THE UNIVERSITY OF MANITOBA

OCCURRENCE OF PHYSIOLOGIC RACES OF LOOSE SMUT OF WHEAT, *Ustilago tritici* (Pers.) Rostr., IN BRAZIL, SOURCES OF RESISTANCE AND INHERITANCE OF RESISTANCE TO RACE T 2 IN THE INTERVARIETAL CROSS KENYA 340 Y.4.A.1. x IAS 52

by

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A THESIS

SUBMITTED TO THE FACULTY OF GRADUATE STUDIES

IN PARTIAL FULFILMENT OF THE REQUIREMENTS FOR THE DEGREE

OF MASTER OF SCIENCE

DEPARTMENT OF PLANT SCIENCE

WINNIPEG, MANITOBA

February 1976

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

MASTER OF SCIENCE

ACKNOWLEDGEMENTS

The writer wishes to express his sincere appreciation to his advisor Dr. J. J. Nielsen, for suggesting the field of study and for his guidance and encouragement throughout the study and in the preparation of the manuscript.

Grateful acknowledgements are made to Dr. W. C. McDonald, Director of the Agriculture Canada, Research Station, Winnipeg, for the facilities provided.

Special thanks are extended to Mr. Alex Holowko for his technical help in greenhouse and growth cabinets.

The author is also indebted to the Food and Agriculture Organization of the United Nations, to the Ministério de Agricultura do Brasil, the Empresa Brasileira de Pesquisa Agropecuária - EMBRAPA, and to the Açã̃o Moageira de Fomento ao Trigo Nacional, whose financial assistance made these studies possible.

In addition to those mentioned, the author is grateful to many others who, directly or indirectly, contributed to this study.

ABSTRACT

OCCURRENCE OF PHYSIOLOGIC RACES OF LOOSE SMUT OF WHEAT, *Ustilago tritici* (Pers.) Rostr., IN BRAZIL, SOURCES OF RESISTANCE AND INHERITANCE OF RESISTANCE TO RACE T 2 IN THE INTERVARIETAL CROSS KENYA 340 Y.4.A.1. x IAS 52

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The variability in Brazil of loose smut of wheat, *Ustilago tritici* (Pers.) Rostr., was studied, based on the reaction of a set of differential cultivars. Twelve physiologic races were identified amongst the 50 collections of spores that were tested. Five of the races would be classified as being similar to Canadian race T 2, but further differentiation was possible by using three supplemental differentials. Two races gave a reaction identical to race T 8 with the standard set of differentials, but they too could be further differentiated on the supplemental differentials. None of the five remaining races resembles any Canadian race previously identified. Four of them are virulent on the durum wheat Pentad and on several cultivars of common wheat in the set of differentials. This appears to invalidate claims that *formae speciales* of *U. tritici* exist on common and on durum wheat.

A collection of 68 cultivars of diverse origin has been established that are resistant to races of loose smut from Brazil, Canada and other countries. Resistance was

confirmed of the cultivars Sinvalocho M.A., Maria Escobar and their derivatives, whose resistance had been reported earlier.

The inheritance of resistance to race T 2 of loose smut was studied in the intervarietal cross Kenya 340 Y.4.A.1. x IAS 52. It was found that the incompatible reaction to race T 2 of the resistant parent Kenya 340 Y.4.A.1. was dominant. Based on the performance of F3 progenies, it appears that a single gene is responsible for the resistance displayed by Kenya 340 Y.4.A.1., but that this gene is independent from those causing the incompatible reaction.

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CHAPTER 1

INTRODUCTION

Loose smut of wheat, caused by the fungus Ustilago tritici (Pers.) Rostr., is one of the diseases of wheat found in Brazil, and all cultivars recommended for cultivation in that country are to some degree susceptible. Data on overall annual losses to this disease are lacking but, in individual fields, levels of infection up to 10% have been recorded causing an equally high loss in yield. Although seed treatments in the form of modern fungicides are available today, breeding for resistance remains the most economical form to prevent the disease.

Before a programme of breeding for resistance to loose smut can be initiated in Brazil, it is essential to obtain information on certain aspects of the disease. First, since loose smut can be subdivided into physiological races that differ in virulence, it should be determined whether such races occur in Brazil and, if so, their characteristics should be established. Secondly, sources of resistance to these races had to be found. And, thirdly, the mode of inheritance of resistance to one of the prevalent Brazilian races, located in a suitable donor, should be studied using one of the presently grown Brazilian cultivars as the susceptible parent. Accordingly, the three phases of the present study followed these objectives.

CHAPTER 2

REVIEW OF LITERATURE

2.1. Physiologic races of loose smut of wheat

The first indication that physiologic races of loose smut of wheat occur appears to have come from Piekenbrock (1927). By inoculating a number of cultivars of wheat with several collections of spores, he was able to identify two physiologic forms. Piekenbrock's work was continued by Grevel (1930) who studied 19 collections of loose smut from Germany, and 29 from countries other than Germany. These collections yielded four physiologic races, three from the German collections, while the fourth race originated from Turkey.

Since then, physiologic races of loose smut of wheat have been found in many countries, e.g. in Bulgaria (Mitov 1958), France (Simon and Croisier 1959), Great Britain (Batts 1955; Doling and Hervey-Murray 1966), The Netherlands (Oort 1944), Poland (Heinrich 1973), Romania (Radulescu 1935), USSR (Krivchenko 1970), India (Dastur 1946), China (Wang 1942), New Zealand (Cunningham 1940), South Africa (Gorter 1964), USA (Bever 1947, 1953).

In Canada, Hanna (1937) described four physiologic races of loose smut of wheat which he identified in Winnipeg from three collections of spores originally made in 1929 from the cultivars Reward, Kota and Mindum. Later, Cherewick (1953) described the ten physiologic races that had been differentiated in Canada up to that time by using, with few

modifications, the group of cultivars selected by Oort (1944) in The Netherlands. At present, the identification of races in Canada is still based on the group of differential cultivars used by Cherewick (1953) although four additional cultivars have been added to identify races not differentiated on the cultivars used by Cherewick.

Unfortunately, the absence of a standard group of differential cultivars, and the different methods of classification of the cultivars regarding reaction to races of loose smut, do not permit a comparison of the results obtained by workers in different countries. Therefore, in the present study, the pattern of virulence of physiologic races from Brazil can only be compared with that of races under study in Canada.

Very little is known about pathogenic specialization of loose smut in South America. In Brazil, Silva (1951) noted possible differences in pathogenicity occurring within the population of loose smut in the State of Rio Grande do Sul. He based this on the fact that the cultivar Planalto was resistant when artificially inoculated with samples from one region of that State, whereas the same cultivar was naturally infected in another region. Mascarenhas and Silva (1954) working with three samples of spores collected in the State of Rio Grande do Sul, and one from the State of Parana, found differences in pathogenicity between the samples when a collection of commercial cultivars was tested in Pelotas, Brazil, during the period 1950-54. Although these two

studies suggested the presence of physiologic races of loose smut of wheat in Brazil, they gave no information as to their number, nor to their variability in pathogenicity.

In all of the above-mentioned studies, the classification of a race was based on one of two reactions of the host-parasite system: spore-formation, or no spore-formation. However, only Oort (1944) has pointed out that a failure to produce spores, which was normally equated with resistance, could be caused not only by physiologic resistance in the proper sense, but also by hypersensitivity of the host. From observations made and experiments carried out, Oort concluded that two different principles were involved in the reaction of plant and parasite. The first was a principle of susceptibility or non-susceptibility; it determines whether the plant will be resistant (no symptoms) or susceptible (shows symptoms). The second principle was that of hypersensitivity or non-hypersensitivity and this determines whether the susceptible plant will show abnormal symptoms, or normal ones with smutted ears. The abnormal symptoms, or hypersensitive reaction of wheat to loose smut, were described by Oort (1944) as follows:

> A strong growth inhibition, which is shown by a shortening of the first three leaves, is the most striking symptom. It may be accompanied by chlorotic stripes and spots and by malformation causing a curling of the leaves. These symptoms appear only clearly when the inoculated seed is sown in the greenhouse under favourable conditions. Many plants showing these symptoms die

> in the two- or three-leaves stage. The survivors recover slowly either by the main axis coming to new growth, or, when this dies, by the developing of side-shoots. The plants which recover remain in all dimensions much smaller than normal plants and are - with a few exceptions - healthy, i.e. they show no smut.

Since no spores are formed on these hypersensitive plants they were termed "field resistant" by Oort. Later, Oort (1963) worked out a genetical model to fit his hypothesis. Two pairs of independent factors controlled the reaction of the host to a given race: one pair determined resistance or susceptibility, the other pair determined whether the plant, if susceptible, would react with hypersensitivity or not. Correspondingly, the parasite also had to carry two pairs of independent factors: one pair of genes determined avirulence or virulence; the other pair determined whether hypersensitivity could be overcome or not.

As mentioned above, the phenomenon of hypersensitivity observed by Oort was not used by other authors as a characteristic to distinguish physiologic races of loose smut of wheat. On the other hand, Kiraly and Lelley (1957) studying six winter wheat cultivars aritificially inoculated with loose smut, confirmed the presence of hypersensitivity in some wheat-smut combinations. They examined inoculated wheat plants for the presence of smut in the culms after heading and while they found the fungus in one half of the depressed plants, it never reached the highest node. In the culms of plants which did not show a hypersensitive reaction, the fungus could not be detected. They concluded

that hypersensitivity of the host either restricts spread of the parasite so that it cannot reach the organ where it could insure its reproduction, or it totally eliminates the fungus and the host recovers from the disease. Mantle (1961), after anatomical examination of plants with abnormal reaction to loose smut of wheat, showed that the term "hypersensitivity" was not the proper one to use for the observed phenomenon. A hypersensitive reaction to fungal invasion is usually defined as being a very localized necrosis, thereby preventing further growth of the parasite. In the abnormal reaction of wheat to loose smut, however, the whole plant is affected, and growth of the parasite is retarded. Mantle (1961) used the more general "incompatibility", and this term will be used in the present study, although even it may no longer be acceptable today. A further clarification concerns the terms "infection" or "infected plants". In the following they are used in the restricted sense of being synonymous with "sporulation" or "plants that show spore-formation"; they do not include plants that show incompatibility which undoubtedly had been infected also.

2.2. Sources of resistance to loose smut

Fischer and Holton (1957) stated that wheat cultivars may react with different degrees of resistance or susceptibility to loose smut. Such reactions are common amongst many different cultivars in widely separated geographical areas. In some cases, resistant and susceptible reactions seemed to be related to the species or type-group of cultivars, while similar relationships could not be established in other cases. After a review of early works the authors said:

> In sum total, the information on varietal resistance has established the availability of high resistance to loose smut in a limited number of commercial varieties and in a wide range of breeding stock. Thus, the development of new, agronomically desirable types with high resistance to loose smut is assured through appropriate breeding procedures based on the nature of inheritance of resistance factors.

For a given area, such a breeding program will usually be based on a screening of the reaction of adapted local cultivars and foreign introductions to smut by means of artificial inoculation with races or field collections from that area. In South America, only a few such studies have been done. Silva (1951) tested 127 wheat cultivars in Brazil with one field collection of loose smut. He found the following to be resistant: Ardito, Bandeirante, Barbela 2711, Bonaerense, Celebration, Charuto, F.P.I. 104137 x 41-116, Florence, Garnet, General Vargas, Hope, (Hope x Med. x A.M.) x Gaza, Joana, Kendee, Klein Acero,

Klein Orgullo, Klein Universal, M-2-38, M-18-38, Maia 9994, Mindum, Minn. 2676, Nordeste, Newthatch, Pilot, Planalto, Purplestraw, Pusa 52, Rival M-4-38, Renascimiento and Santa Marta (37/46). Mascarenhas and Silva (1954) tested 47 cultivars with one field collection of loose smut. The cultivars they found to be resistant were among those found resistant in the earliest test (Silva 1951). A cultivar tested for the first time was Sinvalocho M.A., and it was immune.

In Argentina, Cenoz (1952) inoculated a collection of 441 cultivars at Castelar, Buenos Aires, with two field collections of smut from common wheats. His purpose was to determine the reaction of all cultivars grown in Argentina at that time, as well as of some selected foreign cultivars. Sources of resistance would thus be determined which could be used in Argentina and neighboring countries. As a result of the tests performed between 1945-49, the cultivar Sinvalocho M.A. was the only Argentine cultivar that was determined to be immune to loose smut. Other local cultivars that showed high resistance were: 38 M.A., Buck Quequen, Klein Aniversario, Klein Cometa, Klein Exito, Klein Orgullo, Klein Otto Wulff, Klein Progreso and Reliance Sel. Klein. Almost all of these cultivars have one of the following in their pedigree: Marquis, Reliance, Klein 49a, or Chino 466. Among the foreign cultivars the following were immune: Apex, Axminster, Carina, Chino 466, Chul, Dixon, Fultz, Giza 121,

Heines Kolben, Kendee, Maria Escobar, Newthatch, Rapier, Redhart Strain 5 and Riosulino. All durum wheat cultivars tested, including Mindum and Pentad, were either immune or highly resistant.

Later, in Argentina, Frecha (1967) reported on tests he had conducted from 1963-66. In these, 80 cultivars and lines of wheat including Argentine cultivars and interesting breeding material were artificially inoculated with a mixture of an unknown number of field collections of loose smut. Among the Argentine cultivars, El Gaucho F.A. was found to be immune, whereas Agrolit Vagliano, Pergamino Gaboto M.A.G. and Olaeta Artillero were highly resistant. Among the breeding material the following were immune: Barleta Benvenuto, (Chin. x Aeg. umbellulata) x Thatcher,[6] Chino 466, Dundee x Kenya B.C.4/1.2.1.1., Egypt Na 101, Magnif MG, Maria Escobar, Massaux 5, Sinvalocho M.A. and 38 M.A. With very few exceptions, the cultivars found to be immune or resistant by Frecha were those found to be immune or resistant in the tests reported on by Cenoz (1952). The exceptions were the cultivars Klein Orgullo and Klein Aniversario, reported to be resistant by Cenoz, but susceptible in the tests of Frecha (1968). Probably one of the collections used by Frecha carried virulence on these cultivars, while the virulence of the rest of his inoculum was identical to the virulence of the inoculum used by Cenoz (1952).

A deficiency in these screenings of cultivars for resistance to loose smut in Brazil and Argentine, was the low number of field collections used in the inocula. Also, without knowledge of the pathogenic variation within these few collections and within the population as a whole, the cultivars were likely to have been exposed to only a small part of the virulence present in those countries.

2.3. Inheritance of resistance to loose smut

According to Kilduff (1933) the first report on inheritance of resistance to loose smut of wheat was made by Olson et al. (1920). A review of their work by Matsuura (1929) states: "There are several genes involved for resistance to smut caused by Ustilago tritici in different varieties."

Piekenbrock (1927) working with crosses of a highly resistant cultivar and two susceptible spring wheats found that segregation in the F2 and F3 generations indicated that immunity was inherited recessively. Grevel (1930) confirmed Piekenbrock's conclusion.

Kilduff (1933) studied the F3 and F4 generations of the crosses Kota x Red Bobs and Kota x Garnet. Although Kota was relatively susceptible, Garnet resistant, and Red Bobs immune to the collection of loose smut used, Kilduff could not fit the levels of infection in the segregating material into any ratio that would be indicative of the action of either one or a few genes. He suggested that the resistance of the parental cultivars might have a basis other than physiological.

Rudorf and von Rosenstiel (1934) indicated that the resistance to loose smut of the cultivar 38 M.A. probably depended on three recessive factors. They concluded this from the reaction of the F3 generation of a cross with the susceptible cultivar San Martin.

Tingey and Tolman (1934) made the crosses Hope x Federation, Preston x 01-24 and Hope x Dicklow. They concluded from the segregating generations that at least three factors were involved in the inheritance of resistance to loose smut, that resistance was dominant, and, though dominance was incomplete, the factors had a cumulative effect.

Heyne and Hansing (1955) observed that resistance of Kawvale to race 11 of U. tritici was dominant over the susceptibility of Clarkan and dependent upon at least two factors.

Based on the segregation observed in F3 lines of the cross Thatcher x Redman Selection, Campbell (1948) concluded that the near immunity of Thatcher to the Canadian race 1 of loose smut was controlled by a single gene, which was probably dominant.

Mathur and Kohli (1963) studied the F1, F2 and F3 of a cross between the resistant cultivar N.P. 824 and the susceptible Rio Negro and concluded that resistance was dominant and monogenically controlled.

Agrawal et al. (1963) studied the inheritance of resistance to loose smut in the cross N.P. 775 x N.P. 798. From the reaction of the F1, F2 and F3 they concluded that the resistance of N.P. 798 was conditioned by two pairs of dominant duplicate genes.

Agrawal and Jain (1965), after observing the reaction of the F1, F2 and F3, reported that loose smut resistance of N.P. 790 in a cross with the highly susceptible N.P. 775 was governed by a single dominant factor.

Shestakova and Vjushkov (1974) pointed out that in the material they studied, resistance was determined by a small number of genes with strong additive and weak dominant effects. Accordingly, immunity to race 16 of the cultivar "Bezenchukskaya 98" was determined by 3 genes; the high resistance of "Saratovskaya 36" by 2 genes, and moderate resistance of "Saratovskaya 29" by a single gene.

CHAPTER 3

MATERIALS AND METHODS

3.1. Physiologic races of loose smut of wheat in Brazil

Fifty samples of loose smut were used to study the variability of the loose smut fungus in Brazil. In order to reduce the possibility of working with a mixture of races each sample consisted of only one smutted head. Some of the samples were collected at Winnipeg on cultivars originally inoculated with mixture A or B. Mixture A was made up from spores collected on different cultivars in different areas of Brazil; mixture B was formed only from spores collected on the naturally infected Brazilian cultivar IAS 52. Other samples of spores were received from Brazil in 1973 and these had been collected on naturally infected cultivars (identified by N in table below). Table 1 gives details of the samples tested.

The following differential cultivars were used to identify the virulence pattern of the Brazilian samples of loose smut: (1) Mindum, (2) Renfrew, (3) Florence x Aurore, (4) Kota, (5) Little Club, (6) (van Hoek), (7) Reward, (8) Carma, (9) Kearney, (10) Red Bobs, (11) Pentad, (12) Thatcher x Regent, (13) P.I. 298 554, (14) Sonop and (15) H44 x Marquis.

The above set of cultivars is used at Agriculture Canada, Research Station, Winnipeg, to identify physiologic races of loose smut of wheat. However, this set had to be supplemented by the cultivar Klein 40, because in 1973 it

TABLE 1. Origin of samples used in the study of physiologic races of loose smut from Brazil.

Sample	Collected on cultivar	at	after inoculation with
1	IAS 52	Winnipeg	A
2	IAS 52	Winnipeg	A
3	IAS 52	Winnipeg	A
4	IAS 52	Winnipeg	A
5	IAS 52	Winnipeg	A
6	IAS 52	Winnipeg	A
7	IAS 52	Winnipeg	A
8	IAS 52	Winnipeg	A
9	IAS 52	Winnipeg	A
10	IAS 52	Winnipeg	A
11	L. 8220-54	Winnipeg	A
12	PF 72574	Passo Fundo	N
13	Klein 40	Winnipeg	A
14	IAS 52	Winnipeg	B
15	Line 1262-26	Winnipeg	B
16	PF 72576	Passo Fundo	N
17	Klein 40	Winnipeg	B
18	Kenya 4121	Winnipeg	B
19	Parana 62/1845	Winnipeg	A
20	Parana 62/1845	Winnipeg	B
21	IAS 50-Alvorada	Vacaria	N
22	IAS 50-Alvorada	Passo Fundo	N
23	IAS 51-Albatroz	Passo Fundo	N

TABLE 1. Origin of samples used in the study of physiologic races of loose smut from Brazil. (Cont.)

Sample	Collected on cultivar	at	after inoculation with
24	IAS 52	Vacaria	N
25	IAS 52	Passo Fundo	N
26	IAS 52	Passo Fundo	N
27	IAS 56	Passo Fundo	N
28	IAS 60	Vacaria	N
29	IAS 60	Passo Fundo	N
30	IAS 62	Vacaria	N
31	Cinquentenário	Vacaria	N
32	Encruzilhada	Passo Fundo	N
33	IAS 51-Albatroz	Passo Fundo	N
34	Pat 16	Passo Fundo	N
35	Pat 49	Passo Fundo	N
36	PF 70338	Passo Fundo	N
37	PF 70412	Passo Fundo	N
38	PF 70440	Passo Fundo	N
39	PF 70592	Passo Fundo	N
40	PF 70358	Passo Fundo	N
41	PF 7110	Vacaria	N
42	PF 7159	Passo Fundo	N
43	PF 7197	Passo Fundo	N
44	PF 71108	Passo Fundo	N
45	PF 71111	Passo Fundo	N

TABLE 1. Origin of samples used in the study of physiologic races of loose smut from Brazil. (Cont.)

Sample	Collected on cultivar	at	after inoculation with
46	PF 72390	Passo Fundo	N
47	PF 72121	Passo Fundo	N
48	PF 72199	Passo Fundo	N
49	PF 72202	Passo Fundo	N
50	PF 72238	Passo Fundo	N

was found that the previously resistant Klein 40 was susceptible to race T 9, which originated in Czechoslovakia (Nielsen, unpublished results), as well as to samples of smut from Brazil. Two single-plant lines, SPL 1 and SPL 2, from the Brazilian cultivar IAS 52 were also included as supplemental differentials. These two lines were selected from a set of five single-plant lines of IAS 52 that served to determine which race should be used in the study on inheritance of resistance. When inoculated with Canadian races T 1 to T 10 it was found that the first line (SPL 1) differed in its reaction from the other four (SPL 2 among them). SPL 1 showed low susceptibility to T2 and high susceptibility to T 8, but lines 2 to 5 were highly susceptible to T 2 only.

The differential cultivars were grown in pots in a greenhouse. At mid-anthesis, three heads of each cultivar were inoculated (using hypodermic needle and syringe) with a suspension of spores from each of the 50 samples of spores. Inoculated seed was grown in the field and percent infection established from an estimate of healthy and diseased heads.

Samples that gave identical patterns of virulence were grouped, a typical sample was selected from each group, and a single infected head collected from one of the differentials. Spores from this head were used to reinoculate a second set of differentials to repeat the test. However, the test was not repeated with three groups in which the

patterns of virulence were very clear in the first experiment. To cut down further on the space required in growth cabinets and greenhouses, and since all 50 samples were avirulent on differentials 12 (Thatcher x Regent) and 14 (Sonop) these cultivars were not reinoculated. The differential hosts for this test were raised in growth-cabinets; the seed from inoculated heads was sown in greenhouses with the presence or absence of symptoms of incompatibility being recorded in the seedling stage. Later, percent infection was based on counts of plants with and without spore-formation.

3.2. Sources of resistance to loose smut from Brazil

A collection of cultivars resistant to races of loose smut from Canada and from several other countries (Nielsen, unpublished results) was planted at Passo Fundo, Rio Grande do Sul, Brazil. These cultivars were inoculated with the two mixture of spores from Brazil, A and B, described earlier.

Field inoculation was performed using the high pressure jet spray method (Moore and Munnecke 1949). The cultivar IAS 52 was inoculated as a check. The seed from inoculated heads was planted in 1973 in greenhouses at Winnipeg. At heading, the number of healthy and infected plants per cultivar was recorded and the infection expressed in percentage.

3.3. Inheritance of resistance to loose smut.

The intervarietal cross Kenya 340 Y.4.A.1. x IAS 52 was used to study the inheritance of resistance to loose smut.

The following considerations influenced the selection of these parents. The susceptible parent should be a cultivar grown commercially in Brazil. Accordingly, cultivar IAS 52 was chosen, since it was known to be susceptible to loose smut in the field. The other parent should be resistant to all known races of loose smut and should give an incompatible reaction with a race to which IAS 52 was susceptible. The reason for this choice was that as far as one can gather from the literature, none of the previous studies on the inheritance of resistance appears to have been undertaken employing a parent where non-sporulation was based on incompatibility instead of true resistance. However, use of an incompatible parent to arrive at resistance to loose smut in a breeding programme could be an advantage. Since incompatible plants can be identified in the seedling stage, and since some of these plants recover to produce normal florets and seeds, the selection of resistant plants and their use in possible backcrosses in successive generations would be accelerated. Therefore, cultivar Kenya 340 Y.4.A.1. was chosen as the other parent to determine the mode of inheritance of resistance based on incompatibility. This cultivar was earlier found to be resistant to all

Canadian races and to races from several other countries (Nielsen, unpublished results) as well as to spore mixtures A and B from Brazil (see previous section). Preliminary tests had also shown that Kenya 340 Y.4.A.1. reacted with incompatibility after inoculation with Canadian race T 2, the only race to which all single-plant lines of IAS 52 were found to be susceptible. Kenya 340 Y.4.A.1. showed typical symptoms of incompatibility at the seedling stage, but most plants recovered later without sporulation.

The original cross Kenya 340 Y.4.A.1. x IAS 52 was made in Passo Fundo, Brazil. The F1 and F2 were grown in growth cabinets at Winnipeg in 1973 and 1974. To study the inheritance of resistance on the basis of the performance of F3-progenies, two heads on each of 122 F2-plants were inoculated with Canadian race T2, using a hypodermic needle and syringe.

The inoculated F3-lines were grown in a greenhouse, and symptoms of incompatibility recorded in the seedling stage, and at heading percent infection was determined after counting healthy and infected plants.

A second cross of Kenya 340 Y.4.A.1.x IAS 52 (SPL 2) was made at Winnipeg to determine whether the incompatibility displayed by Kenya 340 Y.4.A.1. was dominant or recessive. The inoculation with race T2 was performed two days after pollination.

CHAPTER 4

RESULTS AND DISCUSSION

4.1. Physiologic races of loose smut in Brazil

Altogether 12 groups of samples, each with a distinctive virulence pattern, could be recognized amongst the 50 samples of loose smut from Brazil. The reaction of the differential cultivars to the 12 samples selected as representative of each of these groups are shown in Table 2. A few Canadian races that give identical reactions to some of the Brazilian samples tested, or that are discussed in the text, are also entered in this table.

The properties of these samples, discussed singly, were as follows. Sample 1 was chosen as being representative of the virulence pattern displayed by a group of 11 samples, the largest group recognized. This sample had a virulence similar to Canadian race T 2 on the standard set of differentials, as well as on the three supplemental differentials, of which Klein 40 was incompatible, but SPL 1 and SPL 2 were susceptible. However, since differential 14 (Sonop) was not reinoculated, it is not known whether it reacted with resistance or incompatibility to sample 1.

The virulence pattern as represented by sample 5 was found in a total of five samples. Based on the first test planted in the field, their pattern on the standard differentials again resembled that of Canadian race T 2, but in the three supplemental hosts Klein 40 was now susceptible, as were SPL 1 and SPL 2. This pattern changed when spores

TABLE 2. Reaction of differential cultivars to Brazilian samples and to Canadian races of loose smut.

Differential	Reaction[1] to inoculation with Brazilian samples												Canadian races					
	1	5	19	18	17	34	12	15	46	31	35	50	T 1	T 2	T 3	T 4	T 8	T 14
1 Mindum															S	S		
2 Renfrew	S	S	S	S	S	S					S			S				
3 Flor. x Aur.	S	S	S	S	S	S				S	S	S		S				
4 Kota							S	S			S	S	S				S	
5 Little Club							S	S	S	S	S	S	S				S	
6 (van Hoek)						S							S					
7 Reward	S	S	S	S	S	S	S	S	S	S	S	S	S	S			S	
8 Carma	S	S	S	S	S	S	S	S	S	S	S	S		S			S	
9 Kearney							S	S	S	S	S	S					S	
10 Red Bobs	S	S	S	S	S	S				S	S	S		S				
11 Pentad									S	S	S	S				S		S
12 Th. x Regent																		
13 P.I. 298 554	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S
14 Sonop														I				
15 H44 x Marquis							S	S		S	I	I					S	
Klein 40	I	S?	I	I	S	I	I	S	I	S	I	I	I	I			I	I
SPL 1	S	S	I	S	S	S	S	S	I	S	S	S		S			S	-
SPL 2	S	S	I	I	I	S	I	I	I	S	I	S		S			I	-

1) For clarity of this table, only the incompatible (I) and susceptible reactions (S = over 10% infection) are given; resistant combinations are left blank.

from a single head collected on the differential Renfrew inoculated with sample 5, were used for reinoculation of the differentials. This time a typical reaction of incompatibility on Klein 40 was observed. In order to clarify this contradictory result, Klein 40 was again inoculated with both the original sample, and with the spores collected on Renfrew. The seeds from the inoculated heads were planted in the field in 1975, and confirmed the results obtained in the greenhouse, i.e., Klein 40 was susceptible to the original sample but incompatible with the sample from Renfrew.

Since the original sample was collected on the Brazilian cultivar IAS 52 which had been inoculated with a mixture of spores, it appears that the smutted head used for the original inoculum was infected by two races - one to which Klein 40 was susceptible, and the other to which it was incompatible. In this case, the smutted head that was collected on Renfrew and used to confirm the results was infected only with the race incompatible with Klein 40. Although sample 5 as used for the repetition gave the same virulence pattern as sample 1 (or Canadian race T 2), the existence of samples that do carry virulence on Klein 40 and on both SPL 1 and 2 cannot be doubted, since one of the four other samples in this group (13), that gave this pattern in the first test was collected on Klein 40 itself. It is further highly unlikely that the other three were also infections of a single head by more than one race.

The virulence pattern of sample 19 was found only once. In contrast to samples 1 and 5, an incompatible reaction was observed on SPL 1 and SPL 2. The original sample was collected on the cultivar Parana 62/1845 after inoculation with mixture A. As will be shown later, this inoculum caused an incompatible reaction in many plants of IAS 52. Since SPL 1 and SPL 2 are single-plant lines from IAS 52, it is reasonable to expect to find a strain with the characteristics of sample 19.

The virulence pattern displayed by sample 18 was also found only once. The original sample had been collected on the cultivar Kenya 4121 inoculated with mixture B. The virulence pattern of this sample as shown in Table 2 is based only on the results obtained in the field with no subsequent re-inoculation. However, SPL 1 showed 90% of infection and SPL 2 showed typical symptoms of incompatibility, with no spore-formation at all. This demonstrated that besides differing in their reaction to Canadian races T 2 and T 8, the single-plant lines SPL 1 and SPL 2 could also separate strains of the pathogen, like samples 1, 19 and 18, that gave apparently identical reactions on 16 other differential cultivars.

Sample 17 was originally collected on Klein 40 which had been inoculated with mixture B. Like sample 18, its virulence pattern is based only on results from one inoculation of the set of differentials. Susceptibility of Klein

40, and the different reactions of the two single-plant lines of IAS 52 distinguish it from the four samples already described.

Without the three supplemental differential cultivars, the five samples so far described would all have been classified as being identical. As with other host-parasite systems, the greater the number of genotypes of the host that are exposed to apparently identical strains of the pathogen, the greater the likelihood that differences between these strains will be found. This reminds us, how arbitrary and, for practical reasons, limited, the concept of physiological races is in the smuts.

The virulence pattern of sample 34 has no equivalent among Canadian races. However, it appears to be closely related to race T 2 and Brazilian sample 1, but with added virulence on differential 6, (van Hoek).

The virulence patterns of samples 12 and 15 are identical to that of Canadian race T 8 if only the reaction of the 15 cultivars in the standard set of differentials are considered. Yet sample 15 can be differentiated on Klein 40 which shows incompatibility with sample 12 and race T 8, but is susceptible to sample 15.

Three of the four samples 46, 31, 35 and 50 displayed an unusually broad virulence that is unmatched in any Canadian race. Samples 35 stands out in particular, with 10 of the 15 standard differentials being susceptible, and one incompat-

ible. Of special interest, however, is another feature common to the four samples. The original samples were collected on cultivars of common wheat in Brazil, but besides being virulent as expected on several differentials of common wheat, the samples are also virulent on differential 11, Pentad, a durum wheat. None of the Canadian races that are virulent on any common wheat differential have this characteristic. Conversely, none of the races (T 3, T 4 and T 14) that are virulent on the durum differentials Mindum or Pentad are virulent on a common wheat, exept on the general suscept, P.I. 298 554. Similarly, Mitov (1968) found the races of loose smut in Bulgaria to be virulent on either durum, or common wheat, but not on both. Accordingly, Mitov proposed the trinomials U. tritici (Pers.) Jens. f. sp. tritici duri, and U. tritici (Pers.) Jens. f. sp. tritici aestivi.

The four samples 46, 31, 35 and 50 do not support the claim by Mitov (1968) that specialized forms of loose smut exist on either durum or common wheat. It may well be that in the evolution of loose smut different races developed on durum and common wheat in geographical isolation from each other. However, cultivation brought these populations together again, and since there is no barrier of incompatibility in crosses between races from common and durum wheat (Nielsen, personal communication), hybridization in nature will combine virulence genes from the two groups of races.

On the side of the host, common wheats are often found in pedigrees of durum cultivars, and vice-versa, most likely resulting in the occasional transfer of genes for susceptibility or resistance from one group of wheat to the other. For instance, the Canadian race T 5, virulent on Thatcher and derivatives (Nielsen 1969) was also virulent on Iumillo, a durum wheat in its pedigree, although avirulent on the durums Mindum and Pentad of the differential set. Conversely, if the formae speciales as proposed by Mitov (1968) existed, a general suscept like differential 13, P.I. 298 554, should not exist. This cultivar, originating from Ethiopia, is a common wheat that is susceptible to all races of loose smut to which it has so far been tested (Nielsen, personal communication). For these reasons, the use of formae speciales within U. tritici should be resisted.

The reaction of all 12 samples, as recorded in Table 2, with the supplemental differentials Klein 40, SPL 1 and SPL 2 was surprising. These three hosts are either susceptible or incompatible, but none is resistant to any of the samples. Differentiation of the 12 samples on these hosts thus appears to be based on the compatibility/incompatibility system proposed by Oort (1944). If his hypothesis is valid, all samples would carry the same gene(s) for virulence on these hosts, but some samples lack the gene(s) to overcome the incompatibility gene(s) in the host. For instance, sample 31 carries all the genes for virulence and all those necessary

to overcome incompatibility of these three hosts; sample 46 also carries the virulence genes, but has none of the genes to overcome incompatibility.

A test of the validity of Oort's hypothesis by crosses between specific cultivars goes beyond the scope of the present study. If it is undertaken in future, Klein 40 should be considered as one of the parents. The symptoms of incompatibility are so pronounced on this cultivar that it is very easy to classify the young plants into normal and incompatible ones. If such test-crosses do indeed prove Oort's hypothesis to be valid, interaction of races and differentials should be recorded in terms of resistant - susceptible - incompatible in the future.

With the exception of samples 17, 18 and 19 the 12 samples discussed have been tested twice, each time with a single sporulating head as source of inoculum. The reactions of the differentials were the same in both tests except for sample 5, where the original sample probably arose from a multiple infection. Each of the samples that passed through the second test therefore appears to be a homogeneous unit, which justifies the term "race" being applied to them in the future. However, lacking a standard system of naming races in loose smut of wheat they are left with their sample number for the time being.

The races described above from Brazil are not likely to be the only ones present in that country and a more exten-

sive search may well increase their number. However, it would appear from the 50 samples studied so far, and in conjunction with the results of the inoculation of a collection of resistant cultivars (see Table 3), that the variability of physiologic races of loose smut in Brazil is due more to new combinations of known genes for virulence, than to the presence of new ones.

4.2. Sources of resistance to loose smut from Brazil.

The reaction of the collection of resistant cultivars to the inocula A and B from Brazil is shown in Table 3. It is obvious, that there is quite a range of cultivars that can be used as sources of resistance to loose smut in Brazil. In fact, some of the resistant cultivars, like Maria Escobar, Sinvalocho, and derivatives of Hope/H 44 (like Selkirk) have in the past been found to be resistant to loose smut in Argentina (Cenoz, 1952; Frecha, 1967) and in Brazil (Silva, 1951; Mascarenhas and Silva, 1954), and were at that time recommended as sources of resistance. Other, more recent, resistant cultivars from South America like Pergamino Gaboto [= (Bage 2018/37 x (H 44 x Sinvalocho)) x Bage 1971/37], obviously derive their resistance from those earlier cultivars. Still others, the pedigree of which is not known, may also have these sources of resistance in their background. The fact that today all commercial Brazilian cultivars are susceptible suggests that although sources of resistance were available,

TABLE 3. Reaction of a collection of cultivars to mixtures A and B of loose smut from Brazil

Variety	C.I. or P.I. number	% of smutted heads when inoculated with mixture	
		A	B
Ayouby		0	0
Barbela Grosso		0	0
Bayody		0	0
Dakar 49	220 426	0	0
Es 518/13	304 388	0	-
Gubieha Auttma	223 155	0	-
Hoopvol	227 056	-	0
Horani Nawani		0	0
Indur compactum		0	0
I 12	83 402	0	0
Kenya x Lemhi[2]		-	0
Kenya 294 B.2.A.3.		0	0
Kenya 294 H.2.A.1.		0	0
Kenya 338 Z.2.G.2.		0	0
Kenya 340 Y.4.A.1.		0	0
Kenya 340 2.6.B.3		0	0
Kenya 351 AS.1.B.2		4	0
Kenya 4121		0	6
Klein 40	234 171	4	6
Line 1262-26		0	2
Line 1290-2258		0	-

TABLE 3. Reaction of a collection of cultivars to mixtures A and B of loose smut from Brazil (Cont.)

Variety	C.I. or P.I. number	% of smutted heads when inoculated with mixture	
		A	B
L. 8220-54	233 766	13	0
L. 8225-54	233 768	26	12
Maria Escobar	150 604	0	0
Maria Escobar x (Newthatch-Marroqui^2)-Kentana (Kenya Gular-Pilot) - (Kenya 58-Newthatch)		0	0
Maribal x Mariache		0	0
Marikenya Linea G	232 799	0	0
Marroqui 588		5	0
Massaux 5		0	8
Mayo (Pelon Colorado-Renown^2-Supremo) xKentana^2		0	0
(Mayo x Peru-Supremo) x Peru-Kenya		0	0
Mentana		0	0
Mercury		-	0
(Mida-Kenya 117 A) x Frontana		0	0
M 2824 x Thatcher-Thatcher^4		0	0
ND 4 x Lee		-	0
ND 52		0	0
ND 62		0	0
Olaeta Aquila		0	0
Olaeta Calandria		0	0

TABLE 3. Reaction of a collection of cultivars to mixtures A and B of loose smut from Brazil (Cont.)

Variety	C.I. or P.I. number	% of smutted heads when inoculated with mixture	
		A	B
Olaeta Gral Mitre		-	0
Orgaz		-	0
Parana 62/1845		10	4
Pergamino Gaboto		0	0
Peru x Supremo		0	0
Pusa 4		-	0
Redondo Negro		0	0
San Giorgio	232 815	-	0
Selkirk		0	0
Simbar Benvenuto		0	0
Sinvalocho M.A.		0	0
Sterling	227 057	-	0
Supremo^{2}x Kenya		-	0
St 464	191 365	0	0
Thatcher^2 x Frontana-Thatcher		-	0
Thatcher x Kenya 338 AA x Triumph^2 x Triticum x Agropyron, = II 60222		0	0
(Thatcher x Sta Cat)-Fr Sel 1 x C.I. 12 632		0	0
(Timstein-Kenya 58 x Gabo) x Lee		0	0
T. turgidum		-	0

TABLE 3. Reaction of a collection of cultivars to mixtures A and B of loose smut from Brazil (Cont.)

Variety	C.I. or P.I. number	% of smutted heads when inoculated with mixture	
		A	B
White Federation		0	0
Willet x Lerma		0	0
Wisc. 245 x Thatcher, = II 53-694		0	0
Wisc. 245 x II 50-17, = II 53-682		0	0
Wt x Norin 10 B26, = II 70-60		0	0
Xerez		-	0
Yaqui 48 x K 58-Newthatch		0	0
49-4789	189 783	0	0
49-4824		0	0
49-4845	189 791	0	0
3515 - 1t-1r-1t-2c		0	0
3669-17 -Saunders x CT 609		0	0
	207 098	0	0
	207 100	0	0
	208 894	0	0
	220 133	-	0
	227 945	0	0
	293 003	0	0
IAS 52 (Susceptible check)		20	34

continued incorporation of resistance to loose smut appears to have been neglected, because other characteristics (e.g. resistance to rusts and Septoria, or aluminium toxicity tolerance) required more urgent attention.

The cultivars showing resistance in this test should be maintained, then tested with the races found during this study, and periodically to field collections of smut from across Brazil. Even then one cannot be certain that the collection of resistant cultivars will have been exposed to all races of the pathogen present in Brazil. However, since these cultivars are of rather diverse origin it is hoped that they also carry different genes for resistance, so that if one is no longer effective, others will likely remain resistant.

This collection of resistant cultivars had been tested earlier to races from Canada and elsewhere and, while the present study was underway, it was tested to race T 10 from Canada, and to races from India, Kenya, Czechoslovakia and Argentina. Several cultivars were susceptible to one or the other of the latter inocula (Nielsen, unpublished results) and without exception, such cultivars, and only such cultivars, were also susceptible to either Brazilian mixtures A, or B, or both (see Table 3). This means that the virulence of the inocula from Brazil was not wider than the virulence of the other races to which the collection had already been tested. Any new genes for virulence, or new virulent combina-

tions of existing ones, would have shown up in infection after inoculation with the Brazilian population. This was not the case.

When looking at the percent infection on entries in the collection and on IAS 52, the susceptible check, it should be borne in mind that the inocula consisted of mixtures of field collections of spores. In this case, a low infection usually indicates that only one (or few) of the components is virulent. The potential of such a virulent component will only be realized by collecting spores on the host with low infection, and reinoculating it with these spores. The increase in the level of infection that can be expected by this procedure is illustrated by the following two examples. IAS 52 showed 20% infection when inoculated with mixture A (Table 3), and the single infected head of sample 1 was collected on this plot; reinoculation of SPL 1 and SPL 2 of IAS 52 with sample 1 gave infections of 80% on both lines. Likewise, Klein 40 had an infection of 6% after inoculation with mixture B (Table 3), and sample 17 was collected on this plot; reinoculation of Klein 40 with this sample gave 20% infection.

The number of sporulating heads after inoculation with a mixture of strains will also be reduced if the variety is susceptible to some of the components of the mixture, but incompatible with others. For instance, 20% of the plants

of IAS 52 were infected with mixture A (Table 3); yet there were another 30% that reacted with incompatibility.

4.3. Inheritance of resistance to loose smut

The reactions of the F1, and F3 were used to study the mode of inheritance of resistance to race T 2 in a cross between the incompatible Kenya 340 Y.4.A.1 and the susceptible IAS 52.

4.3.1. Reaction of F1. The second, inoculated cross Kenya 340 Y.4.A.1. x IAS 52 (SPL 2) yielded eight seeds. They were planted in a greenhouse, and all seedlings showed typical symtoms of incompatibility. The plants recovered, but showed no spore-formation. The reaction of F1-plants to race T 2 was thus identical with the reaction of the incompatible parent Kenya 340 Y.4.A.1., indicating that the gene(s) for incompatibility was dominant.

4.3.2. Reaction of F3. In the F3-progenies the percentage of plants infected with race T 2 varied from 0 to 100%. To study the observed data of the F3, the lines showing no infected plants were classed as "free", and the lines containing any infected plants were classed as "infected".

Of the 122 F3-lines, 23 were free, and 99 were infected. The observed ratio of 23 to 99 closely approaches the 1:3 ratio of 30.5 to 91.5 which would be expected if a single gene was responsible for the incompatibility of Kenya

340 Y.4.A.1. The chi-square (=2.242) test of goodness of fit indicated that the deviation from the theoretical was not significant.

According to Oort's (1944) hypothesis, a cultivar that reacts with incompatibility to a given race carries a gene for susceptibility and, superimposed and independent from it, another gene that confers incompatibility to that race. A second cultivar that is susceptible to the same race also carries the gene for susceptibility, but lacks the gene for incompatibility. It follows that in a cross between these two cultivars there should be segregation only of the genes controlling compatibility/incompatibility. This situation applies to the present example.

On the basis of a single gene, a ratio of 1 homozygous incompatible: 2 segregating: 1 homozygous susceptible was expected in the F3. Since incompatibility is not followed by spore-formation, the 23 F3-lines that were not infected have to be considered to represent the homozygous incompatible group. One would expect all these lines to show a high degree of incompatibility and therefore they should be easy to distinguish. In the 65 lines of the segregating group, whose upper limit of infection was arbitrarily set at 50%, a much smaller percentage of incompatible plants would be expected. Finally, in the homozygous susceptible group there should be hardly any incompatible plants at all. The observations did not agree with this expected distribu-

tion of incompatible lines (Table 4).

TABLE 4. Distribution of lines with different degrees of incompatibility within three groups of the F3-progeny of the cross Kenya 340 Y.4.A.1. x IAS 52.

% infection	Group	Number of lines in this group	Number of lines with % plants incompatible			
			0	1-10%	11-30%	30-50%
0	homozygous incompatible	23	3	6	12	2
1-50	segregating	65	4	18	29	14
51-100	homozygous susceptible	34	4	10	20	0

None of the lines had more than 50% incompatible plants, which does not approach the 88% displayed by the incompatible parent Kenya 340 Y.4.A.1. that was inoculated as a control. Nor did the homozygous incompatible group contain only lines with a high number of incompatible plants. Instead, lines with an up to moderate percentage of incompatible plants appeared to be evenly distributed amongst the three groups. From these data it would appear that more than one gene is involved in controlling incompatibility, and that they are independent of the gene for resistance. In this case it would be impossible to use the symptoms of incompatibility for selection of the resistant genotype in the seedling stage as proposed above.

Obviously, the inheritance of resistance based on the incompatible reaction of Kenya 340 Y.4.A.1. does not follow the model proposed by Oort (1944, 1963). However, more crosses between cultivars that react similarly to this parent and the susceptible parent IAS 52 are needed to clarify whether the present results are typical. The fact remains that cultivars which react with incompatibility to races of loose smut can be used as a source of resistance to these races.

CHAPTER 5

CONCLUSIONS

For the first time, the occurrence in Brazil of physiological races of loose smut of wheat, *Ustilago tritici*, has been demonstrated. Twelve races were identified among 50 field-collections of loose smut.

Five of the races would be classified as being similar to the Canadian race T 2, and two races as T 8 if only the standard set of differential cultivars was used. However, further differentiation of these races was possible on three supplemental differentials.

Another four races were virulent on differential cultivars of common wheat and on Pentad, a differential cultivar of durum wheat. These races appear to invalidate the claim by Mitov (1968) that *formae speciales* of *Ustilago tritici* exist on common wheat and on durum wheat.

The variability of races of loose smut in Brazil appears to be due to recombinations of genes for virulence that were already known, rather than to the presence of new ones.

A collection of 68 cultivars of diverse origin has been established that is resistant to races from Brazil and other countries. Among these cultivars, the old South American cultivars Sinvalocho M.A., Maria Escobar and their derivatives that were reported earlier to be resistant, are still resistant.

In the intervarietal cross Kenya 340 Y.4.A.1. x IAS 52 the incompatible reaction of Kenya 340 Y.4.A.1. was dominant. A single gene appears to be responsible for the resistance of this cultivar.

The data did not confirm the hypothesis by Oort (1944, 1963) that a cultivar that is incompatible with a given race carries a gene for susceptibility and, independent from it a gene for incompatibility which makes this cultivar resistant. Instead, it appeared that more than one gene is involved in causing the incompatible reaction of Kenya 340 Y.4.A.1. to race T 2 and that these genes are independent of the gene for resistance.

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