4.4 THE INFECTION TYPES PRODUCED ON MONOGENIC LINES BY RACES OTHER THAN THE PRIMARY SIX RACES

The range of resistance of the retained monogenic lines was determined by inoculating them with six additional East African races: EA11(40); EA12(40); EA13(34); EA14(11); EA15(11) and EA16(34). Cultures of seven other North American races: C1(17); C2(17A); C18(15B-1L); C20(11); C22(32); C25(38) and C41(32-113) were also used. These races were selected because of their increasing frequency in the rust survey collections in recent years. The EA races were obtained from PBS while the Canadian races were from CDA, Winnipeg Research Station.

On the basis of the information obtained regarding the identity of the isolated lines, only the lines which were apparently unidentified previously were tested to these additional races. Hindi 62 was also included in the tests. In cases where H62 was resistant to a race, then the monogeneity of the line or the parent that contributed the resistance gene to that particular race(s) would be doubted. The monogenic lines

and Hindi 62 were seeded in a concentric order similar to the planting pattern of race differential set of varieties. Two such pot-plantings were repeated for every race.

5. RESULTS

5.1 THE MODE OF INHERITANCE

5.1.1 INHERITANCE STUDIES - KENYA HUNTER x HINDI 62

Kenya Hunter is resistant only to races EA4 and C17, therefore only these two races were tested against the segregating populations. In TABLE 5:10 is presented the parental, F_1, F_2, BC_1F_2 and BC_1F_3 seedling reactions of KHR x H62 crosses to race EA4.

TABLE 5:10 STEM RUST REACTIONS TO RACE EA4 OF PARENTS AND SEEDLINGS OF KENYA HUNTER CROSS AND BACKCROSS TO HINDI 62.

Generation or Parent	Breeding behaviour			Expected	Chi-	P-
	R	Seg	S[x]	Ratio	Square	Value
Hindi 62 (H62)	-	-	S	-	-	-
Kenya Hunter (KHR)	R	-	-	-	-	-
H62 x KHR						
F_1	17	0	0	-	-	-
F_2	2428	-	820	3:1	0.105	.80-.70
$H62^2$ x KHR						
F_2 families	0	37	28	1:1	1.538	.30-.20
F_3 lines*	5	12	0	1:2	0.007	.95-.90

The ratio within the 37 segregating BC_1F_2 families was 912R:316S for a 3:1 ratio; P = .50-.30. The ratio within BC_1F_3 seg. families was 566R:193S for a 3:1 ratio; P = .80-.70.

[x] R = Resistant: Seg. = Segregating; S = Susceptible.

* BC_1F_3 lines are progenies of resistant BC_1F_2 plants.

Dominance in the F_1 generation for resistance appeared complete. The F_2 populations fitted the expected 3:1 ratio for a single dominant gene for resistance. Segregation in the BC_1F_2 and BC_1F_3 generations also produced the expected monogenic ratio. The results verify that a single dominant factor governs the resistance of Kenya Hunter to EA4 in the seedling stage.

Seedling reactions to race C17(56) were, with very few exceptions, definite and easy to classify. The resistant segregates produced 0;, 1 and 2^- pustule types while the susceptibles had 3^+ to 4 infection types. All the plants of the donor parent (KHR) consistently produced 0; or ;1 and H62, 4 or 4^- type pustules.

Reactions to C17(56) are presented in TABLE 5:11.

TABLE 5:11 STEM RUST REACTIONS TO RACE C17(56) OF PARENTS AND SEEDLINGS OF KENYA HUNTER CROSS AND BACKCROSS TO HINDI 62.

Generation or Parent	Breeding behaviour			Expected	Chi-	P-
	R	Seg	S	Ratio	Square	Value
Hindi 62 (H62)	-	-	S	-	-	-
Kenya Hunter (KHR)	R	-	-	-	-	-
H62 x KHR						
F_1	17(MR)	0	0	-	-	-
F_2	674	-	201	3:1	1.920	.20-.10
$H62^2$ x KHR						
F_2 families	0	36	35	1:1	0.014	.95-.90
F_3 lines	6	15	0	1:2	0.054	.90-.75

The ratio within segregating families were:

BC_1F_2 families; 1336R:436S P-value for a 3:1 ratio = 0.70

BC_1F_3 lines; 1321R:470S P-value for a 3:1 ratio = .30 to .20

The distribution of F_2 plants suggested the operation of a single major factor pair in Kenya Hunter governing resistance to race C17(56). Since the F_1 seedlings showed a moderately resistant ;2 pustule type, the gene is only partially dominant. Family distribution in the BC_1F_2 generation and BC_1F_3 lines selected from resistant BC_1F_2 plants clearly confirm the single gene inheritance.

The results of the reciprocal tests on the F_2 families from the backcross of KHR and H62 suggested independent segregation of the factors for resistance to EA4 and C17(56). Four groups of lines were observed: 13 lines segregated for both the races; 17 lines segregated for race EA4 but were susceptible to race C17(56); 9 lines segregated for race C17(56) but were susceptible to EA4. 13 other lines were completely susceptible to both races. This 13:17:9:13 proportion fitted the 1:1:1:1 ratio expected of a digenic inheritance postulated for F_2 backcross families. The calculated P-value for the Chi-square goodness of fit was .20 to .10.

These mode of inheritance studies reveal that Kenya Hunter possesses two major genes for resistance against races EA4 and C17(56). The one giving resistance to EA4 is being isolated into Hindi 62 background. (See sections 4.2 and 5.2 for methods and the results pertaining to the isolation of monogenic lines).

5.1.2 INHERITANCE STUDIES - KENYA LEOPARD x HINDI 62

Kenya Leopard crosses were tested against the three races EA5, EA7 and C17.

In tests with EA5, (TABLE 5:12), the Chi-square test of goodness of

fit for F_2 populations conformed to a 3:1 ratio. The F_1 observations indicated that resistance was not completely dominant. The breeding behaviour of the BC_1F_2 plants and BC_1F_3 lines from the resistant BC_1F_2 both substantiate a single factor inheritance.

TABLE 5:12 STEM RUST REACTIONS TO RACE EA5 OF PARENTS AND SEEDLINGS OF KENYA LEOPARD CROSS AND BACKCROSS TO HINDI 62.

Generation or Parent	Breeding behaviour			Expected	Chi-	P-
	R	Seg	S	Ratio	Square	Value
Hindi 62 (H62)	-	-	S	-	-	-
Kenya Leopard (KLD)	R	-	-	-	-	-
H62 x KLD						
F_1	16	0	0	-	-	-
F_2	552	-	175	3:1	0.334	.75-.50
$H62^2$ x KLD						
F_2 families	0	25	27	1:1	0.019	.95-.90
F_3 lines	7	17	0	1:2	0.047	.90-.80

The ratio within segregating BC_1F_2 families: 529R : 157S; P value for 3:1 was .30 to .20. Within BC_1F_3 lines: 1020R : 340S; P value for 3:1 was .95-.90.

Kenya Leopard is highly resistant (;1) to EA7, whereas the recurrent parent Hindi 62 gave 3^+ to 4 reaction types. The F_1 plants were not as resistant as Kenya Leopard. They exhibited 1^+ to ;2 infection types. Therefore the Kenya Leopard gene is either incompletely dominant or less effective under the influence of Hindi 62 background. The infected seedling leaves were characterized by early senescence. However in the F_2

generation, some of the resistant plants exhibited infection types identical to Kenya Leopard (;1, pustule type), while others were moderately resistant, (;2), like the heterozygous F_1 plants.

The F_2 populations segregated into 458R : 138S (TABLE 5:13). This fitted satisfactorily the postulated 3:1 ratio with a P value of .50 to .25. The observed proportion shows a higher number of plants in the resistant class than expected. This might be accounted for by the fact that some withered plants which were classified as resistant might have been susceptible. This was found true at least in one of the BC_1F_2 plants which had been classified as resistant but proved to be homozygous susceptible in the BC_1F_3 tests. The breeding behaviour of BC_1F_2 and BC_1F_3 (TABLE 5:13) further confirm the one gene hypothesis.

TABLE 5:13 STEM RUST REACTIONS TO RACE EA7 OF PARENTS AND SEEDLINGS OF KENYA LEOPARD CROSS AND BACKCROSS TO HINDI 62.

Generation or Parent	Breeding behaviour			Expected	Chi-	P-
	R	Seg	S	Ratio	Square	Value
Hindi 62 (H62)	-	-	S	-	-	-
Kenya Leopard (KLD)	R	-	-	-	-	-
H62 x KLD						
F_1	18	0	0	-	-	-
F_2	458	-	138	3:1	1.083	.50-.25
$H62^2$ x KLD						
F_2 families	0	33	32	1:1	0.019	.95-.90
F_3 lines	5	13	0	1:2	0.062	.80-.70

Within the BC_1F_2 seg. families: 495 : 163S; P value of .95 to .90 for a 3:1 ratio. Within BC_1F_3 seg. families: 1272R : 402S; P value .50 to .30 for a 3:1 ratio.

Since some of the F_2 plants and the homozygous BC_1F_3 plants displayed resistant infection types similar to Kenya Leopard, it might be inferred that the moderate resistance observed in F_1 was not due to the Hindi 62 background. Probably the Kenya Leopard gene was less effective in a heterozygous condition. It is most likely to be a partially dominant gene.

The genetic data presented in TABLE 5:14 show that Kenya Leopard had one partially dominant gene for resistance against race C17(56). The BC_1F_2 and BC_1F_3 families fitted satisfactorily the proposed monofactorial inheritance even though the F_2 ratio was not in good agreement.

TABLE 5:14 STEM RUST REACTIONS TO RACE C17(56) OF PARENTS AND SEEDLINGS OF KENYA LEOPARD CROSS AND BACKCROSS TO HINDI 62.

Generation or Parent	Breeding behaviour			Expected Ratio	Chi-Square	P-Value
	R	Seg	S			
Hindi 62 (H62)	-	-	S	-	-	-
Kenya Leopard (KLD)	R	-	-	-	-	-
H62 x KLD						
F_1	17	0	0	-	-	-
F_2	927	-	364	3:1	6.683	Less .01
$H62^2$ x KLD						
F_2 families	0	20	20	1:1	-	1
F_3 lines	7	17	0	1:2	0.047	.90-.80

Within the F_2 backcross seg. families the ratio was 697R : 226S for a 3:1 ratio with a P value of .80 - .70. Within seg. BC_1F_3 lines: 1245R : 456S P value = .10 - .05 for 3:1 ratio.

Infection types recorded on the resistant seedlings varied from ;1 to ;2^{+}_{-}. It was assumed that plants giving the ;1 were homozygous for resistance while the ;2^{+}_{-} were heterozygous. Hence most of the backcross operations to isolate the genes were performed on the transplanted plants that had given ;1 infection type.

Kenya Leopard - Hindi 62 backcross families were simultaneously tested to the three experimental races EA5(34), EA7(40) and C17(56). The results (TABLE 5:15) suggest that the same gene confers resistance to both races EA5 and C17(56) because every family that segregated for resistance to one of the races also segregated for the other. Similarly susceptible families for EA5 were also susceptible to C17(56). Resistance to EA7 appeared to segregate independent of the gene for both EA5 and C17(56).

TABLE 5:15 THE COMBINED RESULTS FOR RACES EA5, EA7 AND C17 FOR KENYA LEOPARD x HINDI 62^{2} (BC_1F_2).

		RACES EA5 AND C17		TOTAL	P-Value
		Seg. (3:1)	Susceptible		
RACE EA7	Seg. (3:1)	24	4	28	.80-.70
	Susceptible	11	14	25	
	TOTAL	35	18		
	P-value (1:1)	.05-.01			

From TABLE 5:15 it is noteworthy that out of 53 BC_1F_2 families, 38 gave the parental phenotypes, that is, they either segregated simultaneously to the three races or were susceptible. Only fifteen families displayed some recombinant phenotypes. This deviation from the expected ratio of 1 parental : 1 recombinant phenotypes indicates that the two genes responsible for resistance against races EA5, EA7 and C17 are located on the same chromosome, and linkage is in the coupling phase. A linkage value of 28.3 per cent was estimated from the BC_1F_2 data in TABLE 5:15.

The two resistance genes against races EA4, EA7 and C17(56) predicted for Kenya Leopard are both isolated in the Hindi 62 background.

5.1.3 INHERITANCE STUDIES - TROPHY x HINDI 62

Trophy is a Mexican wheat variety which is used extensively in the Kenya breeding programme and has accounted for more than 50 per cent of wheat acreage in Kenya in 1971. Trophy is highly resistant (;1) to race EA5, moderately resistant $2^{\pm}$ to EA8 and very resistant to C17.

The inheritance proved to be simple when tested with race EA5 (TABLE 5:16). The F_1 populations displayed a hypersensitive reaction with brownish necrotic lesions around the tiny pustules. The F_2 segregation, the BC_1F_2 and BC_1F_3 breeding behaviour all verify a single dominant gene as responsible for resistance to EA5.

As indicated in TABLE 5:17, Trophy produced a moderately resistant $2^{\pm}$ reaction when infected by EA8. F_1 plants were difficult to classify. They showed 3 or x^{+} infection type which is regarded as susceptible indicating a recessive resistance.

TABLE 5:16 THE STEM RUST REACTIONS TO RACE EA5 OF PARENTS AND SEEDLINGS OF TROPHY CROSS AND BACKCROSS TO HINDI 62.

Generation or Parent	Breeding behaviour			Expected	Chi-	P-
	R	Seg	S	Ratio	Square	Value
Hindi 62 (H62)	-	-	S	-	-	-
Trophy (TPY)	R	-	-	-	-	-
H62 x TPY						
F_1	16	0	0	-	-	-
F_2	285	-	152	3:1	0.440	.70-.50
$H62^2$ x TPY						
F_2 families	0	33	32	1:1	0.016	.95-.90
F_3 lines	6	12	0	1:2	-	1.

Within F_2 backcross families seg.: 515R : 182S; P value .50 - .30 for a 3:1 ratio. Within F_3 backcross lines seg.: 851R : 290S; P value .80 - .70 for a 3:1 ratio.

TABLE 5:17 THE STEM RUST REACTIONS TO RACE EA8 OF PARENTS AND SEEDLINGS OF TROPHY CROSS AND BACKCROSS TO HINDI 62.

Generation or Parent	Breeding behaviour			Expected	Chi-	P-
	MR	Seg	S	Ratio	Square	Value
Hindi 62 (H62)	-	-	S	-	-	-
Trophy (TPY)	MR	-	-	-	-	-
H62 x TPY						
F_1	0	0	17	-	-	-
F_2	127	0	454	1:3	4.551	.05-.01
$H62^2$ x TPY						
F_2 families	0	24	28	1:1	0.308	.70-.50
F_3 lines	11	0	0	-	-	-

Within segregating BC_1F_2 families 60MR : 233S; P-value .10 to .05 for a 3:1 ratio.

Some F_2 plants exhibited a $2^{\pm}$ infection type like the resistant parent Trophy. Others had intermediate infection types 23, ;3, 3^{cn} and x^{-}. Classification of this population presented a problem. However, all the above infection types were classified as a resistant group. On this basis the F_2 segregation satisfied a single recessive factor inheritance for resistance.

Segregation among the BC_1F_2 families conclusively supported the single gene inheritance. The behaviour of the F_3 backcross lines selected from resistant BC_1F_2 plants confirmed the hypothetical single recessive gene (all the lines were homozygous although they were less resistant than the donor parent Trophy). These results also suggest the presence of minor gene(s) which enhance the resistance of Trophy to EA8.

The present investigations indicate that Trophy is a good source of resistance to the North American race C17(56). The host-pathogen reaction type is a fleck. The F_1 plants were all moderately resistant with infection ;2 type pustules, thus indicating incomplete dominance inheritance. The genetic analytical data given in TABLE 5:18 shows that the inheritance to race C17(56) is governed by a single gene.

F_2 populations indicated that a single gene conferred resistance to C17(56). The resistant F_2 seedlings exhibited a variation of infection type $;1^{-}$ like the donor parent or $2^{\pm}$ like the heterozygous F_1 population. Data from the BC_1F_2 and BC_1F_3 families selected from the resistant BC_1F_2 progenitors fitted satisfactorily a monogenic inheritance postulated for Trophy.

TABLE 5:18 THE STEM RUST REACTIONS TO RACE C17(56) OF PARENTS AND SEEDLINGS OF TROPHY CROSS AND BACKCROSS TO HINDI 62.

Generation or Parent	Breeding behaviour			Expected	Chi-	P-
	R	Seg	S	Ratio	Square	Value
Hindi 62 (H62)	-	-	S	-	-	-
Trophy (TPY)	R	-	-	-	-	-
H62 x TPY						
F_1	16	0	0	-	-	-
F_2	678	-	218	3:1	0.214	.70-.50
$H62^2$ x TPY						
F_2 families	0	33	32	1:1	0.016	.95-.90
F_3 lines	6	13	0	1:2	0.006	.95-.90

Ratios within segregating families were as follows:

BC_1F_2 969R : 331S P for 3:1 ratio - .70

BC_1F_3 1176R : 402S P for 3:1 ratio = .70 - .50

The 33 BC_1F_2 families that segregated in Canada for C17(56) also segregated for EA5 in Kenya while 32 families were completely susceptible to both races. It is most probable that the same gene in Trophy is responsible for resistance to both C17 and EA5 (TABLES 5:16 and 5:18). The recessive gene effective against EA8 assorted independently of the former gene.

These results indicate that Trophy possesses at least two genes for resistance against races EA5, EA8 and C17(56). Attempts were made to isolate all the resistance to all three races in Hindi 62 through a series of backcrosses.

5.1.4 INHERITANCE STUDIES - TOBARI 66 x HINDI 62

Tobari 66 is another Mexican variety that combines high yield and good resistance. It consistently produced infection types ;1 to race EA5, 1 to 2 to race EA7 and a 0; infection type to race C17(56).

The 15 F_1 plants were as resistant as Tobari 66. Resistance to EA5 is therefore controlled by a completely dominant gene (TABLE 5:19). The F_2 segregation indicates a monofactorial mode of inheritance. Again for Tobari 66 as for Kenya Leopard against EA5 there was a slight deficiency of the resistant plants. However the BC_1F_2 segregation confirmed the monofactorial inheritance. Within segregating families the fit to the expected 3:1 ratio was below the .05 probability level. The discrepancy cannot be attributed to the chance error alone. While, there is strong evidence for a single major gene determining resistance in Tobari 66, it is probable that some modifiers effect the expressivety of the major gene and tend to

distort the expected ratio.

TABLE 5:19 THE STEM RUST REACTIONS TO RACE EA5 OF PARENTS AND SEEDLINGS OF TOBARI 66 CROSS AND BACKCROSS TO HINDI 62.

Generation or Parent	Breeding behaviour			Expected Ratio	Chi-Square	P-Value
	R	Seg	S			
Hindi 62 (H62)	-	-	S	-	-	-
Tobari 66 (TBR)	R	-	-	-	-	-
H62 x TBR						
F_1	15	0	0	-	-	-
F_2	475	-	162	3:1	0.063	.80-.70
$H62^2$ x TBR						
F_2 families	0	25	25	1:1	-	1
F_3 lines	5	12	0	1:2	0.007	.95-.90

Ratio within segregating families were:

BC_1F_2 families: 529R : 219S P-value .05-.01 for a 3:1 ratio

BC_1F_3 lines: 809R : 287S P-value .50-.30 for a 3:1 ratio.

When the BC_1F_3 lines randomly selected from resistant BC_1F_2 families, were tested, the observed 5R : 12 Seg. fitted to the expected 1R : 2 Seg. ratio well.

Tobari 66 is moderately resistant (1 to 2) to race EA7. Hindi 62 is very susceptible (4) to the same race. TABLE 5:20 gives the summary of the genetic inheritance results obtained by crossing the two varieties.

The F_1 plants showed susceptibility 3 to 3^+ infection types to race EA7.

TABLE 5:20 THE STEM RUST REACTIONS TO RACE EA7 OF PARENTS AND SEEDLINGS OF TOBARI 66 CROSS AND BACKCROSS TO HINDI 62.

Generation or Parent	Breeding behaviour			Expected Ratio	Chi-Square	P-Value
	MR	Seg	S			
Hindi 62 (H62)	-	-	S	-	-	-
Tobari 66 (TBR)	MR	-	-	-	-	-
H62 x TBR						
F_1	0	0	16	-	-	-
F_2	213	-	757	1:3	1.141	.30-.20
$H62^2$ x TBR						
F_2 families	0	25	21	1:1	0.196	.70-.50

Within BC_1F_2 segregating families the ratio was 195R : 285S; P for 1:3 = .05 - .01.

Classification into resistant and susceptible categories in the F_2 generation was complicated. Whenever the mean temperature in the greenhouse was above normal, that is, more than 30^oC, the plants gave 3^+ to 4 infection types. According to the standards set in the present studies plants giving 3^+ were classified as susceptible. TABLE 3:6 shows the standards adopted to delineate resistant from the susceptible plants. The results in TABLE 5:20 were recorded under normal Njoro greenhouse temperatures (diurnal mean of 22^oC). The distribution of the F_2 population fitted

the expected 1R:3S ratio for a recessive gene.

Forty-six F_2 backcross families tested to EA7 segregated in a manner that confirmed the single gene hypothesis. The 25 segregating families fitted poorly (P - value of .05 to .01) the expected 1R:3S ratio. The poor fit suggests the operation of some other additional factor or factors modifying the major gene.

Attempts to study the twenty-one BC_1F_3 lines proved futile when all of them were susceptible. This might be explained by assuming that the Tobari 66 gene is temperature sensitive. A hot wave that prevailed at Njoro in October, 1971, during the fourteen day period allowed for infection to develop, might have influenced the reaction. The Hindi 62 background cannot be precluded totally as an influencing factor on the penetrance and the expressivity of Tobari 66 resistance gene against EA7.

Tobari 66 is highly resistant (0;) to race C17(56). The hypersensitivity is characterized on seedlings by greyish necrotic areas which surround the pustules.

The F_1 plants were resistant with ;2^- pustule types. This indicated that the resistance was somewhat reduced in a heterozygous condition or by the genetic background of the susceptible parent Hindi 62. The F_2 generation segregated into approximately 15R : 1S plant classes. Two dominant duplicate gene inheritance was inferred from this data. With reference to TABLE 5:21, the pattern of breeding behaviour between the F_2 backcross families confirmed the digenic mode of inheritance: 24 families segregated while 8 families were fully susceptible. This distribution fits

TABLE 5:21 THE STEM RUST REACTIONS TO RACE C17(56) OF PARENTS AND SEEDLINGS OF TOBARI 66 CROSS AND BACKCROSS TO HINDI 62.

Generation or Parent	Breeding behaviour			Expected Ratio	Chi-Square	P-Value
	R	Seg	S			
Hindi 62 (H62)	-	-	S	-	-	-
Tobari 66 (TBR)	VR	-	-	-	-	-
H62 x TBR						
F_1	16	0	0	-	-	-
F_2	521	-	33	15:1	0.081	.80-.70
$H62^2$ x TBR						
F_2 families	0	24	8	3:1	-	1

Within the BC_1F_2 families segregation was:

16 families seg. 376R : 104S P = .10 - .05 for 3:1 ratio

8 families seg. 269R : 22S P = .50 - .30 for 15:1 ratio.

perfectly the expected 3:1 for BC_1F_2 ratio. Furthermore the distribution of the 24 segregating families was 8 segregating into approximately 15R : 1S and 16 families segregating 3R : 1S. This observed proportion fits a digenic test-cross ratio of 1:1:1:1 when the two non-parental genotypes are pooled, (that is, when the 8 susceptible families are included in the proportion 8:24:8).

It was observed that one of the two duplicate genes conditioned a ;2^{+}_{-} infection type while the other was more effective and conferred a hypersensitive 1^{+} infection type. Presumably when the two genes are combined, a more effective resistance (;1^{-} infection type) results. This inferrence was derived by examining the BC_1F_2 families. The progenies of families segregating 3:1, that is, segregating for one of the genes, displayed infection type 1^{+} while the other monogenically segregating families displayed infection type 2^{+}_{-}. Those families which segregated into 15R : 1S displayed both phenotypes on different plants in addition to the highly resistant phenotype of Tobari 66. To isolate the monogenic lines these reaction differences of the genes were also taken into consideration.

The presented data reveal the presence of four genes for resistance against races EA5, EA7 and C17(56) in Tobari 66. The four genes are being isolated in a common background.

5.1.5 INHERITANCE STUDIES - CONLEY x HINDI 62

The data presented in TABLE 5:22 shows that Conley is moderately resistant, (;2) to race EA7. Hindi 62 on the other hand is fully susceptible.

TABLE 5:22 THE STEM RUST REACTIONS TO RACE EA7 OF PARENTS AND SEEDLINGS OF CONLEY CROSS AND BACKCROSS TO HINDI 62.

Generation or Parent	Breeding behaviour			Expected	Chi-	P-
	MR	Seg	S	Ratio	Square	Value
Hindi 62 (H62)	-	-	S	-	-	-
Conley (CLY)	MR	-	-	-	-	-
H62 x CLY						
F_1	0	0	17	-	-	-
F_2	259	-	850	1:3	1.602	.30-.20
$H62^2$ x CLY						
F_2 families	0	29	27	1:1	0.018	.95-.90

Segregation within BC_1F_2 families was: 92R : 277S; P-value = 1 for a 3:1 ratio.

The F_1 generation was susceptible, 3^+ infection type. This indicates that the gene for resistance in Conley is recessive. The F_2 backcross families segregated into 29 Seg. : 27S, thus substantiating the single gene hypothesis.

During the seedling stage, Conley controlled the EA5 very effectively. The infection type on Conley was ;1 pustules surrounded by a whitish necrotic area. The data on TABLE 5:23 shows the response to EA5 of the parents: Hindi 62 and Conley; F_1 and F_2; BC_1F_2 and BC_1F_3 families used in gene analysis.

TABLE 5:23 THE STEM RUST REACTIONS TO RACE EA5 OF PARENTS AND SEEDLINGS OF CONLEY CROSS AND BACKCROSS TO HINDI 62.

Generation or Parent	Breeding behaviour			Expected	Chi-	P-
	R	Seg	S	Ratio	Square	Value
Hindi 62 (H62)	-	-	S	-	-	-
Conley (CLY)	R	-	-	-	-	-
H62 x CLY						
F_1	18	0	0	-	-	-
F_2	541	-	171	3:1	0.367	.70-.50
$H62^2$ x CLY						
F_2 families	0	30	31	1:1	0.008	.95-.90
F_3 lines	5	13	0	1:2	0.062	.80-.70

Within the segregating families the ratio was:

BC_1F_2 families 392R : 152S P-value = .20 - .10 for 3:1 ratio

BC_1F_3 lines 904R : 303S P-value = .95 - .90 for 3:1 ratio.

The 18 F_1 plants being as resistant as Conley suggested that the Conley gene for resistance to EA5 is completely dominant. The F_2 plants segregated into proportions indicating a monogenic mode of inheritance for Conley. From the 61 BC_1F_2 families a closely fitting 1 Seg. : 1S ratio was obtained in support of the monogenic inheritance. Within the segregating lines a 3:1 ratio was observed in spite of a slight deffi-ciency in the resistant class. The segregation among the BC_1F_3 families, selected from resistant BC_1F_2 plants, also fitted a 1:2 ratio expected

for a progeny test ratio in the absence of a susceptible class. The segregation within the BC_1F_3 families fitted satisfactorily the 3:1 ratio with a P-value of .95 to .90.

Conley is highly resistant (;1) to race C17(56). The results in TABLE 5:24 show complete dominance in the F_1 generation. The infection type ;1^{+}_{-} scored was identical to that of Conley parent. The F_2 population indicated a digenic mode of inheritance by fitting the expected 15R:1S ratio satisfactorily. A total of 37 BC_1F_2 families were also tested. The results obtained fitted the 3:1 ratio expected from BC_1F_2 for a two-gene mode of inheritance: 28 families segregated whereas the remaining nine were susceptible, confirming the two-gene hypothesis. Within the segregating lines 7 lines segregated into 129R:9S thus fitting a 15:1 ratio satisfactorily. The remaining 21 families segregated into 573R:185S, this was also a close fit to the expected 3:1 ratio.

Although the two duplicate genes were more or less equally effective, nearly one-third of the resistant families differed slightly from the rest by exhibiting a light yellow necrotic area around the pustules. The other one-third, segregating into 3:1 ratio had a reaction phenotype which was indistinguishable from the digenically segregating families with white necrosis around the pustules. For the isolation of monogenic lines from Conley, one criterion was to select the white flecked plants segregating for a 3:1 ratio and to discard all white flecked seedlings segregating into 15:1 ratio. Similarly the yellowish speckled plants were earmarked as carriers of the second gene.

TABLE 5:24 THE STEM RUST REACTIONS TO RACE C17(56) OF PARENTS AND SEEDLINGS OF CONLEY CROSS AND BACKCROSS TO HINDI 62.

Generation or Parent	Breeding behaviour			Expected	Chi-	P-
	R	Seg	S	Ratio	Square	Value
Hindi 62 (H62)	-	-	S	-	-	-
Conley (CLY)	R	-	-	-	-	-
H62 x CLY						
F_1	16	0	0	-	-	-
F_2	459	-	27	15:1	0.400	.70-.50
$H62^2$ x CLY						
F_2 families	0	28	9	3:1	0.009	.95-.90

Segregation within BC_1F_2 families was:

7 families seg. 15R : 1S P-value .70-.50

21 families seg. 3R : 1S P-value .80-.70

The inheritance studies reveal four genes in Conley for resistance against races EA5, EA7 and C17(56). Three of them are being isolated into Hindi 62 background.

5.1.6 INHERITANCE STUDIES - C.I.8154-FROCOR^2 x HINDI 62

C.I.8154-Frocor^2 is a very useful breeding material in Kenya. It is resistant to most of the prevailing EA races as indicated in TABLE 3:3.

Dominance of resistance to EA4 was evidenced from the 17 F_1 plants (TABLE 5:25). The plants exhibited ;1 infection type identical to C.I.8154-Frocor^2. The F_2 population fitted the expected 3:1 ratio, suggesting a single dominant gene inheritance for C.I.8154-Frocor^2 against EA4.

TABLE 5:25 THE STEM RUST REACTIONS TO RACE EA4 OF PARENTS AND SEEDLINGS OF C.I.8154-FROCOR^2 CROSS AND BACKCROSS TO HINDI 62.

Generation or Parent	Breeding behaviour			Expected	Chi-	P-
	R	Seg	S	Ratio	Square	Value
Hindi 62 (H62)	-	-	S	-	-	-
C.I.8154-Frocor^2 (CIF)	R	-	-	-	-	-
H62 x CIF						
F_1	17	0	0	-	-	-
F_2	2428	-	820	3:1	0.105	.80-.70
H62^2 x CIF						
F_2 families	0	26	28	1:1	0.018	.95-.90

The within BC_1F_2 families segregated: 703R : 246S for a 3:1 ratio with P-value of .70 to .50.

The 1:1 BC_1F_2 segregation confirms the single gene mode of inheritance hypothesized (TABLE 5:25). Segregation within these BC_1F_2 families did not deviate from the expected 3:1 ratio, thus further confirming the dominant single gene difference between CIF and H62 against race EA4.

C.I.8154-Frocor^2 is highly resistant (;1$^-$) to the East African race 5. The F_1 plants were fully resistant (TABLE 5:26). This indicates that C.I.8154-Frocor^2 is completely dominant over Hindi 62 for resistance against race EA5.

TABLE 5:26 THE STEM RUST REACTIONS TO RACE EA5 OF PARENTS AND SEEDLINGS OF C.I.8154-FROCOR^2 CROSS AND BACKCROSS TO HINDI 62.

Generation or Parent	Breeding behaviour			Expected Ratio	Chi-Square	P-Value
	R	Seg	S			
Hindi 62 (H62)	-	-	S	-	-	-
C.I.8154-Frocor^2 (CIF)	R	-	-	-	-	-
H62 x CIF						
F_1	16	0	0	-	-	-
F_2	812	-	58	15:1	0.258	.70-.50
H62^2 x CIF*						
F_2 families	0	41	12	3:1	0.082	.80-.70

*See text for the breeding behaviour within the BC_1F_2 families.

The F_2 generation revealed the presence of two dominant genes which was confirmed on the basis of data from the BC_1F_2 families. 41 out of 53 BC_1F_2 families segregated for resistance while 12 families were fully susceptible. Among the 41 segregating families: 17 segregated in approximately 15:1 ratio. The remaining 24 BC_1F_2 families segregated into 3R:1S classes. It is to be noted that the segregation of the 53 BC_1F_2 lines into 17 Seg. 15:1; 24 Seg. 3:1 and 12 susceptible families fits the expected dominant duplicate ratio of 1:2:1, that is, when the two classes of families segregating into 3:1 ratio are indistinguishable. The calculated P-value for the observed 17:24:12 was .75 to .50.

All the resistant plants whether segregating for two gene loci or single gene locus were indistinguishable from the C.I.8154-Frocor^2 phenotype. All exhibited a dirty white necrosis surrounding the pustules.

C.I.8154-Frocor^2 provides a very weak resistance to the North American race C10(15B-1) (TABLE 5:27). It exhibits a mesothetic (x^{+}_{-}) type of resistance reaction. The larger pustules 3^{cn} predominated over the smaller more resistant types.

F_1 plants were fully susceptible. This points to a recessive mode of inheritance. In the F_2 generation distinguishing the various resistant classes from the susceptible plants was not clearcut. This might account for the poor fit to the expected 1:3 ratio. Other reasons for the poor fit are also possible. In this context, the action of the modifying factors influencing the mesothetic resistance in C.I.8154-Frocor^2 is highly suspected. Occasionally plants of x^- type were observed in the F_2 genera-

TABLE 5:27 THE STEM RUST REACTIONS TO RACE C10(15B-1) OF PARENTS AND SEEDLINGS OF C.I.8154-FROCOR^2 CROSS AND BACKCROSS TO HINDI 62.

Generation or Parent	Breeding behaviour			Expected Ratio	Chi-Square	P-Value
	MR	Seg	S			
Hindi 62 (H62)	-	-	S	-	-	-
C.I.8154-Frocor^2 (CIF)	MR	-	-	-	-	-
H62 x CIF						
F_1	0	0	17	-	-	-
F_2	108	-	379	1:3	2.084	.20-.10
H62^2 x CIF						
F_2 families	0	25	23	1:1	0.021	.90-.80

The within BC_1F_2 families segregated: 147R : 542S with a P-value of .05 - .01 for 1:3 ratio.

tion. In spite of problems in classification, the F_2 data summarized in TABLE 5:27 indicates the presence of a single recessive major gene in C.I.8154-Frocor^2. The monofactorial inheritance was clearly demonstrated from the backcross F_2 families which fitted a 1:1 ratio expected for a single gene difference. The fitness within BC_1F_2 families was almost as poor a fit to 1:3 ratio as that observed for the F_2 populations.

C.I.8154-Frocor^2 is highly resistant (;1) to C17(56). The CIF in crosses with the susceptible H62 produced F_1 plants which were less resistant (;2) than their resistant parent, ;1 infection type (TABLE 5:28). Therefore a partially dominant inheritance is postulated.

TABLE 5:28 THE STEM RUST REACTIONS TO RACE C17(56) OF PARENTS AND SEEDLINGS OF C.I.8154-FROCOR^2 CROSS AND BACKCROSS TO HINDI 62.

Generation or Parent	Breeding behaviour			Expected	Chi-	P-
	R	Seg	S	Ratio	Square	Value
Hindi 62 (H62)	-	-	S	-	-	-
C.I.8154-Frocor^2 (CIF)	R	-	-	-	-	-
H62 x CIF						
F_1	17	0	0	-	-	-
F_2	876	-	268	3:1	1.510	.30-.20
H62^{2}x CIF						
F_2 families	0	26	34	1:1	1.067	.30
F_3 lines	7	16	0	1:2	0.047	.90-.80

Ratio within the segregating families were:

BC_1F_2 families: 668R : 235S for 3:1 ratio P-value = .50 - .30

BC_1F_3 lines: 1245R : 446S for 3:1 ratio P-value = .10 - .05

In the F_2 generation individuals with the parental types of infection types ;1 and 4 only were recorded with a frequency distribution that fitted a 3:1 ratio for a single dominant gene governing resistance against C17(56) in C.I.8154-Frocor^2.

A more critical information regarding the postulated mode of inheritance was obtained from the F_2 backcross families which yielded 26 segregating : 34 susceptible non-segregating families. The Chi-square test indicates that the F_2 backcross data are in agreement with the hypothesis of a monogenically controlled inheritance to race C17(56). The BC_1F_3 progeny test also confirmed the single gene hypothesis.

The fact that the F_1 and BC_1F_2 families were less resistant (;2) than the resistant (;1$^-$) parent, while some F_2 plants were as resistant as C.I.8154-Frocor^2 deserves further consideration. The most plausible explanation is the effect of the H62 background which subdues the effect of the major gene.

The present studies suggest that five genes confer resistance against races EA4, EA5, C10(15B-1) and C17(56) in C.I.8154-Frocor^2. Three most effective ones are being isolated into Hindi 62 background.

5.1.7 INHERITANCE STUDIES - MINNESOTA 3654/60 x HINDI 62

Minnesota 3654/60 is an advanced line from a cross (Frontana-Kenya 58-Newthatch) Pilot. This line exhibits infection type ;2 to EA4.

The 14 F_1 plants were susceptible suggesting a recessive mode of inheritance (TABLE 5:29). There were 276 moderately resistant (;2) and 844 susceptible plants suggesting a single recessive gene conferring resistance.

The BC_1F_2 data provide confirmatory evidence in support of the monofactorial hypothesis for resistance to EA4 in Minnesota 3654/60.

TABLE 5:29 THE STEM RUST REACTIONS TO RACE EA4 OF PARENTS AND SEEDLINGS OF MINNESOTA 3654/60 CROSS AND BACKCROSS TO HINDI 62.

Generation or Parent	Breeding behaviour			Expected Ratio	Chi-Square	P-Value
	MR	Seg	S			
Hindi 62 (H62)	-	-	S	-	-	-
Minnesota 3654/60 (MIN)	MR	-	-	-	-	-
H62 x MIN						
F_1	0	0	14	-	-	-
F_2	276	-	844	1:3	0.076	.80-.70
$H62^2$ x MIN						
F_2 families	0	25	22	1:1	0.085	.80-.70

The within seg. BC_1F_2 families: 166R : 427S for 1:3 ratio with a P-value of .10 - .05.

Minnesota 3654/60 is normally very resistant to C17(56) race of stem rust, the infection type varying between ;1 to 1^+. The F_1 (TABLE 5:30) of this cross was as resistant as the resistant parent. This is an indication of complete dominance for resistance. The F_2 population indicated that the inheritance was controlled by two independently functioning genes segregating into approximately 13R:3S plant classes.

TABLE 5:30 THE STEM RUST REACTIONS TO RACE C17(56) OF PARENTS AND SEEDLINGS OF MINNESOTA 3654/60 CROSS AND BACKCROSS TO HINDI 62.

Generation or Parent	Breeding behaviour			Expected Ratio	Chi-Square	P-Value
	R	Seg	S			
Hindi 62 (H62)	-	-	S	-	-	-
Minnesota 3654/60 (MIN)	R	-	-	-	-	-
H62 x MIN						
F_1	16	0	0	-	-	-
F_2	476	-	94	13:3	1.909	.20-.10
$H62^2$ x MIN						
F_2*	0	38	8	3:1	1.138	.30-.20

*See text for the breeding behaviour within the BC_1F_2 families.

The results of tests on the BC_1F_2 families indicate that about one-fourth of the families were fully susceptible (TABLE 5:30). The results of the segregating three-fourths of the families were more confounded: 19 of these families segregated into 401R:123S; a proportion that fits a 3:1 ratio (P-value of .50 to .30); another 8 BC_1F_2 families segregated in approximately 349R:70S, 13:3 ratio with a P-value of .30 to .20. The remaining 4 families gave a poor fit to a 1:3 ratio with 36R:82S classes (P-value .20 to .10). In some cases the numbers in the families were too small to make it possible to distinguish definitely between the three

segregating classes. TABLE 5:31 illustrates the breeding behaviour of the BC_1F_2 generation more elaborately.

Although the fitness to 1:1:1:1 expected ratio is not satisfactory, there is a definite indication of two major genes conferring resistance against C17(56) (TABLE 5:31). One of the genes is dominant while the other is a recessive gene. The presence of minor genes cannot be precluded since within a few of the segregating lines the plants gave fairly larger pustule (2^- infection types). Both the two major genes are equally effective since both reaction types varied between ; to 2 infection types on a few plants within the segregating families.

TABLE 5:31 ANALYSIS OF THE BC_1F_2 FAMILIES OF HINDI 62 x MINNESOTA 3654/60 TO RACE C17(56).

	F_2 Backcross Families				Expected
	Segregating			Sus-	Ratio
	3:1	13:3	1:3	ceptible	1:1:1:1
Observed	19	8	4	8	
Calculated	9.75	9.75	9.75	9.75	
Chi-square	11.000				
P-value	.05 - .01				

To isolate the monogenic lines, plants from the families segregating for 3:1 and 1:3 were kept separately for further backcrosses and selections. The three genes detected in the present mode of inheritance studies of Minnesota 3654/60 are all being isolated into Hindi 62. In addition, four other genes which have not been described in the present studies, but confer resistance to races EA5 and C10(15B-1) will also be included in the isolation exercise.

5.1.8 INHERITANCE STUDIES - WISCONSIN 245-II-50-17 x HINDI 62

Stem rust resistance governed by Wisconsin 245-II-50-17 has been relied upon in Kenya for a long time. In the present investigations, genetic information relating to EA7 is reported. The recurrent parent Hindi 62 gave a 4 infection type while Wisconsin 245-II-50-17 (WIS) produced ;1 infection type.

The F_1 plants exhibited a less effective resistance ;2 than the resistant parent signifying an incompletely dominant mode of inheritance. The F_2 varied between the heterozygous phenotype ;2 to the resistant ;1 of Wisconsin 245-II-50-17 but gave a satisfactory fit to the expected 3:1 ratio (TABLE 5:32).

Out of the 46 BC_1F_2 backcross families, 22 segregated for resistance while 24 were susceptible confirming the single gene predicted on the basis of F_2 generation. The BC_1F_3 lines from selected resistant BC_1F_2 progenitors further supported the single gene hypothesis. Within the segregating BC_1F_3 families, the frequency distribution of 1505R:501S fitted perfectly the postulated single gene inheritance for Wisconsin

TABLE 5:32 THE STEM RUST REACTIONS TO RACE EA7 OF PARENTS AND SEEDLINGS OF WISCONSIN 245-II-50-17 CROSS AND BACKCROSS TO HINDI 62.

Generation or Parent	Breeding behaviour			Expected	Chi-	P-
	R	Seg	S	Ratio	Square	Value
Hindi 62 (H62)	-	-	S	-	-	-
Wis. 245-II-50-17 (WIS)	R	-	-	-	-	-
H62 x WIS						
F_1	18	0	0	-	-	-
F_2	783	-	260	3:1	0.003	1 -.95
$H62^2$ x WIS						
F_2 families	0	22	24	1:1	0.022	.90-.80
F_3 lines	11	20	0	1:2	0.029	.90-.80

The ratio within the segregating families was:

BC_1F_2 families: 797R : 256S for 3:1 P-value = .70-.50

BC_1F_3 lines: 1505R : 501S for 3:1 P-value = 1

245-II-50-17 against EA7. Resistant plants in this cross were very easy to distinguish. All were characterized by a white necrotic area surrounding the ;1 pustules.

The gene for resistance against race EA7 indicated in the preceding data (TABLE 5:32) in addition to a partially dominant gene effective against race EA5 but not described here in detail will be isolated from Wisconsin 245-II-50-17. From the eight varieties studied a total of 25 genes were revealed. All of these were isolated as monogenic lines initially.

5.2 THE MONOGENIC LINES

Twenty-five monogenic lines presented in TABLE 5:33 have been isolated and retained for further studies. Each of them was extracted from single plants of Hindi 62^4 x donor resistant parents, that is, from BC_3F_3 lines in the manner described in chapter 4. Only homozygous plants from non-segregating lines have been retained to serve as the carriers of the resistance genes from their respective donor parents. A variety which was tested to more than one race; if resistance to each race in the test was governed by single genes, the corresponding number of lines were retained initially. Likewise varieties possessing more than one gene for resistance to the same race, the corresponding number of monogenes were retained.

The retained lines have been designated names: the first three letters stand for the donor parental abbreviation, whilst the second part of the nomenclature refers to the primary race for which the gene was initially isolated. Whenever a variety had more than one gene conferring

TABLE 5:33 INFECTION TYPES OF THE 25 MONOGENIC LINES TO THE SIX TESTER RACES.

Monogene Line*1	Race Reaction						Gene*2 expression
	EA4	EA5	EA7	EA8	C10	C17	
KHR4	1	4	4	4	4	1^{+}	D
CIF4	1	3^{+}	4	2^{+}	3	3^{+}	D
MIN4	1	2^{-}	3^{+}	3^{+}	3	;1	R
CIF5a	4	1^{+}	4	4	4	1^{-}	R
CIF5b	3^{+}	1^{+}	4	4	4	1^{-}	D
TPY5	3^{+}	1^{-}	3^{+}	4	3^{+}	1^{-}	D
CLY5	4	1^{+}	4	4	4	1^{+}	D
MIN5	1	1	4	3^{-}	3^{-}	1	R
WIS5	3^{+}	;1	3^{+}	3^{+}	12	1	D
TBR5	4	2^{-}	4	4	3^{+}	2	D
TBR7	3^{+}	3^{+}	1^{+}	3^{+}	3^{-}	2^{+}	R
MIN7	3^{+}	3^{+}	1^{+}	3^{-}	3	3^{-}	R
WIS7	3^{+}	1^{+}	2^{+}	3^{+}	3^{+}	1^{+}	PD
KLD7	3^{+}	2^{-}	2^{+}	3^{+}	4	2^{-}	PD
TPY8*3	-	-	-	$3^{\pm}$	$3^{\pm}$	-	R
MINC10a	3^{+}	4	4	4	2	1^{+}	R or D
MINC10b	4^{-}	4	4	4	$2^{\pm}$	4	D
KLDC17	3^{+}	3^{+}	3^{-}	3^{+}	23	;1	PD
TPYC17	4	1^{-}	4	4	4	;1	PD
TBRC17a	4	1^{-}	$3^{\pm}$	3^{+}	3^{+}	2^{-}	PD
TBRC17b	4	4	4	4	12	$2^{\pm}$	D
CLYC17a	4	3^{+}	4	4	1^{-}	1^{+}	D
CLYC17b	3	1	4	4	2^{+}	1^{+}	D
MINC17a	3^{+}	4	4	4	2^{-}	;1	D
MINC17b	1	2^{+}	4	4	23^{-}	2	R
Hindi 62	4	4	4	4	4	4	-

*1All the monogenic linces are in Hindi 62 background (Hindi 62^{4})

*2D = complete dominant PD = partially dominant R = recessive

*3The gene was ineffective after third backcross.

resistance to the same race, then a suffix with a lower form lettering was appended to the name. Thus KHR4 means that the monogene was isolated from Kenya Hunter with EA4 as the avirulent race; CLYC17a refers to the monogene that was isolated from Conley (CLY) which governed resistance to race C17(56). Futhermore resistance was governed by more than one gene; the letter "a" represents one of the genes. In this monogenic formula no distinction between recessive and dominant genes has been indicated.

The infection types of the monogenic lines and Hindi 62 recurrent parent are given in TABLE 5:33. It is obvious that a number of the monogenes had their resistance reduced in comparison to the reactions recorded for the donor parents as presented in TABLE 3:3.

The Hindi 62 lines possessing genes for resistance to races EA8 and C10 from Trophy and C.I.8154-Frocor^2 were more difficult to deal with because the infection types of the lines varied somewhat between the tests and frequently resembled the mesothetic (x^+) infection type. In the progeny of the third backcross, the resistance to races EA8 and C10 was completely ineffective. Isolation of these two monogenes was therefore discontinued. However, the behaviour of the BC_1F_2 families indicated that the same recessive gene in each variety was mainly responsible for the reaction of races EA8 and C10(15B-1) since the same lines segregated or were susceptible to both the races.

5.3 IDENTITY OF THE ISOLATED GENES

5.3.1 THE MONOGENIC LINES IN RELATION TO IDENTIFIED Sr. GENES

The results of the crosses with known Sr lines are presented in the five tables to follow. As described in the chapter on Material and Methods, those Sr lines that were similar to the parental donors or to the isolated monogenic lines in reaction to a particular race were tested to establish the relationship between the established genes (TABLE 4:9) and the genes in the experimental varieties. In a few cases susceptible Sr genes were also crossed to the isolated monogenic lines primarily to test their monogeneity.

All the Sr lines except Sr13 are ineffective against EA4 (TABLE 4:9). Moreover the resistance ($2^{\pm}$) displayed by Sr13 against EA4 was temperature labile and was very ineffective in the present tests.

As outlined in TABLE 5:34 the genes effective against EA4, namely CIF4, KHR4 and MIN4 are not amongst the known Sr genes. Instead of the expected total resistance amongst the F_2 progenies or a digenic segregation, the F_2 plants segregated into a 3:1 ratio, or a 1:3 ratio for KHR4 and MIN4 respectively indicating a monogenic inheritance. However, Sr13 apparently expressed its effect when combined with C.I.8154-Frocor^2. The segregation 217R:10S fits both the ratio 15:1 and 61:3, but closer to the 61:3 mode of inheritance than to a digenic 15:1 ratio. This might be interpreted to indicate that besides the two genes (CIF4 and Sr13) there is at least one more minor gene still accompanying one of the two monogenic lines. However, on the assumption that the isolation of Sr13 was

complete, the CIF4 gene requires more backcrosses to H62 to eliminate this factor.

TABLE 5:34 THE RESULTS OF CROSSES OF MONOGENIC LINES TO SELECTED Sr LINES AGAINST RACE EA4.

Cross	No. of F_2 plants		Expected	Chi-	P-
	R	S	Ratio	Square	Value
KHR4 x Sr13	132	44	3:1	-	1
CIF4 x Sr11	113	32	3:1	0.664	.50-.30
CIF4 x Sr13	217	10	15:1	1.318	.30-.20
			61:3	0.040	.90-.80
CIF4 x Sr15	217	68	3:1	0.198	.70-.50
MIN4 x Sr5	33	119	1:3	0.877	.50-.30
MIN4 x Sr6	55	160	1:3	0.039	.90-.80
MIN4 x Sr13	79	218	1:3	0.405	.70-.50
MIN4 x Sr15	71	219	1:3	0.041	.90-.80
MIN4 x sr17	74	217	1:3	0.029	.90-.80

From the available information the genes isolated from CIF, MIN and KHR are not Sr13 and have not been identified previously. However, additional analyses are necessary before they can be given Sr numerical status.

TABLE 5:35 shows the results of the EA5 rust reactions of the crosses of monogenic lines to the known Sr lines. From this data it is evident

that the monogenic lines CIF5b, CLY5, TBR5 and TPY5 are all similar to Sr11. As a corollary the four monogenes are comparable to one another. This interpretation is drawn from the fact that every F_2 plant progeny from crosses involving CIF5b, CLY5, TBR5 and TPY5 to the Sr11 line bred true for resistance. In contrast, the monogenic lines WIS5, and the recessive line MIN5 which are effective against EA5 do not match any identified (Sr) gene which is effective against EA5. Of the fourteen Sr lines, only Sr11, Sr13 and sr17 gave comparable resistance with the presently isolated genes. Sr15 gave a mesothetic reaction type when conditions were just right but was susceptible on slight temperature fluctuations. The other genes Sr1, Sr5, Sr6, Sr7, Sr8, Sr9a, Sr9b, Sr10, Sr14 and Sr16 were susceptible and presumably do not provide resistance to the race EA5. In spite of their susceptibility some of them (TABLE 5:35) were crossed to the isolated monogenic lines; and all F_2 progenies segregated in the expected 3:1 ratio.

The monogene line CIF5a when crossed to Sr15 fitted most closely to 61:3 trihybrid ratio than to the 15:1 two gene difference. The apparent three gene difference might be due to minor gene factors. Furthermore the mesothetic reaction of Sr15 is quite sensitive to environmental changes. In view of this, misclassification of resistant : susceptible groups might have contributed to the poor fit to a 15:1 dihybrid ratio. However CIF5a is identical to sr17.

The Sr15 gene when crossed to monogenic line MIN5, produced a segregation which fitted a 9:7 complementary inheritance. This is difficult to

TABLE 5:35 THE RESULTS OF CROSSES OF MONOGENIC LINES TO SELECTED Sr LINES AGAINST RACE EA5.

Cross	Number of Plants		Expected Ratio	Chi-Square	P-Value
	R	S			
CIF5a x Sr11	138	48	3:1	0.064	.80-.70
			13:3	6.068	.05-.01
CIF5a x Sr13	290	17	15:1	0.266	.70-.50
CIF5a x Sr15	181	8	15:1	1.312	.30-.20
			61:3	0.087	.80-.70
CIF5a x sr17	172	0	-	-	-
CIF5b x Sr11	141	0	-	-	-
TPY5 x Sr5	118	40	3:1	0.008	.95-.90
TPY5 x Sr6	154	50	3:1	0.026	.90-.80
TPY5 x Sr11	240	0	-	-	-
TPY5 x Sr13	248	8	15:1	4.267	.05-.01
			61:3	1.333	.30-.20
CLY5 x Sr5	138	42	3:1	0.267	.70-.50
			13:3	2.482	.20-.10
CLY5 x Sr6	126	42	3:1	-	1
CLY5 x Sr11	149	0	-	-	-
CLY5 x Sr13	140	48	3:1	0.028	.90-.80
CLY5 x Sr15	103	6	15:1	0.103	.80-.70
MIN5 x Sr6	21	58	1:3	0.105	.80-.70
MIN5 x Sr13	96	20	3:1	3.724	.10-.05
			13:3	0.173	.70-.50
MIN5 x Sr15	121	90	9:7	0.103	.80-.70
MIN5 x sr17	60	89	7:9	0.734	.50-.30
WIS5 x Sr5	80	28	3:1	0.049	.90-.80
WIS5 x Sr6	114	34	3:1	0.324	.70-.50
WIS5 x Sr11	78	6	15:1	0.011	.95-.90
WIS5 x sr17	110	22	3:1	4.889	.05-.01
			13:3	0.376	.70-.50

continued .....

TABLE 5:35 continued

Cross	Number of Plants R	Number of Plants S	Expected Ratio	Chi-Square	P-Value
TBR5 x Sr5	88	27	3:1	0.142	.80-.70
			13:3	1.688	.20-.10
TBR5 x Sr6	75	23	3:1	0.122	.80-.70
TBR5 x Sr11	121	0	-	-	-
TBR5 x Sr13	256	19	15:1	0.204	.70-.50
TBR5 x Sr15	144	48	3:1	-	1
TBR5 x sr17	154	36	3:1	3.712	.10-.05
			13:3	0.005	.95-.90

explain. It might be assumed that the variety Kenora (Sr15) possesses some other factor(s) which modifies the effects of the major genes. But the 7:9 ratio obtained by crossing MIN5 to sr17 is quite normal for F_2 segregates from two recessive independently assorting genes. MIN5 and sr17 are both recessive monogenic lines.

Whilst monogenic lines MIN5 is recessive, Sr11 is completely dominant resistant against race EA5. Furthermore Sr11 produces 0; (TABLE 4:9), MIN5 exhibits clear whitish necrotic lesions around the ;1 pustules (TABLE 5:33). Therefore MIN5 and Sr11 are probably not identical genes.

In TABLE 4:9 it was demonstrated that none of the Sr lines exhibit any resistance to the East African race 7. The results summarized in TABLE 5:36 confirmed this observation. The monogenic lines carrying genes resistant to EA7 when crossed to the established Sr lines, all displayed a single gene inheritance. The apparent exceptions were the KLD7 gene when crossed to Sr11 and sr17. The Sr11 cross segregated closer to a 13:3 ratio (P-value .75 - .50) than to a 3:1 ratio (P-value of less than .01). This behaviour deserves some explanation. Sr11 alone is not effective against EA7, but in combination with KLD7, Sr11 gives some protection. This leads one to surmise that the expression of Sr11 against EA7 is inhibited in a predominantly Marquis background. A larger population for the KLD7 x sr17 is necessary to ascertain which of the two ratios (13:3 or 3:1) is the most appropriate ratio. However, from the results obtained, there is clear proof that KLD7, TBR7, MIN7 and WIS7 monogenic lines have not been identified previously.

TABLE 5:36 THE RESULTS OF CROSSES OF MONOGENIC LINES TO SELECTED Sr LINES AGAINST RACE EA7.

Cross	Number of F_2 Plants		Expected	Chi-	P-
	R	S	Ratio	Square	Value
TBR7 x Sr5	79	266	1:3	0.813	.50-.30
TBR7 x Sr6	89	298	1:3	0.828	.50-.30
TBR7 x Sr11	75	218	1:3	0.056	.90-.80
TBR7 x Sr13	93	297	1:3	0.900	.50-.30
TBR7 x sr17	91	277	1:3	0.145	.80-.70
MIN7 x Sr5	29	95	1:3	0.172	.70-.50
MIN7 x Sr6	48	143	1:3	0.002	1
MIN7 x Sr13	34	156	1:3	5.116	.05-.01
MIN7 x Sr15	58	173	1:3	0.001	1
MIN7 x sr17	46	128	1:3	0.192	.70-.50
WIS7 x Sr5	160	51	3:1	0.077	.80-.70
WIS7 x Sr6	121	44	3:1	0.183	.75-.50
WIS7 x Sr11	132	46	3:1	0.067	.80-.70
WIS7 x sr17	174	56	3:1	0.052	.90-.80
KLD7 x Sr6	251	91	3:1	0.477	.50-.30
KLD7 x Sr11	391	95	3:1	7.706	Less .01
			13:3	0.202	.75-.50
KLD7 x Sr13	320	97	3:1	0.672	.50-.30
KLD7 x Sr15	298	110	3:1	0.837	.50-.30
KLD7 x sr17	118	33	3:1	0.797	.50-.30
			13:3	0.955	.50-.30

The monogenic lines isolated from Minnesota 3654/60 were crossed to Sr6 and Sr7. The results are summarized in TABLE 5:37. All F_2 plants of cross MINC10a x Sr6 were all resistant. Whilst, when monogene MINC10a was crossed to Sr7, the F_2 plants segregated in the manner expected for a digenic ratio. The 13:3 ratio is obtained when two genes, one recessive and the other dominant are segregating independently for the same character. It is therefore concluded that the monogene line MINC10a is identical to Sr6.

Of all the fourteen Sr tester stocks used (TABLE 4:9) only Sr6 and Sr7 exhibited consistent resistance to race C10(15B-1). The remainder were either fully susceptible or erratic. Sr8 was susceptible more often than resistant (3 infection type).

TABLE 5:37 THE RESULTS OF CROSSES OF MONOGENIC LINES TO THE Sr LINES THAT CONFER RESISTANCE TO NORTH AMERICAN RACE C10(15B-1).

Cross	Number of F_2 Plants		Expected	Chi-	P-
	R	S	Ratio	Square	Value
MINC10a x Sr6	54	0	-	-	-
MINC10a x Sr7	161	31	13:3	0.755	.50-.30
MINC10b x Sr6	176	46	13:3	0.780	.50-.30
MINC10b x Sr7	84	0	-	-	-

Referring again to TABLE 5:37 the monogene line MINC10b proved to be identical to the Sr7. This interpretation is based on the observation

that all the F_2 progenies of MINC10b x Sr7 were all resistant. The gene Sr6 when crossed to MINC10b gave F_2 segregates which fitted a 13:3 ratio with a P-value of .50 - .30.

From TABLE 4:9, resistance to race C17(56) is governed by most of the established Sr lines except Sr5, Sr7, Sr10, Sr15 and Sr16. Sr1, Sr8, Sr9a, Sr9b and Sr13 normally conferred a moderate protection while Sr8 and Sr14 were temperature sensitive and tended to produce susceptible reaction (3^+) rather than their respective 2^+ or $2^+\!\!-\!\!3$ infection types. Sr9b was also inconsistent and often produced (infection type 3^+) susceptible reaction. Lines of Marquis Sr6, Sr11 and Renown (sr17) gave hypersensitive resistant reactions.

The summary of the data collected to compare the relationship between the Sr lines and the monogenes TPYC17, TBRC17a and CLYC17b with Sr11 (TABLE 5:38) did not show any segregation. It is therefore inferred that these monogenes are similar to Sr11 and therefore allelic to one another. One of the two Conley monogenic lines CLYC17a, MINC17a, TBRC17b and KLDC17 are possibly identical to Sr6 because they did not display any segregation in the F_2 generation.

C.I.8154-Frocor^2 monogenic line CIFC17 when crossed to both the genes Sr1 and sr17 displayed no segregation in the F_2 generation. Several possibilities could be advanced to explain this. The most probable explanation is that the genes Sr1 and sr17 are present in the monogenic line CIFC17 with some linkage, hence do not assort independently. A larger population would be needed to get new recombinants possessing genes

TABLE 5:38 THE RESULTS OF CROSSES OF MONOGENIC LINES TO SELECTED Sr LINES AGAINST NORTH AMERICAN RACE C17(56).

Cross	Number of F_2 Plants		Expected	Chi-	P-
	R	S	Ratio	Square	Value
KLDC17 x Sr6	111	0	-	-	-
KLDC17 x Sr11	202	16	15:1	0.431	.70-.50
TPYC17 x Sr5	88	29	3:1	0.003	1 -.95
			13:3	2.798	.10-.05
TPYC17 x Sr6	136	9	15:1	0.000	1
TPYC17 x Sr9a	79	28	3:1	0.78	.80-.70
TPYC17 x Sr11	258	0	-	-	-
TPYC17 x Sr13	142	11	15:1	0.230	.70-.50
			61:3	2.142	.20-.10
TPYC17 x Sr14	111	32	3:1	0.524	.50-.30
TBRC17a x Sr1	99	26	3:1	1.176	.30-.20
			13:3	0.345	.70-.50
TBRC17a x Sr6	136	8	15:1	0.119	.80-.70
TBRC17a x Sr11	120	0	-	-	-
TBRC17a x Sr13	218	11	15:1	0.818	.50-.30
TBRC17a x Sr14	128	46	3:1	0.191	.70-.75
TBRC17a x Sr15	144	38	3:1	1.643	.20
			13:3	0.542	.50-.30
TBRC17b x Sr1	152	8	15:1	0.252	.70-.50
TBRC17b x Sr6	85	0	-	-	-
TBRC17b x Sr9a	114	10	15:1	0.439	.70-.50
TBRC17b x Sr11	136	6	15:1	0.394	.70-.50
TBRC17b x Sr13	141	46	3:1	0.016	.95-.90
			13:3	4.199	.05-.01
CLYC17a x Sr1	128	9	15:1	0.024	.90-.80
CLYC17a x Sr6	91	0	-	-	-
CLYC17a x Sr9a	185	12	15:1	0.008	.95-.90
CLYC17a x Sr11	151	13	15:1	1.476	.30-.20
CLYC17a x sr17	177	12	15:1	0.003	1 -.95
CLYC17b x Sr6	112	8	15:1	0.002	1 -.95
CLYC17b x Sr11	271	0	-	-	-

carried forward ....

TABLE 5:38 continued

Cross	Number of F_2 Plants		Expected	Chi-	P-
	R	S	Ratio	Square	Value
CIFC17 x Sr1	97	0	-	-	-
CIFC17 x Sr6	98	7	15:1	0.031	.90-.80
CIFC17 x Sr9b	137	27	15:1	0.563	.50-.30
CIFC17 x Sr14	107	35	3:1	0.009	.95-.90
CIFC17 x sr17	189	0	-	-	-
MINC17a x Sr5	85	5	15:1	0.074	.80-.70
MINC17a x Sr6	186	0	-	-	-
MINC17a x Sr11	145	3	15:1	4.504	.05-.01
			61:3	2.345	.20-.10
MINC17a x Sr13	80	4	15:1	0.317	.70-.50
MINC17a x sr17	161	38	13:3	0.016	.95-.90
MINC17b x Sr6	143	30	13:3	0.225	.70-.50
MINC17b x Sr11	156	41	13:3	0.550	.50-.30
			3:1	1.843	.20-.10
MINC17b x sr17	67	74	7:9	0.813	.50-.30

Srl and srl7 in separation.

Another possibility is that the monogenic line CIFC17 is one of the alleles in a multi-allelic system where srl7 is a recessive allele; srl7 becomes hypostatic in the presence of CIFC17 (Srl) allele. Therefore no susceptible segregants are expected wherever these two monogenes are segregating.

A third possibility is to assume that one of these major genes (possibly srl7) acts as a modifier of CIFC17 gene. Hence the F_2 populations are resistant irrespective of their genotype.

In spite of the above complication monogenic line CIFC17 and Srl are alike on the basis of dominance and the F_2 behaviour (TABLE 5:38).

Referring to TABLE 5:38 again, the second recessive monogenic line MINC17b of Minnesota 3654/60 has no corresponding established Sr gene to match with. It is therefore regarded as a new gene. When crossed to the recessive srl7, the MINC17b monogenic line produced F_2 plant classes in proportions of 67R:74S. This closely fits a 7:9 ratio expected when two recessive genes assort independently.

TABLE 5:39 gives a summary of the relationship between the 25 isolated monogenic lines and the Sr genes. Of the 25 genes studied 15 are obviously identical to identified genes of the Sr series. The remaining 10 are unidentified but may include some duplications.

5.3.2 INTRA-VARIETAL MONO-GENE COMPARISONS

As already described in section 4, under the "Procedure for individual experiments", every BC_1F_2 family was tested to as many races as the

donor parent was resistant if seed was adequate. This was accomplished either by proportioning the backcross seed or by simultaneously inoculating each plant with two different races. In this way it was possible to select and retain the BC_1F_2 families or plants which were resistant to one race but susceptible to the other. In certain cases the same gene conferred resistance to two or more races; families which segregated for one race also segregated for the other race and those which were fully susceptible to one or more races were likewise susceptible to the other race(s). In some cases of digenic inheritance, when both the intra-genes were dominant, the infection types were a guide to the identity of the different genes of the same variety. Recessiveness or dominance of resistance was also a good criterion in telling resistant genes of the same variety apart.

When the above leads were not conclusive enough then the intra-monogenic lines concerned were each crossed to the Sr tester stocks. TABLE 5:39 gives a summary of the genes identified in the previous section 5.3.1.

The monogenic lines MIN4 and MIN5 are progenitors of the same plant from BC_1F_2 family that segregated into 1R:3S. Consequently, the two lines are allelic.

Monogenic line MIN7 was inoculated with EA5 and EA4. The line while completely resistant to EA7 was fully susceptible (4 infection type) to races EA4 and EA5. This is interpreted to mean that the monogenic lines MIN4 and MIN5 are not similar to MIN7. Similarly the recessive monogene

TABLE 5:39 THE IDENTITY OF INTRA-VARIETAL GENES IN RELATION TO THE Sr TESTER STOCKS.

Hindi 62 monogenic line	Corresponding Sr line	Gene* expression
KHR4	-	D
CIF4	-	D
CIF5a	sr17	R
CIF5b	Sr11	D
CIFC17	Sr1	D
CLY5	Sr11	D
CLYC17a	Sr6	D
CLYC17b	Sr11	D
TPY5	Sr11	D
TPYC17	Sr11	PD
MIN4	-	R
MIN5	-	R
MIN7	-	R
MINC10a	Sr6	R
MINC10b	Sr7	D
MINC17a	Sr6	D
MINC17b	-	R
KLD7	-	PD
KLDC17	Sr6	PD
TBR5	Sr11	D
TBR7	-	R
TBRC17a	Sr11	PD
TBRC17b	Sr6	D
WIS5	-	D
WIS7	-	PD

* D = Completely dominant PD = Partially dominant
R = Recessive

MINC17b was a progeny from the BC_1F_2 family which had perpetuated the recessive monogenes MIN4 and MIN5. Since these lines were selected in different countries (Canada and Kenya), the best comparison was made when these homozygous (Hindi 62^4 x MIN) F_3 lines were tested to these races separately. All the three lines (MIN4, MIN5 and MINC17b) proved homozygous resistant to the three tester races EA4, EA5 and C17(56). It is reasonable to conclude that MIN4, MIN5 and MINC17b are similar to each other. Any one of these three monogenes is, therefore, sufficient to protect wheat varieties against stem rust races EA4, EA5 and C17(56). Applying the same argument, the monogenes TPY5 and TPYC17 are both comparable to Sr11 (TABLE 5:39).

The monogenic lines KLD7 and KLDC17 from Kenya Leopard present some difficulty to interpret. BC_1F_2 families which segregated for EA7 also segregated for C17(56). KLDC17 x Sr6 when tested under CDA greenhouse conditions against C17(56) showed no segregation. Similarly, KLD7 x Sr6 under CDA greenhouse conditions against C17(56) showed no segregation. But when both the monogenes were tested under PBS greenhouse conditions the results were different: both segregated when tested to EA7. This might indicate that KLD7 and KLDC17 are linked, one of them being temperature sensitive. At Winnipeg under more exacting greenhouse conditions, those plant segregates possessing either Sr6 recombinant or one of the two monogenic lines cannot be discerned from one another. But when the segregates possessing the temperature sensitive Sr6 genotype alone are tested under the PBS environment, the Sr6 gene becomes ineffective.

Another conflicting observation is that Kenya Leopard is not resistant to race C10(15B-1) while Sr6 is resistant. This might imply that the gene involved in resistance against EA7 and C17(56) is probably different or allelic to the existing Sr6. Another probable explanation to the apparent discrepancy, is that the monogenic isolation process is premature and therefore more tests and backcrosses would be needed before the complete identity of KLD7 and KLDC17 is confirmed.

Referring to TABLE 5:39 again, TBR5 and TBRC17a are allelic to Sr11. The BC_1F_2 family which perpetuated monogenic lines TBR5 and TBRC17a is the same. TBRC17b a duplicate pair of TBRC17a has been determined in the present studies (TABLE 5:39) to be identical to Sr6. The third Tobari 66 gene (recessive) designated TBR7 which confers resistance to EA7 has not been identified as yet. However, being recessive TBR7 is not likely to be identical to one of the two dominant Tobari 66 monogenic lines, TBRC17a or TBRC17b.

The two dominant monogenic lines WIS5 and WIS7 isolated from WIS. 245-II-50-17 are not allelic. This is inferred from the observation that the BC_1F_2 families which segregated for EA5 did not necessarily segregate for EA7. WIS5 and WIS7 are progenies of different BC_1F_2 families which segregated for one of the races and susceptible to the other race. Furthermore, when the BC_3F_3 WIS7 homozygous lines selected for resistance to race EA7 were inoculated with EA5 they were susceptible, similarly, when WIS5 monogenic line was inoculated with EA7 it was also susceptible. This confirms that WIS5 and WIS7 monogenic lines are not identical.

5.3.3 INTER-VARIETAL MONOGENE COMPARISONS

The monogenes KHR4, CIF4, MIN4, MIN5, MIN7, MINC17b, KLD7, KLDC17, TBR7, WIS5 and WIS7 remained unidentified with respect to Sr series. Those from the same variety are also different except MIN4, MIN5 and MINC17b which are identical to each other. Therefore all these MIN monogenes except MIN4 will be discarded.

When a thorough examination of the donor parents of the monogenic lines was performed (TABLE 3:3), it was found that Kenya Hunter is susceptible to EA5, EA7, EA8 and C10. On the assumption of gene-for-gene hypothesis the monogenic line KHR4 would also be ineffective against the above races; and in fact when the KHR4 homozygous line was inoculated with EA5, EA7, EA8 and C10(15B-1) it was susceptible. Therefore it was considered illogical to assume that KHR4 would be identical to other inter-varietal monogenic lines which do not confer resistance to EA4. Therefore, KHR4 was compared to CIF4 only, since MIN4 is a recessive monogenic line while KHR4 and CIF4 are dominant. In the cross KHR4 x CIF4, (TABLE 5:40) no segregation occurred in the F_2 generation, indicating that monogenic lines KHR4 and CIF4 which both confer resistance to EA4 are similar.

Wisconsin 245-II-50-17 confers resistance to all the test races except EA4 and EA8. Genes which are specific to EA4 and EA8 only are, subsequently, not identical to monogenic lines WIS5. From inoculation tests, EA7 and C10(15B-1) are also virulent on WIS5. Therefore WIS5 was compared to those inter-varietal genes which were effective against EA5 and C17(56)

TABLE 5:40 RESISTANT x RESISTANT INTER-VARIETAL COMPARISONS (F_2 POPULATIONS).

Cross	EA4		Exp.	P-	EA5		Exp.	P-	EA7		Exp.	P-	C17(56)		Exp.	P-
	R	S	ratio	value	R	S	ratio	value	R	S	ratio	value	R	S	ratio	value
KHR4 x CIF4	102	0	-	-	0	85	-	-	0	76	-	-	0	76	-	-
MIN7 x TBR7	0	84	-	-	0	66	-	-	115	0	-	-	0	43	-	-
WIS7 x KLD7	0	96	-	-	0	72	-	-	141	0	-	-	0	58	-	-
MIN4 x TBR7	141	38	3:1	.30-.20	161	44	3:1	.30-.20	164	43	3:1 13:3	.20-.10 .50-.30	108	39	3:1	1
WIS5 x KLD7	0	76	-	-	125	50	3:1	.30-.20	142	46	3:1	.90-.80	162	61	3:1	.50-.30

only. Of the monogenes which have not been accounted for, (TABLE 5:39), in terms of Sr lines, none of them fulfils this prescription. It is therefore inferred that WIS5 is different from the presently isolated genes including the established Sr lines.

Another unique pair of resistant monogenic lines are the lines MIN7 and TBR7. The genes involved in these lines are both recessive. It was therefore considered unnecessary to compare these with the dominant monogenic lines WIS7 and KLD7 which also conferred resistance to race EA7. Crosses between MIN7 and TBR7 were conducted; (TABLE 5:40) there was no segregation in the F_2 generation indicating that they are the same. Data in TABLE 5:33 also lead to the same inferrence.

The remaining monogenic lines WIS7 and KLD7 each confers dominant resistance to EA7 only in the present tester races. When KLD7 was crossed to WIS7 the F_2 generation was homozygous resistant, hence the two monogenes are probably identical.

On the basis of these tests, the 25 monogenic lines are composed of at least 10 genes: fifteen lines are duplicates of 5 existing Sr genes while the remaining ten lines include duplicates of 5 unidentified genes.

5.4 THE RANGE OF RESISTANCE CONFERRED BY THE MONOGENIC LINES

The five new unidentified monogenic lines (TABLE 5:41), were tested to seven North American races and to six East African races to check on the spectrum of their resistance. The North American rust cultures were obtained from CDA Research Station, Winnipeg through the courtesy of Dr. G. J. Green. The cultures included: C1(17) 87-65; C2(17A) 6-68;

TABLE 5:41 THE EFFECTIVE RANGE OF THE FIVE NEW MONOGENIC LINES.

Monogenic Line[*2]	RACES[*1] AND INFECTION TYPES												
	East African						North American						
	11 (40)	12 (40)	13 (34)	14 (11)	15 (11)	16 (40)	C1 (17)	C2 (17A)	C1B (15B-1L)	C20 (11)	C22 (32)	C25 (38)	C41 (32-113)
KHR4	$1^{\pm}$	;1	;	1	0;	3^{-}	0;	0;	;1	$;1^{-}$	$;1^{\pm}$	$2^{\pm}$	0;
TBR7	3^{-}	1	;	;	3	2^{-}	0;	0;	$1^{\pm}$	$1^{\pm}$	;1	$3^{\pm}$	23
KLD7	1^{-}	1	1	3^{-}	;1	1^{-}	0;	;	;1	3^{-}	0;	0;	$2^{\pm}$
MIN4	2^{+}	1^{-}	3	1	;1	1^{+}	0;	;	$;1^{\pm}$	0;	$12^{\pm}$	12^{+}	;
WIS5	;1	1^{+}	3	$3^{\pm}$	$3^{\pm}$	1	0;	1^{+}	;	0;	;1	;	;
Hindi 62	4	3^{+}	4	4	4	3^{+}	3	$3^{\pm}$	4	34	3^{+}	23	4

*1 Numerals in parenthesis show the "International" Races.

*2 All the monogenic lines are in the Hindi 62 background (Hindi 62^{4}).

C18(15B-11) 20-68; C20(11) 231-70; C22(32) 39-68; C25(38) 197-65 and C41(32-113) 92-70. The East African races included: EA11(40); EA12(40); EA13(34); EA14(11); EA15(11) and EA16(40). The observed infections are presented in TABLE 5:41.

From TABLE 5:41 it is obvious that the five new genes confer resistance to a wide range of races. They are therefore very useful breeding material.

Nevertheless, the reactions presented in TABLE 5:41 require more confirmatory tests. The tabulated results are a summary of two replications only. It is possible that for races: C1(17); C2(17A); C20(11) and C25(38) where the recurrent, Hindi 62, parent showed some resistance, the infection types displayed in TABLE 5:41 might not be due to the resistance of the isolated donor genes alone but also might have been contributed by Hindi 62 genotype.

6. DISCUSSION AND CONCLUSIONS

The inheritance of reaction in the seedling stage to individual wheat stem rust races was studied using F_1, F_2 and BC_1F_2 families. Frequently progeny tests were conducted on the BC_1F_3 lines derived from the resistant BC_1F_2 progenitors. The primary test races included EA4(295); EA5(34); EA7(40); EA8(40); C10(15B-1) and C17(56). In regard to the methods for genetic analysis, it was found that the study of F_2 families from backcrosses to a susceptible variety was superior over the study of F_2 lines. In backcross analysis, however complex the mode of inheritance seemed to be, it was easier to distinguish segregating from non-segregating families. Furthermore segregation within the BC_1F_2 families added invaluable supplemental information regarding the modes of inheritance.

The results obtained relative to the seedling inheritance of resistance point in the main to a Mendelian manner of inheritance. Temperature and possibly light were also considered to be important factors in the breakdown of seedling resistance in these studies. Resistant reactions to the six races were found to be controlled by at least ten different major genetic factors. Susceptibility was dominant in Minnesota 3654/60 for EA4 resistance; Trophy too had recessive genes conferring resistance against EA8. Tobari 66 and Conley had one recessive gene each against EA7. C.I.8154-Frocor[2] also displayed a recessive gene expression for resistance against C10. However, resistance was dominant in most of the cases studied. For most situations, resistance factors were inherited independently of one another. Linkage of the genes might have distorted a few of the

genotypic ratios. In one clear case mono-gene CIFC17, selected for resistance against race C17; and identical to gene Sr1, was found not to segregate independent of sr17. In another case the two monogenic lines KLD7 and KLDC17 from Kenya Leopard were found to associate together quite frequently with a recombinational value of 28.3 per cent. The results showed that resistance to EA4 is controlled by two major genes; EA5 by four genes; EA7 by one gene; C10(15B-1) by two genes and C17(56) by four genes.

Gene expression for a number of the genes reported in this paper could be explained by at least partially dominant factors and for several of them, such as the gene in the variety C.I.8154-Frocor^2 conferring resistance against C10(15B-1) or the Trophy gene for EA8, the heterozygotes conditioned an infection type that was boarderline between a type 2 and a type 3. During the course of these studies it was also noted that varieties or lines of Tobari 66, C.I.8154-Frocor^2 and Trophy conferring a moderate resistance reaction were erratic and became ineffective at times especially when the test conditions were sub-optimal. Some of the variability in these expressions was undoubtedly due to modifiers. The varieties carrying these apparent variable factors were crossed and backcrossed three times to Hindi 62 and selection for resistance to respective races was done continually. As the backcrossing proceeded, selection became more difficult. When the lines were in BC_3F_3, all the plants were susceptible. To recover good resistance from such lines, it would be necessary to add some of the modifying genes. From the present results,

it was evident that the inheritance of rust resistance is extremely complex. The evidence now available from numerous studies (Loegering et al. 1957, Green et al. 1960 and Berg et al. 1963) also support the existence in many wheat genotypes of extensive systems of loci which modify the effects of major genes for stem rust resistance. The modifiers make it difficult both to identify the effects of the major genes, and to study the possible complementary or epistatic interactions between these genes. The importance of modifiers or perhaps of a genic-environment balance on rust resistance is again emphasized in the present work. While the genes reported here determine whether a variety was or was not resistant to the experimental races, the degree of resistance they condition was variable in different crosses. In these cases it was not known whether the difference was due to specific modifiers carried by Hindi 62 or to the genic-environment interaction in general. For instance, it was clear that there was at least one modifier of the resistance to race EA8 conditioned by a single major recessive gene in Trophy. This observation is in agreement with the findings of Anderson et al. (1956) and Sheen et al. (1965) who reported enhanced and reduced resistance reaction by modifying factors. Besides, the minor factors, major genes (Sr9 and Sr10) have been reported to act as modifiers of the gene Sr7. This modifier effect probably explains many of the difficulties encountered in maintaining full resistance while backcrossing to produce rust resistant varieties and monogenic lines.

When genes conditioning resistance to the same race were combined in the inter-varietal or intra-varietal gene interrelationship comparisons,

the resulting host reaction was typically that associated with the locus conferring the higher level of resistance and the F_2 segregations were generally in good agreement with ratios calculated on the expected parental genotype. The lone exception was when monogenic line MIN5 was crossed to Sr15 where a good fit to 9:7 ratio was obtained. The latter suggested the occurrence of inter-allelic interactions in the determination of host reactions. In the absence of non-allelic interaction between major loci, the genetic basis of the host-parasite relationship postulated by Flor (1946) requires as many alleles to condition host resistance as there are pathogenicity genes in the pathogen. Apparently from the present results the wheat host displayed a relatively smaller number of loci in comparison with the wide array of genes for virulence in P. graminis races employed in these studies. Evidences relating to possible multiple allelism at various loci conferring host resistance are limited and inconclusive. Resolving close linkages governing resistance and the problem of critically demonstrating the identity or non-identity of loci from different hosts present difficulties which pose a limitation to the analysis of this host-pathogen system. However, the backcross (test-cross) analytical procedure may prove more efficient than the traditional F_2 population analysis for the resolution of resistance factors.

In spite of analytical complications, the results demonstrated that the eight experimental varieties and strains: KHR, KLD, TBR, CLY, CIF, WIS, TPY and MIN; carried at least five identified genes, Sr1, Sr6, Sr7, Sr11 and sr17 for rust resistance. They also carried five promising new

resistance genes. The present results corroborate the predictions made by Evans et al. (1969) and Green et al. (1970) that there were gene duplications and a few unidentified resistance genes effective against the East African races amongst the current Kenya sources of black stem rust resistance. They concluded this when the Sr genes identified in North America were largely ineffective against East African races. The present studies also showed a very high predominance of Sr11 in the current Kenya varieties and breeding material. This finding is in concordance with Harder's 1972 finding that Sr11 was effective to 52 per cent of all the 1970 rust isolates in Kenya and Tanzania, and that probably it (Sr11) would occur in most of the Kenya wheat varieties. Another interesting observation from these results was the fact that of the older Sr lines identified (Knott et al. 1956 and 1962), amongst the Kenya cultivars, only genes Sr6 and Sr11 with broad spectrum of resistance and high effectiveness over the past and the current EA races appear to have survived the selection pressure over the years.

The five new genes could not be traced back to any common ancestor since they (experimental cultivars) were of diverse geographic origin and parentage. Besides, none of their parents had ever been studied under stem rust race situation similar to Kenya nor had they ever been genetically analyzed in Kenya. Nonetheless, in general the results in this paper agree well with those reported for the progenitors of some of the varieties by other investigators, wherever the genes were also effective against the four EA and two North American races in the present experiment.

Conley, for instance, inherited Sr11 from the variety Lee for resistance to EA5 and C17(56) and Sr6 from the variety McMurachy. Trophy has Timstein as one of its parents, hence, it has derived its Sr11 gene from Timstein (Knott et al. 1956 detected Sr11 in Timstein). Kenya Leopard and Wisconsin 245-II-50-17 have sister lines, C.I.13632 and C.I.13633 progenitors respectively. In the present studies the mono genes KLD7 and WIS7 were found to be identical. The two varieties, WIS and KLD, are likely to have inherited the mono genes KLD7 and WIS7 (unidentified identical genes) from these two sister progenitors which convey resistance against race EA7. Allard et al. (1954) reported that lines C.I.13632 and C.I.13633 have at least two linked genes in common for stem rust resistance. Tobari 66 has K324, Timstein, Newthatch in its parentage. It is therefore expected that Sr11 was inherited from Timstein while most probably K324 provided the Sr6 gene. The other new gene TBR7 might have come from other parents such as Tzpp, Mentana or Marroqui. Minnesota 3654/60 on the other hand has Frontana, Kenya 58, Newthatch and Pilot for its parents. Therefore it might have inherited the Sr6 from K58 and Sr7 from Newthatch. The other recessive genes detected in the present studies could not be attributed to any of its parents since no previous records are available.

That the progenies or derivatives from any cross might not inherit all the resistance genes from their progenitors was not unusual. Hence the genotypes of the eight donor parents cannot necessarily correspond fully with the combined parental genotypes. For example, Frontana which

carries Sr8 (Knott et al. 1956) has been used extensively in the Kenya breeding programme. However, in 1962 Knott could not detect Sr8 from any Kenya variety or line. Likewise Sr8 was not detected in the present experimental material which were extracted from the Kenya established and SRPC cultivars and strains though the differential race C17(56) was used in the present studies.

The number of genes predicted in this thesis for each of the varieties; KHR, KLD, TBR, CLY, CIF, WIS, TPY and MIN, are valid as far as the present test races are concerned, but it is doubtful that these are the only loci homozygous for genes conditioning resistance in these varieties. Actually all of these varieties probably have additional homozygous genes that condition resistance. These were not apparent in these studies presumably because of the tester pathogens used and perhaps because of the similarity of loci for resistance present in the resistant and the susceptible parents used. Thus one can never be sure that, in a genetic study of only pathogenicity of the pathogen or only reaction of the host, one has demonstrated the presence of all the genes conditioning avirulence in the pathogen or resistance in the host.

The use of genetic symbols such as KHR4 or CLYC17a adopted in this thesis was intended as a temporary device only. When the respective loci postulated herein have been proved beyond doubt by further experiments, the loci involved shall then be given Sr or sr designations in series with the already numbered genes as proposed by Ausemus et al. (1946).

From the present evidences it was very tempting to conclude that the

existence of a large number of physiologic forms was a less serious threat from a breeding point of view than it first appeared. It was clear that the inheritance of resistance governed by a single gene pair such as MIN4, KHR4 or Sr11 protected several varieties from a wide spectrum of races. MIN4, for instance, conveyed resistance against races EA4, EA5, EA11, EA12, EA13, EA14, EA15, EA16 and to the North American races C1(17), C2(17A), C18(15B-1L), C22(32), C25(38), C41(32-113) and probably to many more races which were not included in the present studies. Thus any line of a cross involving MIN4 which is resistant to one of these forms will, in all probability, also be resistant to other races in this group.

The present results also revealed that it must not be supposed that all the races are characterized by the same type of reaction on different varieties or lines though the same reaction gene(s) might be involved. As a matter of fact the differences were striking for certain host-pathogen relationships. A comparison between the reactions to races C10 and C17 (TABLE 5:33) will serve to illustrate this point. MINC17a (Sr6) exhibited 2$^-$ infection type to C10(15B-1). The same mono gene produced ;1 to C17(56). In contrast KLD17 (Sr6) exhibited 2,3 infection type when inoculated with C10(15B-1) but the infection type was again ;1 when inoculated with C17(56). This observation is in agreement with most of the recent works with stem rust of wheat (Green et al. 1960, Loegering et al. 1967 and Bârtos et al. 1970).

In the course of this project to determine the mode of inheritance, isolation of genes, their interrelationship and spectrum of effectiveness,

a number of accomplishments and conclusions reached have so far been discussed in this chapter.

The significance of the presented results to the wheat improvement programme in Kenya will be discussed briefly. The mono gene lines described here could be used in both basic and applied research. With regard to applied research, it is advocated that one of the prime necessities in the successful breeding for rust resistance is gene identification and gene management. Too often it is not known whether a group of resistant wheat cultivars possess the same or different genes for resistance. Some progress in this direction has been accomplished in the present investigations.

The knowledge of the major genes implicated in a resistant reaction should help to plan intelligently a planting schedule for varieties to escape the rust onslaught. For instance Sr6 is well known to be influenced by temperature fluctuations. Hence varieties, such as Trophy which might depend on a temperature sensitive gene like Sr6 for their resistance to certain EA races would normally be recommended for planting earlier in the season to enable them to ripen before the high temperature renders the gene ineffective.

The isolated mono genes should also be incorporated into the Kenya stem rust differential system. Fruitful results are envisaged from the use of mono genes as differential hosts because epistatic gene interaction and modifier effects will be greatly eliminated. The factors mentioned above alter the infection types in response to environment causing

identification of races most difficult and unreliable.

The programme to determine the interrelationships of the genes for rust resistance carried by various varieties including all the Stem Rust Parental Collection (SRPC) cultivars, will be continued and extended using the present monogenic lines as tester checks. This procedure should enhance greatly the grouping of the SRPC material and other sources of resistance in an orderly system on the basis of resistance genes. Eventually, by applying various suitable breeding methods these genetically identifiable material will be deployed to evolve cultivars of superior resistance to wheat black stem rust.

Another important feature of the present work is the extensive use of test crosses involving the Marquis lines carrying single genes for rust resistance. This procedure should, in the future contribute immensely to the identification programme at PBS by making it relatively easier and faster to determine whether a variety carries any of the known genes for resistance or possess unknown genes.

The present investigation had its weaknesses too. Only six races of black stem rust were employed in the isolation of the monogenic lines and for the genetic analysis. Therefore genes which provide resistance to races other than those studied were not detected. Furthermore only a small number of races were employed in order to gain some idea on the spectrum of resistance of the isolated monogenic lines. This shortcoming is due to the laborious technique incident to the identification of these genes; the number of cultures of both races and host populations

and generations that can be handled is much more limited than if the character studied were readily discernible by visual inspection. Also a precise localization of the genes was not completed. Nevertheless, the work is in progress on monosomic and telosomic analytical techniques which will give a more accurate determination of the location and the association of the presently described mono genes. A remarkable difficulty in the present studies on the mode of inheritance was to decide on plants which were resistant and which to classify as susceptible. An arbitrary division was set up: all the seedlings having infection type 3⁻ or better were classified as resistant. Unfortunately such a classification might not always be genotypic although in the present investigations, the ratios obtained indicated that the "standards" set were reliable in predicting the genotypes.

More backcrosses to the newly discovered genes are being continued for complete transference of these genes to Hindi 62 background. When the backcrossing is completed, the desired lines will be sent to other scientists to ascertain the spectrum of resistance and further identity of these mono genes. Ultimately the genes shall be given Sr gene designations.

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