Bunt and Smut Diseases of Wheat

Concepts and Methods of Disease Management

CIMMYT

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Contents

Abstract

Wilcoxson, R.D., and E.E. Saari, eds. 1996. Bunt and Smut Diseases of Wheat: Concepts and Methods of Disease Management. Mexico, D.F.: CIMMYT.

Five bunt and smut diseases are regularly associated with wheat (*Triticum* spp.). These are: common bunt (*Tilletia* spp.), dwarf bunt (*T. controversa*), Karnal bunt (*T. indica*), loose smut (*Ustilago tritici*), and flag smut (*Urocystis agropyri*).

This fourth practical manual in CIMMYT's Wheat Disease Manual Series gathers together information necessary for understanding these diseases. It presents background information and concepts and methods that will help those not familiar with the bunts and smuts to correctly identify the different pathogens.

General principles and guidelines for disease management are presented to help breeders and pathologists in controlling the diseases and selecting for resistance. Comprehensive reviews of the literature focus attention on pertinent reports for each disease.

Other Manuals in This Series

Stubbs, R.W., J.M. Prescott, E.E. Saari, and H.J. Dubin. 1986. Cereal Disease Methodology Manual. Mexico, D.F.: CIMMYT.

Eyal, Z., A.L. Scharen, J.M. Prescott, and M. van Ginkel. 1987. The Septoria Diseases of Wheat: Concepts and Methods of Disease Management. Mexico, D.F.: CIMMYT.

Roelfs, A.P., R.P. Singh, and E.E. Saari. 1992. Rust Diseases of Wheat: Concepts and Methods of Disease Management. Mexico, D.F.: CIMMYT.

Preface

Fourth in a series of wheat disease technical manuals being developed at CIMMYT, this publication is a much needed and timely reference concerning the bunt and smut diseases of wheat.

The first disease manual, the “Cereal Disease Methodology Manual,” published and distributed in 1986, was more general in scope. Shortly after its publication, a group of CIMMYT scientists conceived the idea of producing a whole series of technical manuals on specific wheat diseases, tailored for a developing country audience. Instrumental in getting the series started were Drs. H.J. Dubin, A.R. Klatt, J.M. Prescott, E.E. Saari, R.W. Stubbs, and E. Torres. Awar d-winning manuals on the septoria diseases and the rusts were published in 1987 and 1992, respectively. Now, in 1996, this manual on bunts and smuts gathers under one cover the accumulated wisdom of a select group of disease specialists. A fifth manual devoted to the bacterial diseases of wheat is slated to come out in the latter part of this year.

As evident from historic references, the bunts and smuts have had a long relationship with wheat and its relatives. For example, common bunt and loose smut have afflicted wheat cultivation since the beginning of recorded history. Besides those two diseases, this manual covers dwarf bunt, Karnal bunt, and flag smut. Although there are other bunts and smuts that attack wheat, their distribution and economic impact are relatively insignificant.

In preparing this manual we have tapped the expertise of Wheat Program pathologists and of outside specialists. We take this opportunity to express our special appreciation to Blair Goates (common and dwarf bunt), Jens Nielsen and Percy Thomas (loose smut), Barbara Ballantyne (flag smut), Peter Burnett and Omar Mamluk (overview of the diseases), and Roy Wilcoxson (overall technical editing), without whose contributions this manual would not have been possible.

The usefulness of this publication is enhanced by the extensive literature review of the subject matter (nearly 700 citations) and summary of practical information that it provides. Other publications and reviews are available, but no single reference describes relevant concepts and how to apply them to the management of these diseases as does this manual. Finally, while it is true that the information included here is intended for workers who have to deal with bunts and smuts in developing countries, we have no doubt it will be equally useful and well received in developed countries.

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Wheat Bunts and Smuts

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Global Status and Importance

The Diseases

Five bunt and smut diseases are regularly associated with wheat (*Triticum* spp.) and related grasses: common bunt, dwarf bunt, Karnal bunt, loose smut, and flag smut (Fischer and Holton 1957, Holton 1967, Martens et al. 1984, Wiese 1987, Mathur and Cunfer 1993). There are a few other bunt diseases that are relatively insignificant in distribution and economic impact (Grasso 1968, Sharifnabi and Hedjaroude 1992). The pathogens of bunt and smut diseases are distinct except that *Tilletia tritici*, *T. laevis*, and *T. controversa* appear to be closely related. Details of the relationships are discussed in the chapter on common and dwarf bunts in this manual. Several common and scientific names have been applied to the diseases and their pathogens. Variation in the nomenclature occurs depending on the depth of knowledge and priorities of the authorities. **Table 1.1** lists currently accepted names and important synonyms. **Figures 1.1-1.5** depict the worldwide distribution of these diseases.

Common bunt and loose smut. These diseases have been associated with wheat cultivation since the beginning of recorded history (Fischer and Holton 1957, Stakman and Harrar 1957, Chapters 2 and 4 in this manual). In early historical writings, different diseases of wheat such as rusts, mildew, bunts, and smuts were not clearly differentiated until the microbial causes of plant diseases were discovered. Despite this, there seems to be no question that common bunt (CB) and loose smut (LS) have been present from the earliest agricultural experiences of man. The term “corn”, used in early writings, refers to wheat rather than to maize (*Zea mays* L.). However, today this fact is occasionally forgotten.

A long association of bunts and smuts with wheat and wheat relatives is indicated because the bunt and smut fungi are obligate parasites, highly specialized in pathogenicity. Undoubtedly, these fungi originated in the same center—the Near East—as the wheats and wheat relatives. This concept is supported by the large number of host resistance genes identified from the Near East (Fischer and Holton 1957, Leppik 1970, Hoffmann 1982, Zohary and Hopf 1988).

CB and LS fungi are well adapted to Near East environmental conditions and cause the most important diseases of wheat, after the rust diseases (Holton and Heald 1941, Fischer and Holton 1957, Stakman and Harrar 1957, Mamluk et al. 1990, Chapters 2 and 4 in this manual). Both diseases are widespread and are effectively perpetuated with seed, either on seed surfaces or as infections. Therefore, dispersal from the Near East center of origin was assured as seeds were carried to other parts of the world. Wind probably was not important in early dispersal of CB because of hand harvesting and threshing at a central site. However, wind may be important in modern agriculture because combine harvesting liberates teliospores, depositing them onto the surface of the field, releasing them into the atmosphere, where they may be carried long distances (Fischer and Holton 1957, Zadoks and Schein 1979, Bonde et al. 1987, Yarham 1993).

The CB pathogens survive between crops in soil for varying periods of time, especially if moisture is absent and monoculture is practiced (Fischer and Holton 1957, Line 1993b). This situation fits into the Mediterranean agricultural climate and pattern (Kassam 1988).

The LS pathogen, in contrast, survives in embryos and is favored by cool moist weather at flowering time. Consequently, LS is frequently more common at higher elevations where flowering periods are extended. This situation fits into areas of the Near East where elevations often exceed 1000 m, and cool nights and dews

Table 1.1. The common names and scientific nomenclature used in this manual, and some of the more frequently encountered synonyms in the literature dealing with the bunts and smuts of wheat.

I.	*Tilletia laevis* Kühn, Common bunt
	Common names: Common bunt, stinking bunt, covered smut, hill bunt, complete bunt, low bunt, high bunt
	Scientific synonyms: *T. levis* Kühn, *T. foetida* (Wall.) Liro, *T. foetens* (Berk & Curt.) Schröt.
II.	*Tilletia tritici* (Bjerk.) Wint., Common bunt
	Common names: Common bunt, stinking bunt, covered smut, hill bunt, complete bunt, low bunt, high bunt
	Scientific synonyms: *T. caries* (DC.) Tul., *T. tritici* (Bjerk.) R. Wolff
III.	*Tilletia controversa* Kühn, Dwarf bunt
	Common names: Dwarf bunt, short smut, stunt smut, stubble smut, TCK smut
	Scientific synonyms: *T. contraversa* Kühn, *T. brevifaciens* Fisch.
IV.	*Tilletia indica* Mitra, Karnal bunt
	Common names: Karnal bunt, partial bunt, new bunt, kernel bunt, KB
	Scientific synonyms: *Neovossia indica* (Mitra) Mundkur
V.	*Ustilago tritici* (Pres.) Rostr., Loose smut
	Common names: Loose smut
	Scientific synonyms: *U. nuda* (Jens.) Rostr., *U. nuda* var. *tritici* Schaff.
VI.	*Urocystis agropyri* (Preuss) Schröter, Flag smut
	Common names: Flag smut
	Scientific synonyms: *U. tritici* Koern.

occur with high frequency (Tahir and Hayes 1988). The cool moist weather at anthesis in northern latitudes also favors loose smut.

Dwarf bunt. This pathogen is closely related to CB pathogens and undoubtedly originated in the Near East. However, in the Near East, it is restricted to higher elevations with extended snowcover (Parlak 1981; Özkan and Damgaci 1986; Bamdadian 1993a,b). Teliospores of the dwarf bunt (DB) fungus are capable of long-term survival in soil (Holton and Heald 1941, Fischer and Holton 1957, Line 1993a). The DB pathogen has a wider virulence spectrum than the CB pathogens (Hoffmann 1982, Line 1993a), which may be important in survival on *Triticum* species since sown wheat areas in mountain regions are scattered compared to the plains where common bunt prevails.

Flag smut. Flag smut (FS) is of uncertain origin, but it probably originated in Asia (Chapter 5 in this manual). Suggestions that it originated in Australia or the United States seem questionable, since wheat has been grown in these countries for a relatively short time and the evolution of a pathogen in this short period seems unlikely. The disease may have had its origin somewhere in Asia because pathogenic variability of the FS fungus seems to be most diverse in collections from China and India (Chapter 5). The life cycle of the fungus would have allowed for ready dispersal on seed and straw, especially with primitive threshing procedures. Survival of teliospores in soil for extended periods is another important factor (Line 1993c). The dry summer between crop seasons does not seem to be critical for extended survival. Our knowledge concerning origins of the FS fungus is still limited.

Karnal bunt. Karnal bunt (KB) was first recorded in the northern plains of India and Pakistan, where it has been endemic and most likely evolved (Bhatti and Ilyas 1986, Singh 1986, Warham 1986, Joshi et al. 1983, Agarwal et al. 1993a). Spread of KB from the Indian Subcontinent was not recognized until the 1970s, when it was confirmed in Mexico. Since then, spread of KB within India and Mexico has been documented with the widespread distribution of seed of susceptible cultivars produced in endemic areas (Singh et al. 1983, Singh 1986, Carreon Zuniga 1992, Lira Ibarra 1992, Agarwal et al. 1993a). Yet KB has been established only in certain areas. Recently, KB was reported from western Nepal (Singh et al. 1989), an area that probably lies within the natural range of the pathogen.

Thousands of tons of wheat seed were produced in the KB endemic area of north India and shipped to Bangladesh in late 1970s and early 1980s (Ahmed et al. 1986). It is doubtful that all of this seed was free of KB. Yet there are no reports of KB in Bangladesh (Ahmed 1986). There are, however, reports of KB from countries other than the endemic areas (Lambat et al. 1983, Agarwal et al. 1993a). However, KB has not been confirmed by on-site inspections in the concerned countries. Contaminated seed lots from endemic areas were known to be trans-shipped through some of these countries.

The Pathogens

Common bunt. Two pathogen species are involved, and teliospores typical of both species are sometimes found in the same sorus. In earlier literature (reviewed by Holton and Heald 1941, Fischer and Holton 1957, Holton 1967), durum wheats were suggested to be more resistant to *T. tritici* and bread wheats more resistant to *T. laevis*. Sometimes the reverse was reported. Fischer and Holton (1957) suggested that these differences could be explained by physiological specialization, selection pressure, and pathogen hybrids showing the full range of teliospore types (Chapter 2 in this manual). More recently, Mamluk and Zahour (1993) indicated a strong selective advantage of bread wheat for *T. laevis*, whereas durum wheat was equally infected by both CB species.

Topography may influence the presence of CB pathogen species. *Tilletia laevis* dominates collections from higher elevations, and *T. tritici* prevails at lower elevations (Holton and Heald 1941; Yirgou 1967, Niemann et al. 1980, Yüksel et al. 1980). Today, there is a resurgence of CB due to ineffective resistance, new cultivars that lack resistance, and inadequate seed treatments (Mamluk et al. 1990, Mamluk and Zahour 1993, Line 1993b).

Dwarf bunt. This pathogen occurs at higher elevations or locations where snowcover is persistent (Line 1993a). Snow insulates from freezing, which allows the pathogen and host-pathogen interaction to develop slowly. In endemic areas, serious losses have been recorded. This fungus is closely related to CB fungi (Russell and Mills 1993, Chapter 2 in this manual).

Loose smut. The LS fungus occurs wherever wheat is grown but incidence is lower in warmer, dry climates where the wheat plant cycle is short. LS can be controlled by resistant cultivars or effective seed treatment fungicides (Agarwal et al. 1993b). There is a resurgence of LS because cultivars are not resistant and seed treatments are either not used or carelessly used.

Flag smut. The distribution of FS is regionalized. It causes severe damage where it is endemic on susceptible cultivars. Interestingly, FS has not been reported from ancient wheat growing areas such as Saudi Arabia, Yemen and Ethiopia (Kamal and Al Agbari 1985, Hulluka et al. 1991, El-Meleigi et al. 1993). Resistant cultivars and effective seed treatment fungicides have controlled FS in a number of countries.

Karnal bunt. The pathogen is endemic in the Indian Subcontinent and Mexico (Anon 1989a, Singh 1986, Warham 1986).

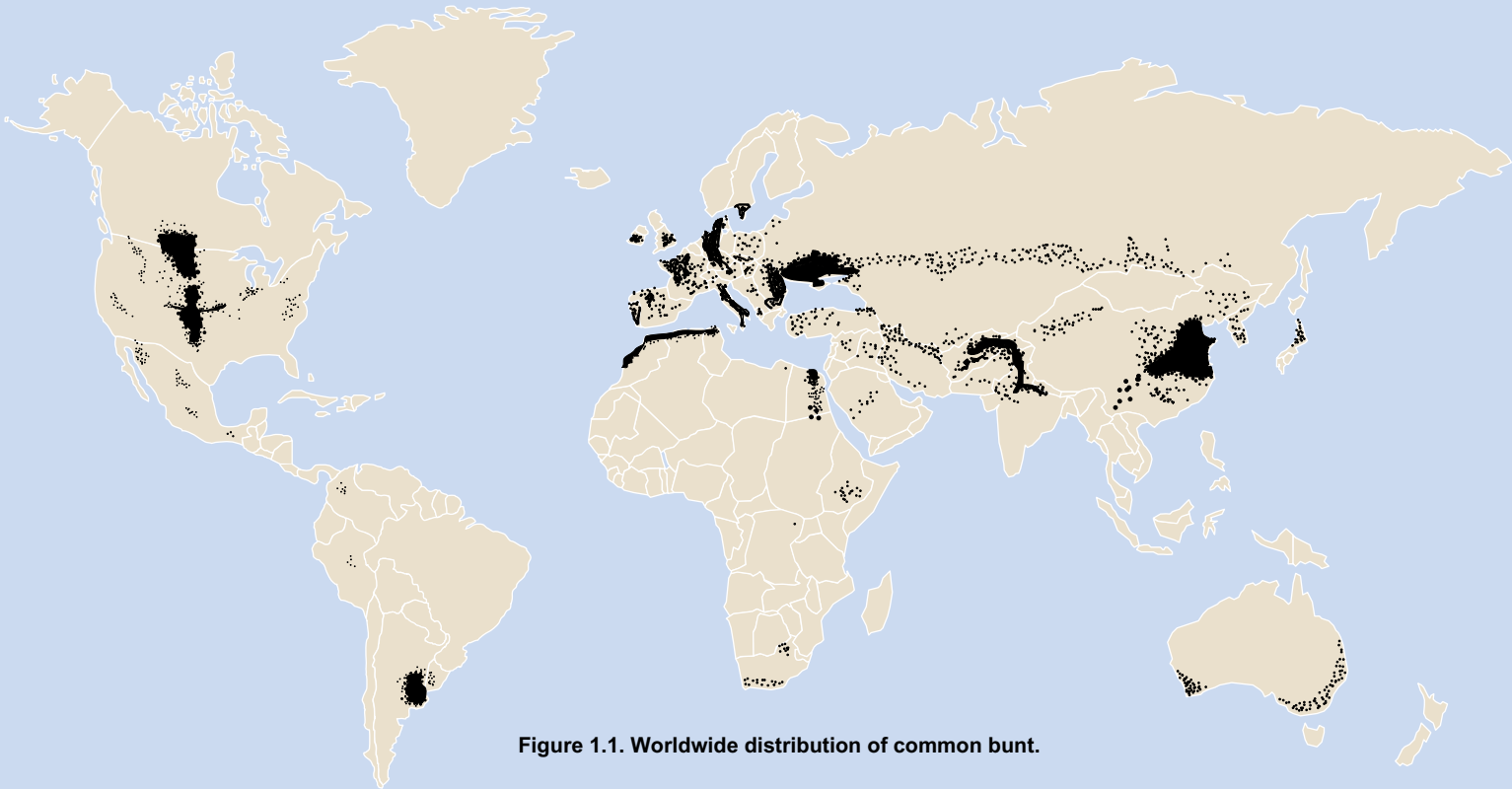

Figure 1.1. Worldwide distribution of common bunt.

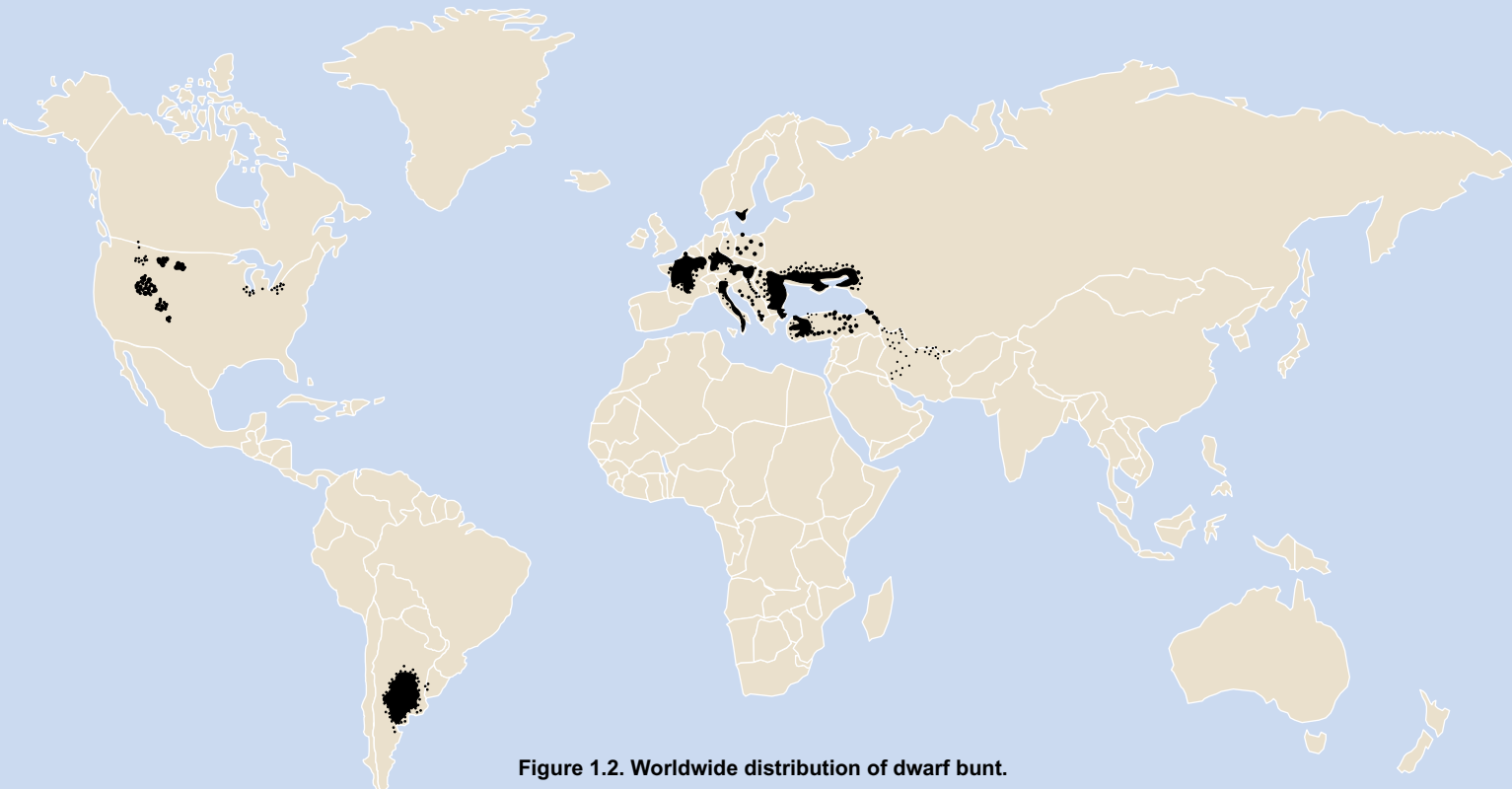

Figure 1.2. Worldwide distribution of dwarf bunt.

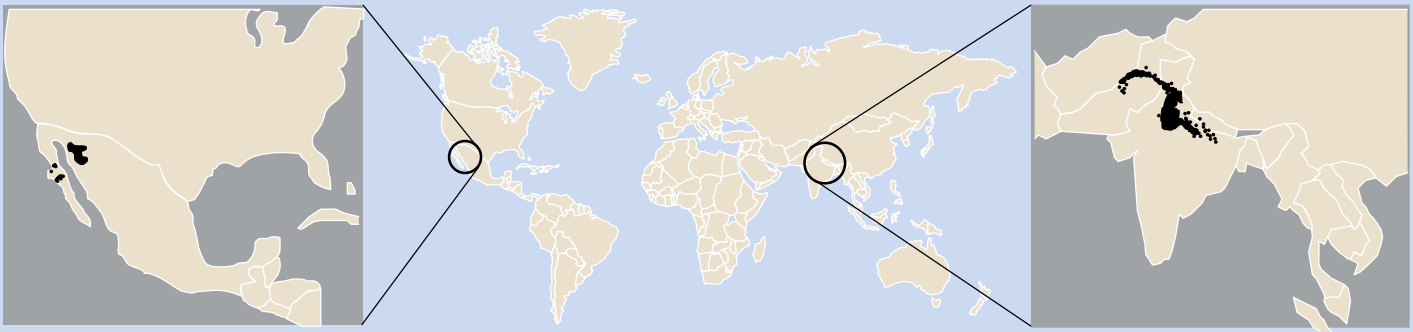

Figure 1.3. Worldwide distribution of Karnal bunt.

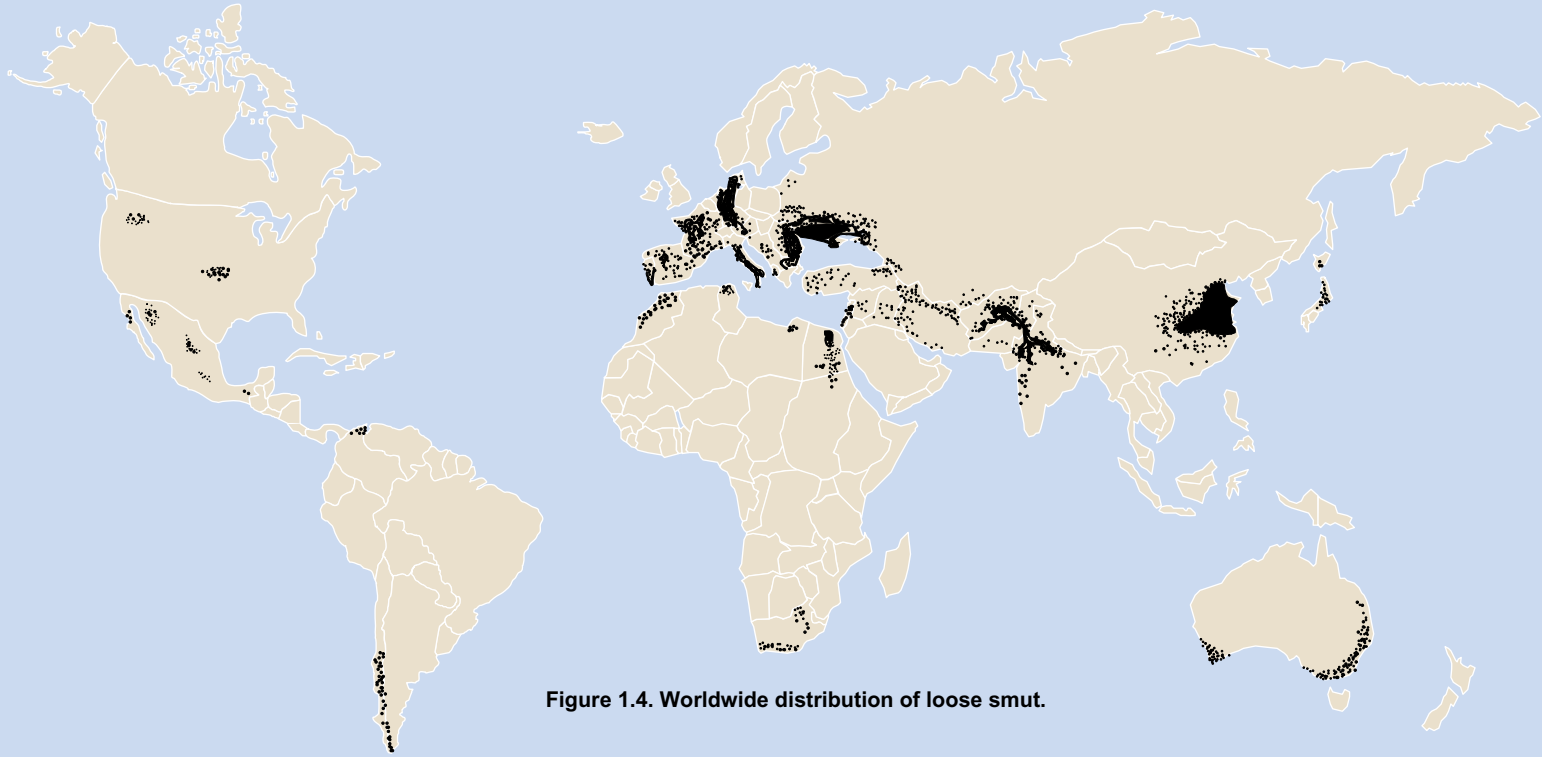

Figure 1.4. Worldwide distribution of loose smut.

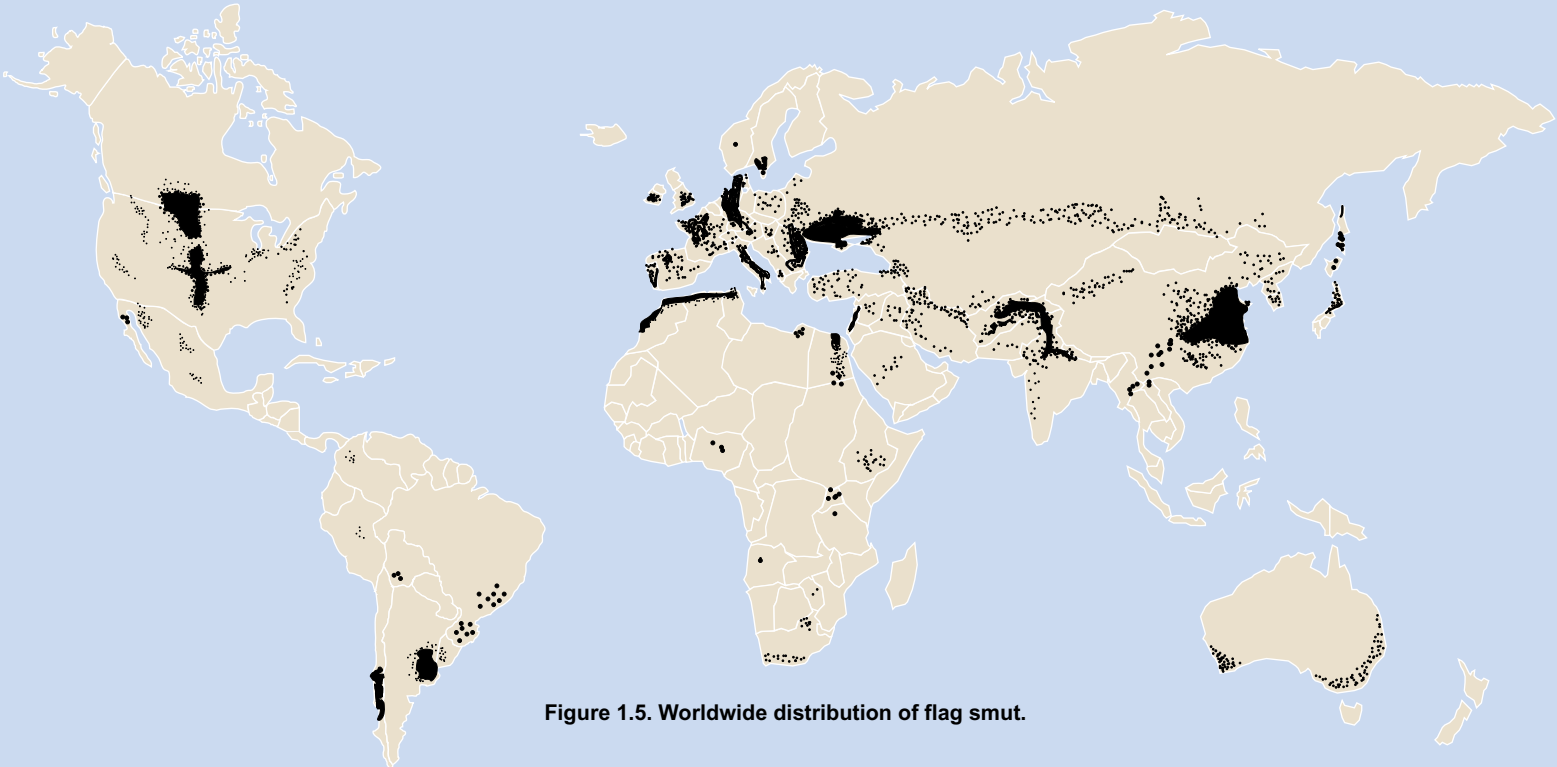

Figure 1.5. Worldwide distribution of flag smut.

Reports from other countries have not been substantiated. However, it is possible that the KB fungus has a wider distribution than is currently recognized. Development of resistant cultivars has been a long difficult process. Chemical control procedures are available but are not completely effective (Chapter 3 in this manual).

Life cycles. The life cycles of the bunt and smut fungi are complex and detailed. Specific information is recorded in this manual, and life cycles are diagrammed in **Figures 1.6-1.9**.

Regional Distribution

West Asia and the Near East

The Near East is probably the center of origin for wheat and related species, as well as for CB, DB, and LS pathogens (Leppik 1970, Zohary and Hopf 1988). The KB fungus is clearly not endemic to this region (Warham 1986, Singh 1986, Agarwal et al. 1993a) and the FS fungus probably originated in Asia (Line 1993c, Chapter 5 in this manual).

In this region, CB and LS may be the most widespread and important wheat diseases, after the rusts (Fischer and Holton 1957, Iren 1981, Mamluk et al. 1990, Mamluk and Zahour 1993). They are, certainly, the most frequently reported bunt and smut diseases. Actual losses are not well documented, but yield losses of 1-7% are common (Mamluk 1992). In general, percentage yield loss due to LS and FS are roughly equal to the percentage infection of spikes or plants, whereas losses due to CB are somewhat lower because not all kernels in a spike are affected (Mamluk and Zahour 1993). Losses caused by CB were estimated at 5-7% in 1979 (Hoffmann 1982). Even when yield losses are minor, contamination of grain with CB is often sufficient to reduce quality and cause marketing problems (Williams 1983).

In Turkey, bunt and smut diseases of wheat rival rusts in importance (Finci 1981, Iren 1981, Parlak 1981, Finci et al. 1983, Bicici et al. 1991). Early surveys established that about 10% of the wheat fields carried some level of CB, and some individual fields had 60-90% infected plants (Parlak 1981). *T. laevis* was the most common pathogen in Turkey, accounting for almost 88% of the identifications, whereas *T. tritici* was dominant at lower elevations in southeastern Turkey (Yüksel et al. 1980, Iren 1981, Finci et al. 1983). A greater number of races of *T. laevis* than of *T. tritici* were identified from bunt collections (Finci 1981, Finci et al. 1983). Production losses have been estimated at 10-20%, depending on the year and the authority (Yüksel et al. 1980, Iren 1981, Parlak 1981). Further, only 40-50% of the seed is treated, so bunt incidence quickly increases in susceptible cultivars. A number of newer cultivars are susceptible to CB (Atac 1988), and more recently 15%

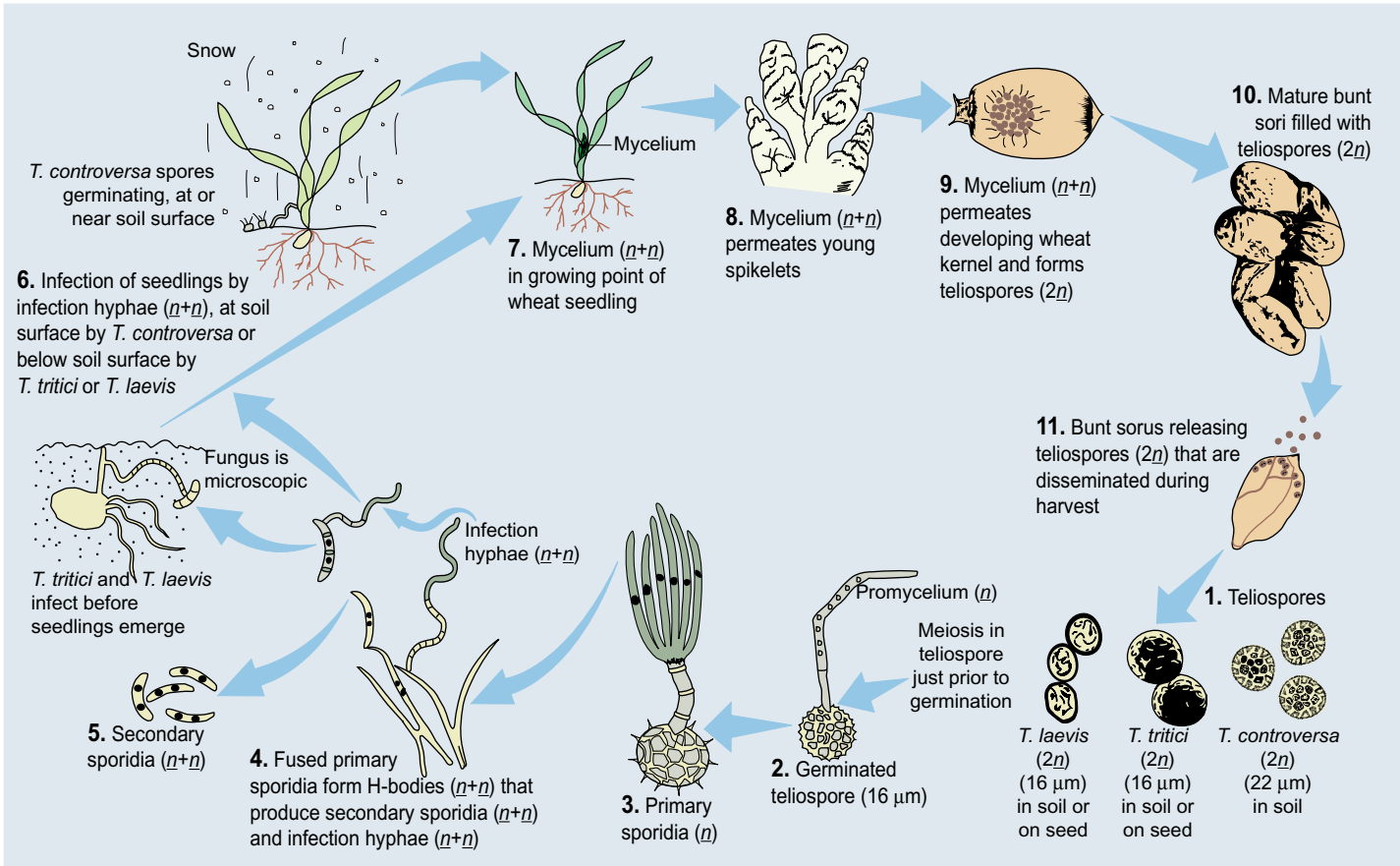

Figure 1.6. Disease cycle of common bunt caused by *Tilletia tritici* and *Tilletia laevis* and dwarf bunt caused by *Tilletia controversa*.

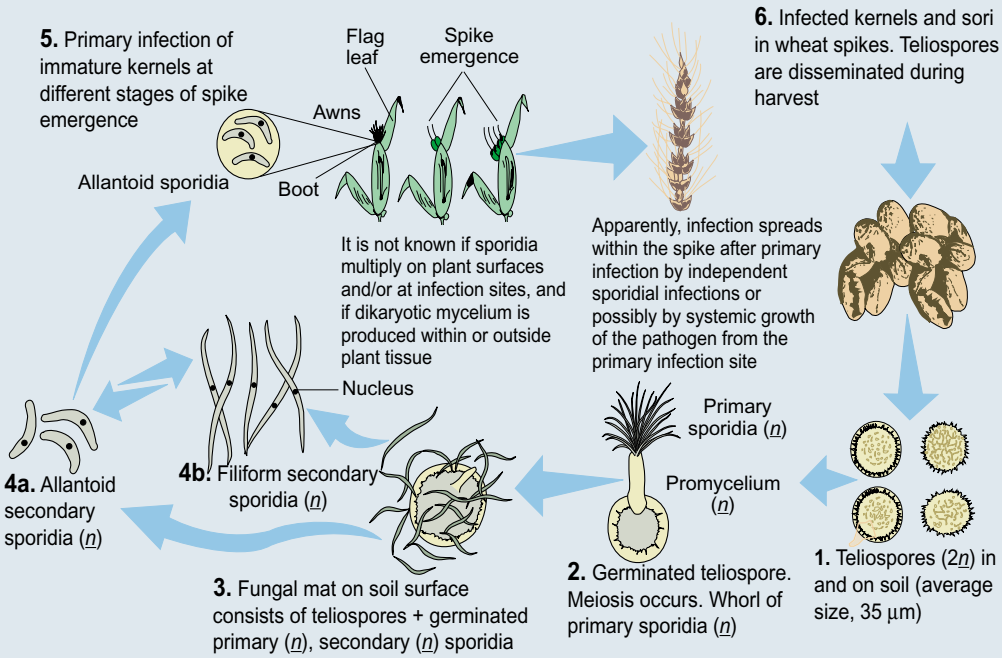

Figure 1.7. Disease cycle of Karnal bunt caused by *Tilletia indica*.

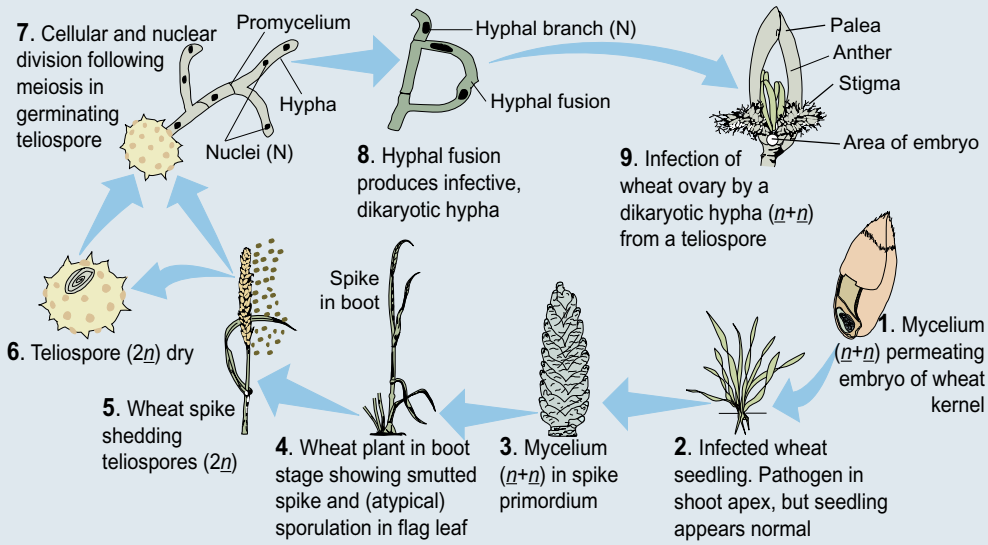

Figure 1.8. Disease cycle of loose smut caused by *Ustilago tritici*.

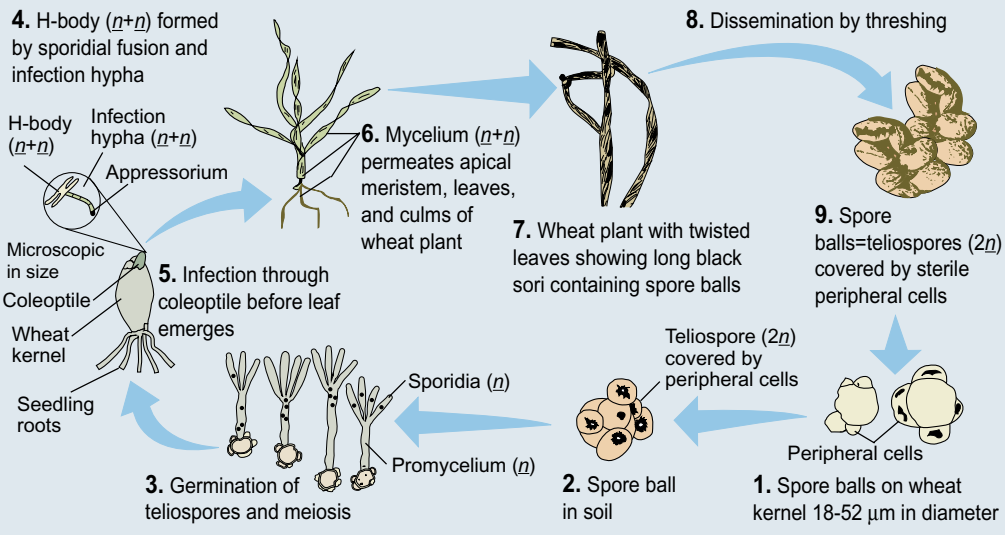

Figure 1.9. Disease cycle of flag smut caused by *Urocystis agropyri*.

of fields of southeastern Anatolia showed CB incidence of up to 28% (Bicici et al. 1991).

In Syria, CB was "...the most important disease of wheat..." some 30 years ago (Mulder 1958) and continues to be hazardous (Mamluk et al. 1989, 1990; Mamluk 1992). The disease was recorded in 50% of wheat fields surveyed in three of four production zones (Mamluk et al. 1990). In this survey, neither *T. laevis* nor *T. tritici* predominated, but their presence was correlated with the host species. Only 3% of samples tested contained a mixture of both pathogens. The highest severity was a 60% incidence in the bread wheat cultivar Mexipak and a 33% incidence in the durum wheat landrace cultivar Shyhani.

In northern Syria, 83% of grain samples delivered to commercial flour mills were heavily contaminated with CB teliospores (Williams 1983), indicating heavy infection in fields where grain originated or in adjacent fields. Some of the widely grown cultivars (Mexipak, Cham 1 and Cham 3) are susceptible in Syria (O.F. Mamluk, unpubl.).

In Iran, bunt and smut are the most important diseases of wheat after the rusts (Zad 1972, Khazra and Bamdadian 1974, Sharif and Bamdadian 1974, Akbari and Zolghadri 1988, Mardoukhi 1989). CB causes losses of 25-30% in parts of Iran (Bamdadian 1993a,b). This disease is found throughout the country, but it is concentrated in the northern and northwestern regions (Akbari and Zolghadri 1988; Bamdadian 1993a,b). Five species of *Tilletia* are described in Iran. In one study, *T. laevis* was most common, being in 94 % of 729 samples analyzed (Sharifnabi and Hedjaroude 1992), and *T. controversa* was second most common, being present in 4.5% of the samples. *T. tritici* was present in only 0.1% of the samples. *T. triticoides* and *T. intermedia*, two new species for Iran,

were relatively rare, are restricted in distribution and may be hybrids of more common species (Fischer and Holton 1957, Chapter 2 in this manual). The reported dominance of *T. laevis* may also reflect the predominance of bread wheats in the area and at higher elevations of northern crop areas.

In Afghanistan, CB is second in importance to the rusts (Shah Samin 1969). *T. laevis* is the dominant species (Gattani 1964, Osmanzai and Quayoun 1972). Losses have been estimated at 5% for the nation (Peterson et al. 1967) and up to 20% in some provinces (Gattani 1964). Cultivar Mexipak was highly resistant to CB collected from local cultivars from highland areas (Osmanzai and Quayoum 1972).

In northern Iraq, there is a large rainfed area, and CB is the major disease of wheat (Hermis and Hussain 1979). In one survey involving different wheat growing areas, *T. tritici*, *T. laevis*, and *Tilletia* spp. were recovered. Cultivar Mexipak was resistant to all isolates from this survey (Ibrahim et al. 1985). In another study, a large number of hybrids between *T. tritici* and *T. laevis* were recorded among 4129 diseased spikes (Ibrahim 1988). In contrast, Tamimi (1986) analyzed 665 spike collections from different wheat locations in Iraq and found *T. tritici* to be the most common species from most locations.

In Lebanon and Jordan, the climate is arid with low rainfall, which minimizes some wheat diseases, but CB is widespread (Mazahreh 1981, Mamluk et al. 1984). Many farmers treat seed, so bunt incidence is relatively low (Ghosheh 1975). In Lebanon, only *T. laevis* has been identified (Weltzien 1963, Saad and Nienhaus 1969, Khatib et al. 1970). A high incidence of CB occurred in the northern Bekka Valley of Lebanon as a result of sowing susceptible CB-free cultivars without seed treatment. This experience suggests high levels of soilborne inoculum (E.E. Saari 1974, unpubl.).

In Egypt, CB occurs throughout the country, including the Sinai, on both bread and durum wheats, but is more common on bread wheats. Losses are small because of seed treatment but sometimes may be considerable (Abdel-Hak and Ghobrial 1969a, Anon. 1969).

In Saudi Arabia and Yemen, early surveys did not report CB (Martin 1970, Hindorf et al. 1978). However, a recent survey in central Saudi Arabia registered *Tilletia* spp. but did not quantify its presence (El-Meleigi et al. 1993). Another report indicated CB in local wheat near Abha (Asir Region), western Saudi Arabia, at 30-50% incidence (Hall 1975). *T. laevis* but not *T. tritici* was reported in Sanaa Governorate, Yemen, but it is a minor disease (Kamal and Al Agbari 1985).

DB has been reported from Turkey (Yüksel et al. 1980, Parlak 1981, Hoffmann 1982, Özkan and Damgaci 1986), Iran (Mardoukhi 1989, Bamdadian 1993a,b), and Iraq (Hoffmann 1982, Trione 1982). One of the most severely affected areas is eastern Anatolia, Turkey and northwest Iran at elevations of 1300-2000 m (Özkan and Damgaci 1986; Sharifnabi and Hedjaroude 1992; Bamdadian 1993a,b). The area infested with DB in Turkey is limited to about 2500 ha (Parlak 1981). Losses, however, are as high as 80% (Yüksel et al. 1980, Parlak 1981, Özkan and Damgaci 1986).

In Turkey, DB infects *Hordeum bulbosum*, *H. murinum*, *H. marinum*, *Agropyrum repens*, and *Aegilops umbellalata* (Özkan and Damgaci 1986). This report has been confirmed, but cross inoculations to wheat have not finalized the role of these hosts in the epidemiology of the smut. The DB fungus is known to have a wide host range (Chapter 2 in this manual).

LS occurs in almost all locations of the West Asia and Near East Region, even in oasis areas (Anon. 1969, Waller and Bridge 1978, El-Meleigi et al. 1993). A high incidence has been reported from many countries, especially on old landrace cultivars, but also on more recently released high yielding cultivars. LS is often overlooked and its importance underestimated due to its early occurrence in the life cycle of the wheat plant.

LS occurs naturally on *Aegilops* spp. and on wheat almost everywhere it is cultivated in Syria (Mamluk et al. 1990). It occurs on rye in Turkey (E.E. Saari and O.F. Mamluk, unpubl.) and Iran (Bamdadian 1993a,b). The disease occurs sporadically in Syria, with incidence at about 5% (Azmeh and Kousaji 1982; Mamluk et al. 1990, 1992). It has been reported from Saudi Arabia and Yemen (Kamal and Al Agbari 1985, Abdul Sattar and Haithami 1986, El-Meleigi et al. 1993).

In Turkey, LS incidence ranges from 0.1 to 20%, depending on the cultivar and year (Iren 1981). Surveys in 1972-74 reported LS in 32% of the fields. Maximum LS incidence was 40% in eastern Turkey, 27% in Central Anatolia, and 15% in the Marmara Region (Parlak 1981). Overall incidence was calculated at 16% in infected fields. In a more recent survey covering the Central Anatolian Plateau, almost every field of cultivar Gerek 79 had some LS, with maximum incidence at 33% (E.E. Saari and O.F. Mamluk 1992, unpubl.). Gerek 79 was the most widely grown cultivar in the Central Plateau, with an estimated acreage exceeding 1 million hectares.

In Egypt, LS has been known for many years, though its importance has been minor because resistant cultivars were grown and certified seed was extensively used (Anon. 1969). In the early 1980s, susceptible cultivars such as Sakha 6l and Sakha 69 were associated with a marked

increase of loose smut in farm yields (Shafik et al. 1990, Sherif et al. 1991). While the disease seemed to "disappear" in the 1960s, more recent surveys recorded it in 90% of Sahka 61 fields at 0.01-0.1% incidence (Bassouni et al. 1988).

In Iran, LS is widely dispersed, but is more prevalent in northern areas around the Caspian Sea (Shariff and Bamdadian 1974; Bamdadian 1993a,b). Incidence of disease ranges up to 2% in susceptible cultivars.

In Jordan and Lebanon, LS is recorded but is a minor disease (Weltzien 1963, Anon. 1969, Saad and Nienhaus 1969, Khatib et al. 1970, Mamluk et al. 1984).

In Afghanistan, LS is widespread but of little importance. Losses are generally below 1%, except in Jalalabad and Laghman Provinces, where they may be greater (Peterson et al. 1967, Shah Samim 1969).

FS has been reported from Turkey (Parlak 1981), Iran (Bamdadian 1993a,b), Jordan (Mamluk et al. 1984) and Afghanistan (Shah Samin 1969) but is relatively minor in each of these countries. In Syria, FS occurs sporadically but may be increasing in importance (Mamluk et al. 1990). FS also occurs on *Aegilops* spp. in Syria (Mamluk et al. 1990) and on barley in Turkey.

In Egypt, FS has been known since 1923 and may have been introduced with large seed shipments from Australia after World War I (El-Helaly 1948). The disease was serious in the Southern Delta and Middle Egypt and less serious in Upper Egypt. Bread wheats were more susceptible than durum wheats, which were immune (Abdel-Hak and Ghobrial 1969b). The highest incidence was 58%, but over-all, incidence was about 1% (El-Helaly 1948, Abdel-Hak and Ghobrial 1969b). The disease is now of minor importance through the use of certified seed and resistant cultivars (Anon. 1969).

FS has not been reported from Saudi Arabia or Yemen.

KB has not been confirmed from the Middle East, Syria, Lebanon, or Turkey (Diekmann 1987, Mamluk et al. 1990; E.E. Saari and O.F. Mamluk, unpubl.; B. Skovmand and H. Braun, 1986, unpubl.; Kianci, H. Braun, and E.E. Saari, 1990, unpubl.). It has been reported in seed from the above three countries (Lambat et al. 1983, Zillinsky 1983), but these findings have not been confirmed by reports from the field.

Conclusions. CB, DB, and LS are important diseases in the West Asia and Near East Region. Although bunts have been managed in the past, they are now regaining importance probably due to: 1) a relaxed attitude toward seed treatment, 2) the use of ineffective fungicides, 3) the possible insensitivity of bunt pathogens to seed-treatment chemicals, 4) the decreased use of resistant cultivars, and 5) the increased use of mechanized threshing equipment. Continuous wheat cropping, a lack of effective seed treatments, and decreased use of resistant cultivars may contribute to an increase in the importance of soilborne inoculum. The role of resistant cultivars in the evolution of bunt diseases and of new races of the pathogens requires careful evaluation. Mexipak illustrates the influence of a resistant cultivar, its positive effects, and its shortcomings when overcome by the pathogens.

South Asia

All bunt and smut diseases, except DB, are present in the Subcontinent (Goel et. al. 1977, Hafiz 1986, Ram and Arora 1986, Singh 1986). The center of origin for KB is presumed to be northem India and Pakistan. The bunts and smuts are collectively second in importance to rusts, and losses vary depending on the locality and year (Goel et al. 1977; Hafiz 1986; Bhatti and Ilyas 1986; Joshi et al. 1978, 1986). In Nepal, CB and LS are important, especially at higher elevations (E.E. Saari unpubl.). In Bangladesh, only LS has been reported (Ahmed 1986).

In Pakistan, CB is important in upland areas of Baluchistan, the foothills of the Punjab, Northwest Frontier Province (NWFP), Northern Territories, and Muree Hills (Ghafoor and Khan 1976, Mirza and Khan 1983, Hafiz 1986, Jamil Khan et al. 1992). Both CB pathogens are present, with *T. laevis* being the most common (Hassan 1974). The disease is more important on local cultivars (Hashmi and Ghulam Ahmed 1988), but this may reflect the difference between introduced seeds and farmer-maintained seed. Infection incidence of 20-25% has been reported from hill areas, with 60-70% incidence in some individual fields (Hafiz 1986).

In India, CB is referred to as hill bunt because it is largely confined to northern hill regions. It has been sporadically reported in the plains, probably as a result of seed coming from the hills. The disease does not survive in the plains (Goel et al. 1977, Joshi et al. 1986, Singh 1986). Information on incidence and losses due to CB in the hills is limited. Some older reports suggest losses of 30-40% in some areas and in another report infection ranged from 10-96% (Singh 1986). In 1986, losses were 2-3% as a result of growing resistant cultivars (Singh 1986).

In India, LS is a damaging disease. Losses average 2-4% and in some areas of the plains they are as high as 5% (Joshi et al. 1986, Ram and Arora 1986). However, incidence can be 33% in the hills (Joshi et al. 1986). The disease declined in the late 1960s and early 1970s with the extensive cultivation of resistant cultivar Kalyansona in northern India. With the

release of susceptible cultivars, LS incidence has again increased (Sood 1988).

In Pakistan, LS is widely distributed throughout the country, with incidence ranging from a trace to 20% depending on the year (Hassan 1968, 1972; Khanzada and Aslam 1982; Hafiz 1986; Bhutta and Ahmed 1991). LS declined with extensive cultivation of resistant cultivar Mexipak, but is now increasing in importance in Pakistan as new susceptible wheat cultivars are widely adopted (Hassan 1974; Khanzada and Aslam 1982; Bhatti and Ilyas 1986; O.F. Mamluk, 1984, unpubl.).

In Pakistan, FS occurs in the foothill areas of northern Punjab and NWFP (Purdy 1965, Hassan 1972, Ghafoor and Khan 1976, Khan et al. 1984). The disease also occurs in adjacent Jalalabad, Afghanistan (Hassan 1970). Incidence is dependent upon climate and the cultivars that are grown (Hassan et al. 1979, Hafiz 1986). From 2 to 25% of fields carry diseased plants, with some fields carrying 60-70% infected plants (Hassan 1972, Hafiz 1986). A high incidence was associated with susceptible cultivars such as Barani 70 and Pothowar during the 1972-76 seasons (Hafiz 1986).

In India, FS is common in the northern foothills and at isolated locations in Rajasthan. It also occurs in Bihar, Delhi, Haryana, Himachal Pradesh, Madhya Pradesh, and Uttar Pradesh (Goel et al. 1977, Ram and Arora 1986). Disease incidence can be as high as 80% in local cultivars, although severe infection also occurs on newly released susceptible cultivars. In general, FS incidence is low in India.

In Pakistan, KB is most common in foothill districts of the north, especially in Sialkot and Gujranwala Districts (Hassan 1968, 1970, 1972, 1974). It is now found in central Punjab and NWFP (Hassan et al. 1979, Bhatti and Ilyas 1986, Ahmad and Attaudin 1991) where incidence averages 2-5% and occasionally 20% (Hassan 1974, Hafiz 1986). A survey for KB in certified seed lots from different areas of Pakistan indicated that infected kernels increased from 10% in 1982 to 19% in 1983 to 28% in 1984 (Begum and Mathur 1989). Washing tests found KB teliospores in 30% of seedlots that were visually free of KB. Seedlots from southern and western Pakistan were free of KB infection and teliospores.

In India, KB has increased in importance since 1975 (Singh 1986). Prevalence remains high in northwestern regions, notably in Punjab and western Uttar Pradesh. In 1981, KB was recorded in 93% of wheat samples collected from Punjab (Joshi et al. 1986, Singh 1986). The percentage of kernels infected, however, has remained low for most cultivars, generally about 1%. However, it was significantly higher in susceptible cultivars such as WL 711, which averaged 9% infection and a maximum of 24% in the epidemic of 1982 (Joshi et al. 1983, Singh 1986). KB incidence has gradually declined in recent years, which may reflect, in part, release of cultivars that are less susceptible and weather less favorable for KB development. KB is sensitive to unfavorable environmental conditions, which strongly influence disease development (Singh 1986, Nagarajan 1989, 1991).

Conclusion. Bunts and smuts of wheat are chronic in South Asia and in some areas cause severe losses. Seed treatment has not been consistently practiced at the farm level. Cultivation of susceptible cultivars has increased the importance of the bunt and smut diseases in cooler areas. In warmer areas of Bangladesh, India, and Nepal, the bunts and smuts, with exception of LS, have virtually disappeared. LS incidence is low in southern areas but continues to be economically important in the northern and hill areas of the Subcontinent.

North Africa

In Tunisia, FS ranks as a major disease of wheat. Seventeen percent of fields surveyed carried infected plants with the highest incidence at 40% infected plants (Kamel et al. 1987). In Morocco, 19% of durum wheat fields were infected with FS, but incidence was low (Lyamani 1990). The disease has also been reported from Libya (El-Zayat et al. 1977).

In Tunisia, Algeria, and Morocco, LS is common and widespread, but seldom severe (Anon. 1976, Rolli 1977, Kamel et al. 1987, Ezzahiri and Elyamani 1992, Lyamani 1990; O.F. Mamluk unpubl.). However, in Libya, in the Kufra Irrigation Project, up to 4% infection has been recorded on susceptible cultivars (El-Zayat et al. 1977).

In Algeria and Morocco, CB is present (Anon. 1976, Rolli 1977, Lyamani 1990, Ezzahiri and Elyamani 1992; O.F. Mamluk, unpubl.). In Morocco, uncertified seed often carries teliospores of the pathogens (Anon. 1976). In Libya, CB incidence is often 1-4% infected plants in many fields (El-Zayat et al. 1977).

In North Africa, DB has not been confirmed at present. It was reported from Libya (Anon. 1968), but accuracy of this report is questioned. Snow cover favors the biology of the pathogen and disease development, which raises doubts concerning the fungus presence in Libya.

Conclusion. The bunt and smut diseases of wheat can affect production and cause measurable losses in North Africa. Good control is feasible with seed treatments but many farmers are not willing to spend the money or effort required. The release of susceptible cultivars has increased the incidence of bunt and smut diseases in general.

East, West, and Southern Africa

Wheat is a new crop for most countries of Africa south of the Sahara Desert, except in Ethiopia and Sudan. Ethiopia is a center of diversity for durum wheat and barley (Zohary and Hopf 1988), and Sudan has cultivated wheat for centuries (Anon. 1969). The wheat crop in most of eastern, central and southern Africa was introduced in the past 100 to 200 years with European exploration and settlement.

In Ethiopia, most diseases of wheat are found, including the bunts and smuts, except for KB and DB (Yirgou 1967, Hulluka et al. 1991). CB is serious at higher remote elevations on landrace wheats (Yirgou 1967, Niemann et al. 1980). Annual losses caused by CB are about 5% of production and in heavily infected areas they may be 10-20% (Niemann et al. 1980).

In South Africa, CB has been recorded (Scott 1990), but losses are nominal due to seed treatment. LS and FS are present, but are not serious, and FS may have been eradicated (Scott and Le Roux 1992).

In other countries of Africa that grow wheat, LS occurs but is not serious. The other smuts and bunts have not been registered in these countries. However, in the high eastern regions of Kivu, Zaire, CB incidence of 50% has been recorded (Mwitirwa 1988).

Conclusion. The bunt and smut diseases are not important factors in wheat production in many countries of the region, except Ethiopia, higher elevations of Zaire, and, possibly, South Africa.

Far East Asia

Knowledge of bunt and smut diseases of wheat in Far East Asia is limited. Relatively little wheat is grown in the countries concerned except China. China, however, is a major wheat producer with 10 production zones (He and Chen 1991). CB, LS, and FS occur on wheat in China, but DB and KB have not been recorded. The bunts and smuts were major diseases up to the 1950s (Fischer and Holton 1957, Johnson and Beemer 1977). However, today, these diseases are of minor importance because of extensive use of chemical seed treatments, resistant cultivars, and strong enforcement of seed health and quarantine principles.

In China, LS occurs to some extent in most wheat production zones, but is more important in the northern zone (Zone VII). CB is a persistent problem in the far western zones of the Qinghai-Tibetan Plateau (Zone I) and Xinjiang (Zone X). FS was a serious problem at one time, but presently is found only in traces (Fischer and Holton 1957, Purdy 1965).

Conclusion. Wheat is not extensively cultivated in Far East Asia except in China. In China, the bunts and smuts were important diseases in the past, but today strong seed health measures have reduced their importance. However, the potential of the bunts and smuts remains high, and in remote areas they undoubtedly continue to cause some losses.

Europe and Central West Asia

The bunts and smuts have been major diseases throughout Europe (Fischer and Holton 1957, Anon. 1991). Before chemical seed treatments were extensively used, losses caused by CB and LS were substantial. Incidence of CB was often more than 50% and sometimes the crop was destroyed (Fischer and Holton 1957, Cramer 1967, Jones and Clifford 1978). In general, the use of effective chemical seed treatments reduced CB incidence to less than 1% in the United Kingdom as well as in Europe in general (Cramer 1967, Jones and Clifford 1978, Johnsson 1991a, Yarham 1993).

The CB fungi are found throughout Europe and Central West Asia (Figure 1.1), but *T. laevis* has not been reported in Great Britain and Ireland (Anon. 1977).

CB occurs throughout the former Soviet Union, especially in the black soil regions of the Central Region, the north Caucasus Krasnodar region and the Ukraine (Zhivotkov et al. 1989).

DB is common throughout eastern and northern Europe, but is absent from the United Kingdom and Ireland (Anon. 1968, Tragner-Born and Kaspers 1981). It is present in Armenia, Azerbaijan, western Ukraine, Moldova, and northern Caucasus mountain areas (Gair et al. 1972, Zhivotkov et al. 1989).

LS is widely present and causes significant losses when susceptible cultivars are sown in environments favorable for infection. Up to 20% incidence has been recorded, but incidence is below 1% at present (Jones and Clifford 1978). The extensive use of certified seed, chemical seed treatments, and resistant cultivars has brought LS under control.

LS in the former Soviet Union is prevalent wherever wheat is grown. Incidence is highest in southern Kazakhstan (Baraev 1983) and Siberia (Zhivotkov et al. 1989).

FS occurs on grasses in many countries of Europe, but the pathogen form on wheat is not widely distributed (Purdy 1965, Anon. 1991, Line 1993c, Chapter 5 in this manual). Incidence of FS is not serious. In the former Soviet Union, FS distribution is limited to more southern regions of Georgia, Armenia, Azerbaijan, and some Central Asian republics (Zhivotkov et al. 1989).

KB has not been established in Europe (Warham 1986, Chapter 3 in this manual).

Conclusion. Extensive use of certified seed and chemical seed treatments has reduced the importance of bunts and smuts in much of Europe. In the republics of the former Soviet Union, the diseases cause significant losses, and in the future, the bunt and smut diseases and associated losses will probably increase in importance.

The Americas

In North America, information on bunts and smuts has been extensively reviewed (Fischer and Holton 1957, Martens et al. 1984, Wiese 1987, Mathur and Cunfer 1993). Information has been brought up to date in Chapters 2, 3, 4 , and 5 of this manual.

In South America, CB occurs on bread wheats in most countries (Anon. 1977, 1990). In Argentina, it has historically been a major disease, causing serious losses (Fischer and Holton 1957, Mamluk and Zahour 1993), but durum wheats are resistant to CB (Antonelli 1983). In Brazil, CB is not a problem (Mehta 1993).

LS occurs in all South American countries that cultivate wheat (Fischer and Holton 1957, Anon. 1982). Generally, it is a minor disease, but extensive cultivation of highly susceptible cultivars has increased disease incidence in Argentina (Antonelli 1983). In Brazil, LS incidence is generally low, but susceptible cultivars show 6-12% infection (Mehta 1993).

FS has limited distribution in South America (Purdy 1965, Anon. 1991, Line 1993c). DB has been reported only from Argentina and Uruguay (Anon. 1968) and KB has not been established in South America (Warham 1986).

Conclusion. The importance of bunts and smuts in North America has been well documented. In South America, the diseases occur, and reasonable control measures and resistant cultivars are generally available to manage them. The release of susceptible cultivars and relaxing of seed treatment practices suggests that resurgence of the diseases is possible in some areas.

Oceania

In Australia, CB was a serious disease and caused significant losses before the extensive use of chemical seed treatments (Holton and Heald 1941, Fischer and Holton 1957). Today, losses have been reduced to very low levels (Brennen and Murray 1988). However, the potential importance remains high because soils are infested with low levels of inoculum from which epidemics can quickly develop (Andrews and Ballinger 1987, Murray and Brown 1987). The detection of CB teliospores in seed lots treated with seed dressings is cause for concern (Ballinger and Gould 1988).

LS is widespread in Australia, but currently, it causes little loss (Murray and Brown 1987, Brennen and Murray 1988). KB and DB are not registered in Australia.

FS was a disease of major importance (Purdy 1965, Line 1993c, Chapter 5 in this manual), but is now controlled through use of resistant cultivars and chemical seed treatments. Currently, losses are light (Brennen and Murray 1988). The disease still has potential to be serious when susceptible cultivars are sown (Murray and Brown 1987, Ballantyne 1993).

Conclusion. The bunt and smut diseases can cause damage but resistant cultivars and seed treatments have controlled them at present. Experience suggests that resurgence of the diseases will quickly occur if management practices are relaxed.

Control strategies

Chemical Control

Appropriate chemical seed treatments effectively control bunt and smut diseases of wheat. Several new systemic fungicides are highly effective against seed and soilborne inoculum. The major constraints are high cost of seed treatment and poor distribution of treated seed, especially in developing countries. In low-input agriculture areas with wheat as the sole crop, farmers seldom use treated certified seed every planting season. Further, they usually do not have seed treatment facilities. Currently, most private and public-sector seed companies that produce certified seed also treat seed with fungicides. Seed treatment chemicals are often unavailable or are not effective in developing countries, and benefits have not been felt in these countries. The use of chemicals in agriculture, even seed treatment chemicals, will likely decrease in the future because of the growing awareness of farmers of the problems associated with usage.

Resistant Cultivars

The most feasible approach for managing the bunt and smut diseases of wheat is to use resistant cultivars. Development of such cultivars is costly, but they directly benefit farmers, especially small farmers in developing countries, by assuring production. Because of the likely decrease in the use of chemical seed treatments, there is a growing need to increase development of disease resistant cultivars to combat the bunt and smut diseases of wheat.

A major deterrent in the development of bunt and smut resistant wheat cultivars is the lack of knowledge on genetic variability of bunt and smut pathogens. Such studies have been relatively few in comparison with such studies on pathogens of other diseases. This may be because of intense labor and large amounts of time required for determining races and biotypes of bunt and smut fungi. The lack of studies may also be due to the assumption that the bunts and smuts can be easily managed by resistant cultivars and seed treatments. The result has been a low research priority for the bunts and smuts in most countries.

CHAPTER 2

Common Bunt and Dwarf Bunt

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History

Common bunt (CB), caused by *Tilletia laevis* Kühn and *T. tritici* (Bjerk.) Wint., and dwarf bunt (DB), caused by *T. controversa* Kühn, are two of the world's most destructive wheat diseases. CB was used by Tillet (1755) in some of the earliest demonstrations of the cause and contagious nature of parasitic plant diseases. Tillet's work, along with other classical studies on bunt disease, helped lay the foundations of modern plant disease science (Prévost 1807; Tulasne and Tulasne 1847, 1854; De Bary 1853; Brefeld 1883). In recognition of Tillet's pioneering work, the genus of the bunt fungi was named *Tilletia*. The origin of the term "bunt" is obscure, but may have originated from a local pronunciation of "burnt" (Durán and Fischer 1961).

CB of wheat has been recognized since ancient times. DB escaped notice as a disease distinct from CB for many years, although DB of wheat was probably seen in 1892 in North America (Harwood and Holden 1892) and 1907 in Europe (Todorova 1958). DB was observed in North America and Europe in the early 1930s (Fischer and Tyler 1952, Wagner 1948) and was called "short smut," "stunt bunt," "stubble smut," "dwarf smut," or "dwarf bunt." Herbarium specimens provide evidence that the DB pathogen was present by 1847 in Europe, 1860 in North America (Fischer and Durán 1956b), and 1915 in South America (Lindquist et al. 1954).

DB was first officially recognized as distinct from CB in 1935 (Young 1935). For many years, the pathogen was thought to be a physiologic race of the CB organism *T. tritici*. However, this concept did not continue because unlike CB, DB teliospores did not produce infection after seed inoculations, and the disease was not controlled by seed treatments (Holton et al. 1949a, Warmbrunn 1952). Also, DB teliospores did not germinate at temperatures normally conducive to germination of *T. tritici* (Young 1935, Holton 1943). In addition, DB teliospores were distinct morphologically and survived for several years in soil, unlike those of CB. The DB pathogen was designated as a separate species in the early 1950s (Wagner 1950, Fischer 1952, Warmbrunn 1952).

During the first half of the 20th century, CB occasionally caused more losses in yield and quality in the USA than any other wheat disease (Fischer and Holton 1957). In many areas of the world, losses due to CB are surpassed only by those caused by rust diseases. CB virtually disappeared in the USA in the late 1950s after the widespread use of polychloronated benzene fungicides in areas with both seedborne and soilborne inocula and by the use of organic mercury fungicides in areas of seedborne inoculum. However, bunt was not eradicated and significant yield losses still occur when seed-treatment fungicides are improperly used (Mathre and Johnson 1976, Williams and Gough 1984, Ballinger and Gould 1989, Williams 1988). Hexachlorobenzene, one of the best fungicides for CB control, was abandoned due to environmental problems, but control of CB has been successful with other seed-treatment fungicides (Hoffmann and Waldher 1981). Efforts to control CB by means of resistant cultivars are important in many parts of the world.

In the USA, only a small percentage of the western winter wheat acreage is affected by DB, and the disease occurs sporadically and usually in low amounts. However, significant losses can occur when snowcover is continuous for approximately two months. In areas where the favorable environment occurs regularly, highly resistant wheat cultivars are commonly grown that have essentially eliminated yield and quality losses due to DB. Historically, resistance has occasionally been overcome (Hoffmann 1982) by virulent isolates present in low frequency in the pathogen population that have increased on resistant cultivars, or by the evolution of isolates with new virulence genes. Thus, a continual search for resistance sources and the development of resistant cultivars are required. The primary sources of resistance against DB in the USA have remained effective for the past 20 years.

Distribution and Importance

CB occurs on both spring- and winter-planted wheat and is present throughout the wheat growing regions of the world (**Figure 1.1**). See Chapter 1.

DB occurs on fall-planted wheat and is usually restricted to areas where snow cover provides favorable conditions for teliospore germination and infection. DB occurs primarily in the Americas, Europe, and West Asia (Niemann 1956; Fischer and Durán 1956a; Durán and Fischer 1956, 1961; Purdy et al. 1963; Anon. 1968) (**Figure 1.2**).

Bunt may infect more than 70% of the spikes when plants are not protected by resistance or chemicals and are grown in

disease-conducive environments where inoculum levels are high. DB reduces yield about 0.8% for every 1% infection of spikes (Slinkard and Elliot 1954, Hoffmann and Sisson 1987), but CB reduces yield about the same as the percentage of infection because it does not affect tillering as DB does. In addition to yield losses, prices are often reduced for contaminated grain due to decreased palatability of wheat products. Also, a quarantine placed on wheat contaminated with DB teliospores has restricted the importation of wheat into the People's Republic of China from areas where DB occurs (Trione 1982).

Hosts

Dwarf Bunt

In addition to *Triticum* spp., *T. controversa* also occurs on numerous grasses including species of *Aegilops*, *Agropyron*, *Agrostus*, *Alopecurus*, *Arrhenatherum*, *Beckmannia*, *Bromus*, *Dactylis*, *Elymus*, *Festuca*, *Holcus*, *Hordeum*, *Koeleria*, *Lolium*, *Poa*, *Secale*, and *Trisetum* and X *Triticosecale* (Hardison et al. 1959, Schuhmann 1960, Durán and Fischer 1961, Hoffmann and Waldher 1964, Ozkan 1971). Many grass hosts were determined after artificial inoculation and are considered hosts only under exceptional circumstances. Even with artificial inoculation, disease incidence is very low for the majority of them. Cultivated winter barley, *Hordeum vulgare* L., is considered a host, but very few barley genotypes are susceptible, and even the most aggressive DB races are only weakly pathogenic on them (Dewey and Hoffmann 1975).

Common Bunt

In addition to *Triticum* spp., most grass species listed above that are susceptible to DB are also susceptible to one or more races of CB (Hardison et al. 1959; Durán and Fischer 1961).

Pathogens

Taxonomy

The CB and DB fungi are Heterobasidiomycetes in the order Ustilaginales and the family Tilletiaceae. *T. tritici* has a reticulate exospore that appears spiny when focused medially with a light microscope, whereas *T. laevis* has a smooth exospore. These two fungi are essentially identical except for differences in spore wall characteristics, which are controlled by very few genes (Fischer and Holton 1957). Natural morphological variants that have a full range of teliospore morphology between *T. tritici* and *T. laevis* have been observed (Flor 1933, Gassner 1938, Bremer et al. 1952, Holton 1954a) and occasionally they have been designated as separate species (Gassner 1938, Savulescu et al. 1942). The morphological intermediates probably represent hybrids. *T. tritici* and *T. laevis* have been hybridized and can produce a full range of morphological variants (Holton 1942).

Taxonomy is complicated by the fact that CB fungi hybridize with each other as well as with the DB organism (Holton 1954b, Holton and Kendrick 1956). Rare, apparently natural hybrids between *T. tritici* and *T. controversa* have been identified that have intermediate teliospore morphology, physiology, or effect on host plants (Holton and Kendrick 1956, Böning 1958, Kendrick et al. 1964).

Obviously, the CB and DB fungi are closely related. Several studies based on the biochemistry and molecular biology of the organisms have failed to differentiate them or have shown only slight differences (M.R. Bonde, pers. comm.; Banowetz et al. 1984; Weber and Schauz 1985; Kawchuk et al. 1988; H. Leung, pers. comm.), whereas studies with these techniques have differentiated other pathogenic fungi to the species or subspecies level (Kim et al. 1982, 1984; Hellman and Christ 1991). Additionally, virulence of the CB and DB fungi is regulated in wheat by the same set of resistance genes (Holton et al. 1949b, Hoffmann and Metzger 1976, Metzger and Hoffmann 1978).

The close relatedness of these bunt fungi has led to the suggestion that they be treated as varieties of a single species (Holton and Kendrick 1956, Kawchuk et al. 1988). *Tilletia indica* (Mitra), which causes partial bunt or Karnal bunt of wheat, has some similarities to CB and DB fungi, but it is taxonomically and biologically distinct. Some authorities have placed *T. indica* in the genus *Neovossia*.

Common bunt. In the recent past, accepted names for the CB fungi were *T. caries* and *T. foetida*. However, the use of the year 1753 as the starting date for the nomenclature of Ustilaginales, under the International Code of Botanical Nomenclature, rather than the old date of 1801, requires that official names revert to *T. tritici* and *T. laevis*, respectively.

Dwarf bunt. Soon after the discovery that *T. controversa* had a life cycle and biology distinct from that of *T. tritici*, a new species was proposed: *Tilletia tritici nanifica* in 1950 (Wagner 1950) and *T. brevifaciens* in 1952 (Fischer 1952). However, Conners (1954) determined that the fungus was the same organism described on *Agropyron* as *T. contraversa* (Kühn 1874), thereby setting precedent. The spelling *contraversa* by Kühn, probably was an error because he used *controversa* in later publications and the Latin derivation of *controversa* is more sensible than *contraversa* (Niemann 1956, Savile 1962). Since 1960, spelling with an “o” has predominated, but spelling is still somewhat controversial (Johnsson 1991b).

Pathogenic Specialization

Well defined pathogenic races exist in DB and CB fungi (Hoffmann and Metzger 1976). Races of *T. tritici* are labeled “T”, races of *T. laevis* are labeled “L”, and races of *T. controversa* are labeled “D”.

Table 2.1. Virulence of common bunt races against wheat bunt (Bt) resistance genes. X indicates an infection level above 10%. From Hoffmann and Metzger, 1976; R.J. Metzger, per. comm.

	Host resistance genes (Bt)													
Race	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
T-1							X								
T-2	X						X								X
T-3		X					X								
T-4	X						X								
T-5	X	X					X								
T-6	X						X								
T-7	X	X					X								
T-8	X	X					X								
T-9					X		X								
T-10					X										
T-11		X	X												
T-12	X				X		X								
T-13	X	X	X										X		
T-14	X														
T-15	X	X			X		X								
T-16		X		X	X	X	X								
T-17				X		X	X								
T-18	X			X		X	X								
T-19	X	X	X				X								
T-20	X	X													
T-21	X	X		X		X	X								
T-22		X		X		X	X								
T-23	X	X		X		X	X		X						
T-24		X	X	X		X	X								
T-25	X						X			X					
T-26	X	X					X			X					
T-27	X	X		X		X	X			X					
T-28		X		X		X	X		X						
T-29	X	X					X		X	X					
T-30	X	X		X		X	X		X	X					
L-1							X								
L-2	X						X								X
L-3		X					X								
L-4	X						X								
L-5	X	X					X								
L-7	X	X					X							X	
L-8		X		X		X	X		X						
L-9	X	X	X												
L-10		X	X				X								
L-16	X	X		X		X	X								

Table 2.2. Virulence of dwarf bunt races against wheat bunt (Bt) resistance genes. X indicates an infection level above 10%. From Hoffmann and Metzger, 1976; Hoffmann, 1982.

	Host resistance genes (Bt)									
Race	1	2	3	4	5	6	7	8	9	10
D-1					X *		X			X
D-2				X	X *	X	X			
D-3	X	X	X	X		X	X			
D-4	X	X			X *		X			
D-5	X			X	X *	X	X			
D-6	X	X		X	X *	X	X			
D-7		X	X	X		X	X			
D-8	X	X	X				X			
D-9	X	X	X	X	X *	X	X			
D-10		X	X		X *		X			
D-11				X	X *	X	X			X
D-12	X			X	X *	X	X			X
D-13	X	X		X	X *	X	X			X
D-14		X	X	X		X	X			X
D-15	X	X	X	X	X *	X	X			X
D-16	X	X	X				X		X	
D-17		X	X		X *		X		X	

* X indicates virulence to an Elgin/Hohenheimer cross. It was determined later that Bt-5 in this selection was not fully expressed, and that there is little virulence to the full Bt-5 gene of Hohenheimer.

The races are defined on the basis of their virulence/avirulence to host resistance genes (**Tables 2.1** and **2.2**; (Hoffmann and Metzger 1976, Hoffmann 1982). In addition to numbered races, several other pathogenic types have been identified (Hoffmann 1982).

Wheat lines that are monogenic for individual bunt resistance genes that were previously identified (Briggs, 1926, 1930, 1933; Gaines and Smith 1933; Stanford 1941; Schaller et al. 1960) or developed by R.J. Metzger are included in a standard set of differential cultivars (**Table 2.3**). The virulence/avirulence against particular resistance genes can be determined by testing bunt isolates against the differentials.

Virulence of DB and CB fungi is regulated in wheat by the same resistance genes (Holton et al. 1949b, Hoffmann and Metzger 1976, Metzger and Hoffmann 1978). A cultivar that is resistant to a race of CB will also be resistant to a race of DB that has the same or fewer virulence genes. Breeders testing for DB resistance can initially screen for resistance with races of CB, which are easier to work with regarding inoculation and reliable environment for infection. Inoculation with

Table 2.3. Differential cultivars used to determine pathogenic races. Resistance genes 1-15 are designated by "Bt." All are winter wheats except the last two, which are spring types. Bt-11 through Bt-15 are informal designations.

Cultivar	Resistance gene	CI or PI number
Heines VII	Bt-0	PI 209794
Sel 2092	Bt-1	PI 554101
Sel 1102	Bt-2	PI 554097
Ridit	Bt-3	CI 6703
CI1558	Bt-4	CI 1558
Hohenheimer	Bt-5	CI 11458
Rio	Bt-6	CI 10061
Sel 50077	Bt-7	PI 554100
PI 173438 x Elgin	Bt-8	PI 554120
Elgin X PI 178383	Bt-9	PI 554099
PI 178383 x Elgin	Bt-10	PI 554118
Elgin x PI 166910	Bt-11	PI 554119
PI 119333	Bt-12	PI 119333
Thule III	Bt-13	PI 181463
Doubbi	Bt-14	CI 13711
Carleton	Bt-15	CI 12064

mixed races of CB has produced hybrids with new virulence gene combinations that have proven useful for identifying resistance genes (Metzger and Hoffmann 1978).

High disease levels develop only if complementary virulence genes of the pathogen exist for all the resistance genes of a particular host. For example, race T-16 possesses virulence against the resistance genes *Bt-2*, *Bt-4*, *Bt-5*, *Bt-6*, and *Bt-7*. If a plant has any resistance genes other than these, it will be resistant to race T-16. However, if the host carries only these resistance genes, or if it lacks one or more of them, it will be susceptible to race T-16. This is a good example of the classic gene-for-gene system (Flor 1947).

Teliospore Morphology, Germination, and Physiology

Morphology. Teliospores of bunt fungi have relatively thick, three-layered walls (Hess and Gardner 1983, Hess and Trione 1986). Those of *T. controversa* and *T. tritici* have a net-like, reticulate exospore covered by a hyaline, gelatinoid sheath (**Figures 2.1a,b** and **2.2**). The sheath is prominent in *T. controversa* but is relatively thin and inconspicuous in *T. tritici* when viewed with a light microscope. The exospore of *T. laevis* teliospores is smooth (**Figure 2.3**). The interior of dormant teliospores contains numerous spherical translucent lipid bodies. Among teliospores are hyaline, smooth-surfaced, thin-walled, spore-like, sterile cells that form during teliosporogenesis.

The taxonomy and identification of *Tilletia* spp. on wheat and grasses are based primarily on teliospore morphology, but host range and the environment required for teliospore germination are also important. There is often considerable variation in the depth of reticulations among teliospores isolated from individual sori, from different races, as well as from geographical areas (Holton and Kendrick 1956, Holton et al. 1968). There is an overlap of about 10% in the morphology of the reticulate species of wheat bunt fungi that makes positive identification of an individual teliospore difficult or impossible. However, when numerous teliospores are observed, species can almost always be correctly identified with light microscopy. Most of the overlap in morphology occurs because typically some *T. controversa* teliospores within individual sori have short reticulations like those of *T. tritici* Isolates of *T. tritici*, which have individual teliospores with a reticulation depth close to the average of *T. controversa*, are extremely rare. Deep reticulations and a prominent sheath that often expands beyond the reticulum in *T. controversa* are the best morphologic characters for differentiating this species from *T. tritici*

Below are technical descriptions of teliospores and sterile cells mounted in Shear's mounting fluid, as described in the "Techniques for Study" section on page 21 (Durán and Fischer 1961).

Figure 2.1a. Teliospores and sterile cells of *T. controversa* Sterile cells lack pigment and ornamentation.

Figure 2.2. Teliospores and sterile cells of *T. tritici* Sterile cells lack pigment and ornamentation.

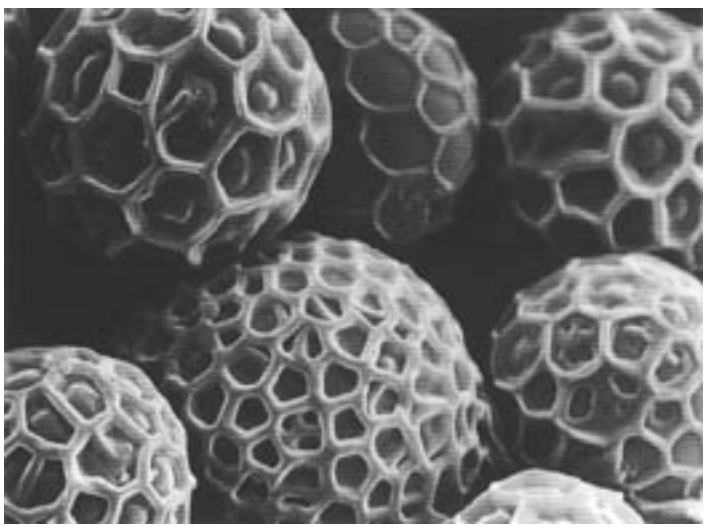

Figure 2.1b. Teliospores of *T. controversa* showing exopore morphology. (Courtesy W.M. Hess.)

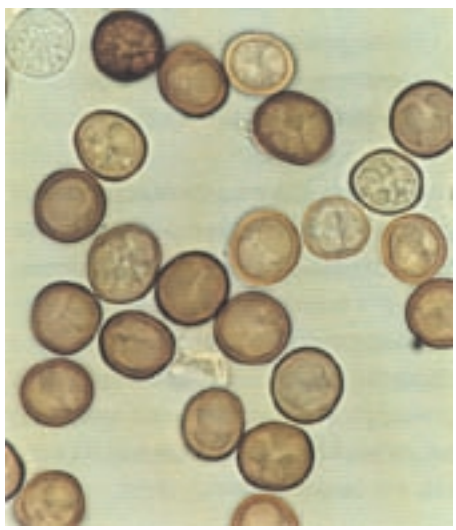

Figure 2.3. Teliospores and sterile cells of *T. laevis* Sterile cells lack pigment and ornamentation.

- *Tilletia tritici*—Teliospores are light pale yellow to gray or reddish brown, generally globose, less frequently subglobose, occasionally ovoid, 14-23.5 μm in diameter, occasionally up to 25 μm. The exospore usually has polygonal reticulations 0.5-1.5 μm deep that vary in diameter and are occasionally somewhat cerebriform (Figure 2.2). Sterile cells are globose to subglobose, 9.8-18.2 μm in diameter, and hyaline to subhyaline.

- *Tilletia laevis*—Teliospores are light pale to dark olivaceous brown, globose, ovoid, occasionally elongate, and 14-22 μm in diameter, but occasionally smaller (13 μm). The exospore is smooth (Figure 2.3). Sterile cells are globose to subglobose, but occasionally irregularly shaped or contorted, 11-18 μm in diameter, and hyaline to subhyaline.

- *Tilletia controversa*—Teliospores are yellowish brown to reddish brown, mostly globose or subglobose, embedded in a hyaline gelatinoid sheath 1.5-5.5 μm thick and 19-24 μm in diameter, though occasionally 16.8-32.0 μm, including the sheath (Figure 2.1). The exospore usually has regular polygonal reticulations 1.5-3 μm deep, but are occasionally irregular to subcerebriform. Regular polygonal areolae are 3-5 μm in diameter. Sterile cells are regularly globose, 9-22 μm in diameter with smooth walls, hyaline or faintly greenish or brownish, and sometimes encased in a hyaline gelatinoid sheath 2-4 μm thick.

Germination. The process of teliospore germination is similar in the DB and CB fungi. The promycelium (basidium) grows through a hydrolyzed area of the spore wall and extends to a variable length depending on environment. On agar, the promycelium sometimes grows extensively before producing primary sporidia, but on soil, it is usually extremely short. Excessive moisture promotes long promycelia. Occasionally, the promycelium develops a branch, but this is more common in *T. controversa* than in the CB fungi. Filiform primary sporidia (basidiospores) grow from the tip of the promycelium to form a compact bundle (**Figure 2.4**). *T. controversa* typically has 14-30 primary sporidia, whereas CB fungi have 4-16 (usually 8-12). The cytoplasm of the teliospore passes into the promycelium and then into the primary sporidia. Septa form in the promycelium behind the migrating cytoplasm. Mature sporidia can be removed from the promycelium with a slight touch. There are usually two mating types within a given bundle of primary sporidia, which are arbitrarily designated by a (+) or a (-). Pairs of primary sporidia of opposite mating type within the bundle fuse with each other, usually near their mid-region, by means of short conjugation pegs (Kollmorgen et al. 1978, 1979). The structure formed by fusion of primary sporidia is called an H-body (**Figure 2.5**). Rarely, only one mating type occurs in a bundle and then fusion of sporidia does not occur. Although primary sporidia usually fuse in pairs, fusion between three or more sporidia has been observed.

Figure 2.4. Germinated teliospore of *T. tritici* showing promycelium, primary sporidia, and H-bodies that formed by fusion of sporidia.

Fused primary sporidia produce infection hyphae, vegetative hyphae, or secondary sporidia of allantoid or filiform type (**Figures 2.6** and **2.7**). Infection hyphae are usually more robust than vegetative hyphae and often have a sinuous curving growth habit. The allantoid secondary sporidia (secondary basidiospores) form on short sterigmata and are forcibly discharged (Goates and Hoffmann 1986). Secondary sporidia may produce infection hyphae, vegetative hyphae, or additional allantoid or filiform sporidia (Goates and Hoffmann 1979).

Physiology. The germination rate of the teliospores of *T. laevis* and *T. tritici* differs with isolates and/or pathogenic races (Lowther 1950) and occurs over a wide range of temperatures. Teliospores germinate most rapidly at 18-20°C, but most uniformly at 14-16°C. Germination occurs after 4-5 days at 15°C, and after 10-14 days at 5°C, under optimal laboratory conditions. Fewer primary sporidia are produced at 5°C than at 15°C

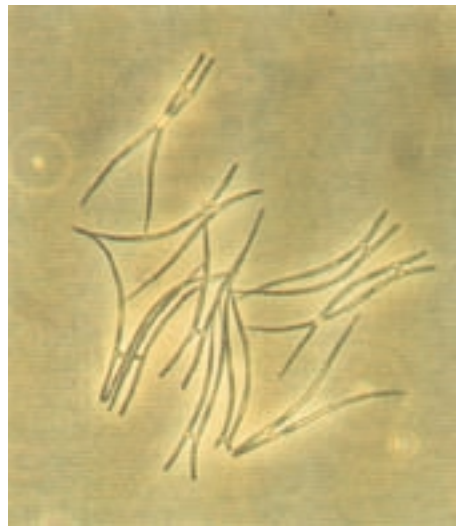

Figure 2.5. H-bodies of *T. tritici*.

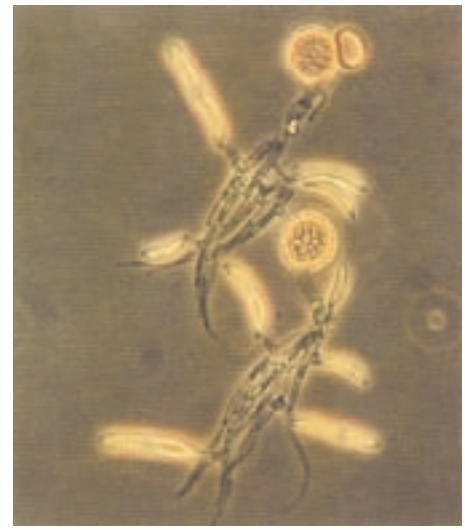

Figure 2.6. Allantoid secondary sporidia of *T. tritici*.

(Goates and Hoffmann 1987). Teliospore germination of the CB fungi is stimulated very little by light, unlike *T. controversa* (Gassner and Niemann 1954).

The geographical distribution of *T. controversa* is limited to areas that have a prolonged period of persistent snow cover. Snow insulates the ground and provides the stable low temperatures and high humidity needed for teliospore germination and infection.

T. controversa teliospores usually germinate within 3-6 weeks under optimal laboratory conditions, but the time varies considerably among isolates. Some European isolates require more time than North American isolates (Baylis 1958). Some isolates germinate uniformly, whereas others are erratic. Cardinal temperatures of germination are: -2 (minimum), 3-8 (optimum), and < 15°C (maximum) (Hoffmann 1982). Incubation at 5°C is recommended for routine work. Germination at above 19°C provides a quick way to differentiate *T. tritici* from *T. controversa*

Germination of *T. controversa* teliospores is stimulated by low levels of light when supplied in environments conducive to germination (Baylis 1958, Gassner and Niemann 1954). Little or no germination occurs in darkness with some isolates (Pichler 1953, Aebi 1956). Light in the green range reduces germination, whereas that in the blue range increases it, compared to controls in white light (Ettel and Halbsguth 1964). Radiation between 400 and 600 nm is most effective at stimulating germination (Aebi 1956). For routine work, light may be supplied during incubation by two 40-watt cool white fluorescent lamps that are built into incubators.

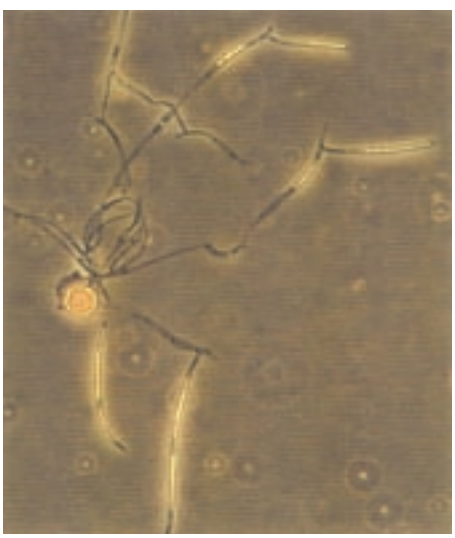

Figure 2.7. Filiform secondary sporidia of *T. tritici*.

Teliospores of *T. controversa* that are stored in the laboratory germinate readily in a favorable environment, but those in sori that stay in the field over the winter are dormant from March to June. Fewer than 1% of the teliospores germinate when sori are collected from the soil surface about May 1 (Zogg 1959, Hoffmann and Goates 1981, Hoffmann 1982). The dormancy may be induced by prolonged low temperature and moisture during the winter, and may be broken by warm temperatures and drying during spring and summer.

High germination percentages of CB and DB teliospores occur when pH is neutral to acidic, but germination is reduced at pH 7.8-8.2 (Baylis 1958). Soil type is not a critical factor of disease development (Holton et al. 1949a, Warmbrunn 1952, Baylis 1958).

Teliospores of *T. controversa*, *T. laevis*, and *T. tritici* may remain viable for more than 20 years when stored in a dry atmosphere at ambient temperature in the laboratory. Although the upper limits of *T. controversa* spore longevity in the field are not known, at least a low percentage of teliospores were viable for 10 years in soil under natural conditions (Tyler and Jensen 1958). The chemical trimethylamine, which is responsible for the fishy odor of bunt fungi, inhibits germination (Ettel and Halbsguth 1964). Other unidentified endogenous compounds of teliospores can also inhibit germination (Trione 1977). Teliospores that remain in sori do not germinate, probably because of high concentrations of self-inhibiting compounds.

In soil under natural field conditions, teliospores of the CB fungi are viable for about two years (Woolman and Humphrey 1924). Field nurseries will be free of viable teliospores when they have been out of wheat production at least two years and are located away from possible sources of windborne inoculum.

Nuclear Cycle

For all three pathogens, dormant teliospores contain a single diploid nucleus that undergoes meiosis and usually one or two synchronous post-meiotic divisions just prior to germination. After germination, the haploid nuclei pass into the promycelium with the cytoplasm and one nucleus migrates into each primary sporidium where it undergoes mitosis. Then one of the haploid nuclei returns to the promycelium. The nuclei left in the promycelium migrate into anucleate primary sporidia or degenerate (Goates and Hoffmann 1987). Thus primary sporidia are haploid. The pathogenic dikaryophase is initiated when primary sporidia of opposite mating type fuse to form H-bodies. Hyphae or secondary sporidia produced by H-bodies are dikaryotic and usually binucleate. However, they may contain variable numbers of nuclei (Goates and Hoffmann 1979).

The pathogen maintains the dikaryotic state throughout the period it resides in the host until karyogamy occurs during teliosporogenesis in nascent sori. However, dikaryotic nuclei may disassociate and reassociate before teliosporogenesis begins, as suggested by observations of uninucleate and multinucleate hyphae within the host or after isolation from the host (Dastur 1921; Churchward 1940; Trione 1964, 1974; Churchill and Mills 1985).

Infection and Disease Cycle

T. laevis and *T. tritici*

The life cycle of CB fungi is illustrated in **Figure 1.6**. Infection occurs below the soil surface, shortly after the seed germinates and prior to emergence. Teliospores on seed or in the soil germinate and eventually produce infection hyphae that penetrate the coleoptile. Hyphae become established initially in both resistant and susceptible cultivars (Hansen 1958, Swinburne 1963) but do not progress to the apical meristem of resistant plants. Hyphae must inhabit apical meristems before internode elongation or disease does not develop. After penetrating the coleoptile, hyphae enter the first leaf base and then go through successive leaf bases, or down the leaf base to the area directly beneath the apical meristem. Intercellular hyphae are present in the apical meristem by about the 5-leaf stage (Swinburne 1963). Further development of hyphae is the same as described below for DB.

Detection of hyphae in the apical meristem of seedlings can accurately predict disease development (Kollmorgen and Ballinger 1987). To check seedlings for hyphae, tissue is cleared by autoclaving in 30% potassium hydroxide, stained with 1% aqueous trypan blue, and examined with a microscope.

The incidence of CB is enhanced by seeding at a depth of 7 cm compared to 4 cm (Swinburne 1963). As soil depth increases, temperatures are more favorable for infection, but in addition, infection and establishment of hyphae in the apical meristem are enhanced because the formation of the crown node is delayed (Swinburne 1963).

CB incidence is high when soil moisture ranges from near the permanent wilting point to field capacity, but is optimum when moisture is midway between field capacity and the permanent wilting point (Purdy and Kendrick 1963). Infection is optimum at 5-10^{o}C soil temperature, and slight infection occurs at 22^{o}C (Purdy and Kendrick 1963). Infection occurs at higher soil temperatures with natural soilborne inoculum than it does with seedborne inoculum (Purdy and Kendrick 1963). This is because soil may contain germinated spores during planting, but with seedborne inoculum few spores will have germinated when the plants are at a susceptible growth stage.

Environmental conditions after infection can influence disease development. The resistance of some cultivars decreases if low temperatures (approximately 10^{o}C) occur during early plant development (Smith 1932; Griffith et al. 1955; Zscheile 1955, 1966). Certain cultivars that are facultative for winter or spring habit are susceptible when planted in the fall, but resistant when planted in the spring. This indicates that the expression of certain resistance genes is temperature dependent. Photoperiod can also affect bunt infection. Plants given 14.5 to 16 hours of light per day have greater incidence of smut (Zscheile 1966). Other unknown environmental factors can also influence the expression of resistance. A particular race/cultivar combination can show either virulence or avirulence depending on geographical location, while in the same tests, other race/cultivar combinations are unaffected (Rodenhiser and Holton 1942).

CB and DB can occur in the same field, but only occasionally occur on the same plant. However, both pathogens have been observed in the same sorus (Fischer and Holton 1957). Competitive interactions between races and the species have been observed (Bamberg et al. 1947, Rodenhiser and Holton 1953).

Symptoms. Symptoms of CB may not be apparent until after heading, when sporulation begins in the very young ovary. Immature infected spikes are usually darker green, and remain green longer, than healthy spikes. Mature infected spikes are usually slightly lighter in color and often have a slight bluish-gray color when compared to uninfected spikes (**Figure 2.8**). Infected culms may be slightly stunted, but they are generally normal in height. Sori are generally kernel-shaped (**Figure 2.9**), but usually less spherical than those of DB. Florets are only slightly flared, but otherwise spikes have a near normal appearance (Figure

Figure 2.8. Wheat spikes from six healthy (left) and diseased (right) cultivars showing color and morphological changes associated with common bunt infection.

2.8). Other symptoms include failure to extrude anthers, production of nonviable pollen, young ovaries with green walls, increased numbers of floral primordia, lengthened rachises, partially smutted spikes and kernels (**Figure 2.10**), and a fishy odor. These symptoms are described in more detail in the DB section below.

T. controversa

The life cycle of DB is illustrated in Figure 1.6. The primary source of inoculum is teliospores deposited in soil from a previously diseased crop, or that were deposited on soil by wind from adjacent areas. The disease is initiated when infection hyphae penetrate seedlings after teliospores germinate at or near the soil surface. High levels of disease incidence depend on a long period of stable, low temperature and moisture, which is provided by a deep persistent snow cover (Tyler and Jensen 1953, 1958). DB does not occur on spring-sown wheat because of the lack of this critical environment, in addition to the spring dormancy of teliospores discussed earlier. Infection from seedborne inoculum is rare even with extremely high levels of seed infestation (Holton et al. 1949a, Wagner 1949, Grey et al. 1986).

Figure 2.9. Sori of *T. tritici*, similar to those of *T. laevis*.

Figure 2.10. Partially bunted wheat kernels due to *T. tritici* or to *T. controversa*, which resemble those due to *T. indica*

There are no detailed histological studies on initiation of natural infection by *T. controversa*; however, it is generally believed that infection hyphae penetrate the host through tiller initials. On artificially inoculated coleoptiles, hyphae grow systemically from leaf primordia to the leaf bases and then through the node to the apical meristem and tiller initials (Fernández et al. 1978). Hyphae are intercellular and move with the apical meristem during internode elongation. Although hyphae have been detected in certain resistant cultivars (Fernández et al. 1978), for an unknown reason they do not reach the apical meristem. Teliosporogenesis occurs in ovarian tissue (Trione et al. 1989), which is almost completely consumed by the fungus. The ovary wall is modified and forms the exterior of the kernel-shaped sorus. Teliospores are released when the fragile ovary wall is ruptured during threshing or when spikes get very wet, causing the sorus to swell and rupture.

When snow cover is uninterrupted, infection can occur from December through April in the Pacific Northwest of the USA , but most infection occurs during late December through February (Purdy et al. 1963). Under natural conditions, plants are most susceptible when tillering begins or when only a few tillers are present (Hoffmann and Purdy 1967), although some infection occurs in plants that have many tillers, or in plants in the single-leaf stage. Plants are more susceptible when seeded at a shallow depth (1 cm) compared to deep seeding (6 cm) (Tyler and Jensen 1953, Meiners et al. 1956, Hoffmann and Purdy 1967). In addition, the incidence of DB is greater where soil is compacted, such as in tractor and drill wheel tracks (Holton et al. 1949a, Meiners et al. 1956).

Symptoms. After early spring growth, the first symptoms of DB infection may be seen on leaves as small, faint or distinct yellow spots and/or streaks (**Figure 2.11a,b**). The extent of flecking varies with host genotype, severity of infection, and to some extent with environment. Flecking occurs in resistant as well as in susceptible genotypes (Schuhmann 1960), but some highly susceptible genotypes show very little or no flecking. Infected plants produce an abnormally high number of tillers that are dwarfed (**Figure 2.12**). The culms of diseased heads are usually, but not always,

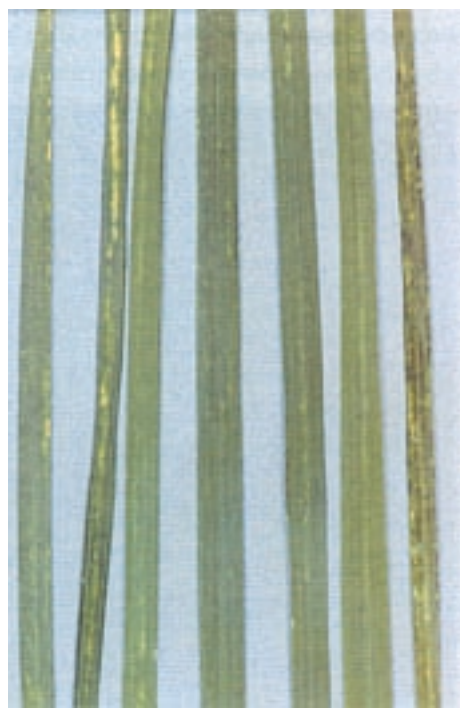

Figure 2.11a. Early symptoms of infection by *T. controversa* on wheat seedling leaves.

shortened and in some resistant genotypes, culms of diseased heads are close to normal height. The number of tillers in fully infected, susceptible plants usually exceeds the number in healthy plants by about 50% (Purdy et al. 1963). The anthers of infected florets fail to extrude, and the pollen is not viable; therefore, fertilization does not occur. The immature ovaries of infected florets are dark green, whereas those of healthy ovaries are very light green. The dark green color is usually apparent before sporulation is macroscopically visible. Teliosporogenesis begins near the center of young ovaries when they are about 1.5 mm long. As the ovary grows, hyphal growth and spore formation expands outward until almost all traces of host tissue inside the ovary wall are consumed. Fully developed sori are generally kernel-shaped, but are more spherical and often larger than normal kernels. This spreads the lemma and palea apart, giving the diseased spike its characteristic appearance (**Figure 2.13**). Mature sori (called bunt balls) consist almost entirely of teliospores and are covered by the thin, modified ovary wall (**Figure 2.14**). Typically, the sori of DB are more round than those of CB. Partially bunted kernels occur, but are uncommon (Figure 2.10) and have characteristics similar to Karnal bunt caused by *T. indica*. Sori emit a strong odor, that is similar to that of rotting fish, which is caused by the chemical trimethylamine. One of the common names, stinking smut, is aptly applied.

The disease commonly occurs in only some of the tillers of infected plants. Usually all, or almost all, of the florets of an individual spike are smutted. However, partial smutting of heads, for example all or a portion of the kernels on one side of a spike, or just a few infected ovaries per spike, is common, particularly in resistant genotypes. Infected spikelets often have an increased number of floral primordia.

Often, the rachis of a bunt infected spike elongates further than in healthy spikes. This essentially converts club-type spikes into common-type spikes, and common-type spikes of certain genotypes become more lax. This characteristic is highly variable among wheat genotypes and is not expressed in some. Increased tillering, dwarfing, and other morphological changes of infected plants appear to be regulated by plant hormones (Trione and Sayavedra-Soto 1988).

Figure 2.12. Dwarfed wheat plant with excess numbers of tillers due to infection by *T. controversa* (right) compared to healthy plant.

Figure 2.11b. Dwarfing of wheat seedling infected with *T. controversa* (left) compared to a healthy seedling.

Control Strategies

Resistance

The development of bunt-resistant cultivars may be the best method to control the disease when resistant sources are available. Fungicides are effective, but they are expensive and may present problems associated with toxicity, environmental hazards, and availability or distribution. Furthermore, chemicals may not control the disease as effectively as resistant cultivars.

Resistant cultivars have successfully controlled DB in the USA, especially in certain dryland areas of southern Idaho and northern Utah where winter wheat production would not otherwise be possible. In these areas, DB has been reduced to trace levels that are insignificant to production and quality. Resistance may be overcome by the selective increase of virulent races or by the development of new combinations of virulence genes in the bunt population (Holton and Vogel 1952, Kendrick and Holton 1961, Purdy et al. 1963). This requires that new sources of resistance be continually sought after and incorporated into cultivars. Intensive

screening of thousands of lines from the United States Department of Agriculture's National Small Grains Collection has identified only a few lines of *Triticum aestivum* and *T. durum* that are highly resistant to races that have a broad based virulence.

PI 178383, originally collected in Turkey, is the principal source of DB resistance currently in use in the USA. PI 178383 has resistance genes *Bt-8*, *Bt-9*, and *Bt-10*, plus an unidentified factor. The resistance from PI 178383 has remained stable in the USA for more than 20 years, primarily because North American isolates lack virulence to *Bt-8*. However, a race that is highly virulent against the resistance genes of PI 178383 has been identified in European isolates of DB (Hoffmann 1982). This clearly demonstrates that potential sources of resistance depend highly on the virulence genes of bunt races that are present in a particular area.

Incorporation of resistance to CB into commercial cultivars in the USA and elsewhere has not had much attention due to adequate control with seed treatment fungicides. However, breeding for resistance to CB is a major part of breeding programs in many countries.

Genes. Resistance to CB and DB is regulated in wheat by the same genes. Thus far, 15 major resistance genes, *Bt-1* to *Bt-15*, have been identified (Hoffmann and Metzger 1976; R.J. Metzger, pers. comm.). The genes occur alone or in combinations in certain wheats. Wheat lines that are monogenic for each *Bt* gene (Table 2.3) are used for pathogenic race identification. An additional major resistance gene, *Bt-Z*, that originated from a translocation into wheat from *Agropyron intermedium*, has been useful for controlling CB in the Soviet Union (Singovets 1974, Mozgovoi et al. 1987, Varenitsa et al. 1987).

Figure 2.14. Sori of *T. controversa*

Figure 2.13. Flaring of awns on a spike due to infection by *T. controversa* (right) compared to a healthy spike.

When bunt resistance genes were first identified, they were designated by the letters *M*, *H*, *T*, and *R*, based on the source cultivars Martin, Hussar, Turkey, and Rio, respectively (Briggs 1926, Schaller et al. 1960). However, these letter designations were replaced as follows: *Bt-1* = M_1, *Bt-2* = *H*, *Bt-4* = *T*, *Bt-6* = R, and *Bt-7* = M_2 (Metzger 1970). Heritable genes other than the major *Bt* genes have been identified that have a weak or modifying effect on host resistance (Smeltzer 1952, Baker 1967). Because the influence of these genes is slight, they have been ignored in resistance breeding programs.

Inheritance. Inheritance studies are complicated because all plants of susceptible genotypes often are not infected when tested, and resistant genotypes are rarely completely immune. In addition, weak modifying genes that are sometimes linked to major resistance genes can influence infection data. Also, many resistance genes show incomplete dominance, which requires testing beyond the F2 generation. Interaction of several resistance genes within a single host complicates the detection of inheritance of single genes. Nevertheless, a significant amount of information has been accumulated on the inheritance of resistance genes. With the exception of a single study (Woodward and Holton 1949), inheritance studies have used CB for screening. But the inheritance data apply to both diseases because DB and CB are controlled by the same resistance genes (Holton et al. 1949b, Woodward and Holton 1949, Hoffmann and Metzger 1976, Metzger and Hoffmann 1978).

The mode of inheritance has been established for genes *Bt-1* through *Bt-10*. *Bt-3* is probably recessive (Churchward 1931, Smith 1933, Taylor 1958), *Bt-1*, *Bt-*

5, *Bt-8*, *Bt-9*, and *Bt-10* have complete dominance (Briggs 1926; Gaines and Smith 1933; Taylor 1958; Sears et al. 1960; Waud and Metzger 1970; Metzger and Silbaugh 1971; Metzger et al. 1977, 1979; Singh and Chopra 1986, 1987), whereas *Bt-2*, *Bt-4*, *Bt-6*, and *Bt-7* have partial dominance with approximately 50% penetrance of heterozygotes (Briggs 1930, 1933; Stanford 1941; Schaller et al. 1960; Schmidt et al. 1969).

Bunt resistance genes have been located on certain chromosomes by analysis of crosses with monosomic wheats, by linkage to morphological factors of known chromosome location, or by linkage to resistance genes where the chromosome location was determined with monosomic wheats (Sears et al. 1960, Metzger et al. 1963, Schmidt et al. 1969, Metzger 1970, Waud and Metzger 1970, Metzger and Silbaugh 1971, Metzger et al. 1979, Singh and Chopra 1987).

Chemical Control

Common bunt. Hyphae infect plants shortly after the seed germinates and before seedlings emerge, which makes control possible with fungicide seed treatments because a high concentration of the fungicide is placed directly in the infection court. Currently, seed treatment chemicals that contain carboxin or pentachloronitrobenzene are commonly used in the USA and provide excellent control of CB. However, both of these chemicals, at commonly used application rates, are less effective at controlling infection from high levels of soilborne inoculum (Hoffmann and Waldher 1981, Gaudet et al. 1989). Despite this, the continued use of fungicides that control seedborne inoculum has apparently reduced the number of soilborne teliospores to the extent that they are not presently a significant problem. CB may occur in trace amounts even where seed treatments are commonly used (Mathre and Johnson 1976, Ballinger and Gould 1989). This low incidence occasionally provides sufficient inoculum to produce significant disease when seed is not properly treated (Williams and Gough 1984, Gaudet et al. 1992).

Chemicals that are effective at controlling infection from both seedborne and soilborne inoculum include carboxin, etaconazole, hexachlorobenzene, thiabendazole, triadimefon, triadiminol, and pentachloronitrobenzene; those that control only seedborne inoculum include benomyl, chloroneb, fuberidazole, maneb, pyrocarbolid, and TCMTB (Hoffmann and Waldher 1981, Gaudet et al. 1989). In tests with seedborne inoculum only, the triazole fungicide myclobutanil has given excellent control (Efthimiadis 1988, Gaudet et al. 1989). The effectiveness of certain fungicides such as maneb and carboxin varies with the environment (Gaudet et al. 1989). The systemic fungicide triadiminol (Baytan), which controls both seedborne and soilborne bunt, has been recently registered for use on wheat in the USA. Triadiminol has consistently given better control than the other fungicides mentioned above (Hoffmann and Waldher 1981, Gaudet et al. 1989, Wainwright and Morris 1989, Williams 1990). The manufacturer's recommended application rates for triadiminol must be closely followed to reduce the effect of growth regulating properties. A promising new fungicide called difenoconazole gives almost complete control of both seedborne and soilborne inoculum at a very low application rate (Williams 1991).

Dwarf bunt. Although certain systemic seed treatment fungicides significantly reduce DB infection, they have not been widely utilized on a commercial basis in the USA because of their cost, phytotoxicity, erratic effectiveness, dependence on late seeding, and the availability of highly resistant cultivars.

Chemical seed treatment requires sufficient concentration of an effective chemical in the plant during the extended winter period when infection occurs. Control with systemic seed treatments has been difficult and often unreliable (Hoffmann et al. 1983), apparently because the chemicals are diluted or metabolized by the plant prior to infection. For this reason, seed treatments are more effective when seed is planted late, so that relatively small seedlings go into winter (Hoffmann and Sisson 1987).

After extensive testing, only the systemic fungicides bitertanol, thiabendazole, triadiminol, and etaconazole were effective (Hoffmann 1971, Tragner-born and Kaspers 1981, Hoffmann et al. 1983). However, each of these fungicides has deficiencies. Bitertanol and thiabendazole provide good control only in certain years and locations, and may cause yield reductions when bunt incidence is low (Hoffmann and Sisson 1987, Dewey and Albrechtsen 1974). Etaconazole and triadiminol are phytotoxic at effective rates. In recent tests, the new fungicide difenoconazole has shown remarkable control as a seed treatment at very low rates with no apparent phytotoxicity (Goates 1992, Sitton et al. 1993).

Cultural Practices

As discussed earlier, DB incidence can be reduced by deep sowing, or by very early or late planting to avoid the plant stages that are the most susceptible. However, these practices may not provide effective control and may reduce yields and cause plants to be more vulnerable to other diseases. In contrast, CB incidence may be reduced by seeding shallow and significantly reduced or controlled by seeding into warm soil (Purdy and Kendrick 1963, Gaudet and Puchalski 1990). The effects of timing and method of cultivation, no-till, and stubble burning on teliospore germination or survival, or incidence of DB and CB, have not been investigated.

Techniques for Study

Teliospore Morphology and Species Identification

Teliospores are typically mounted for viewing with light microscopy in Shear's mounting fluid. This fluid consists of 30 ml 2% potassium acetate in 0.2M phosphate buffer pH 8, 12 ml glycerine, and 18 ml 95% ethanol (Graham 1959). After mounting, the slides are heated over an alcohol flame until boiling begins; this causes the reticulate exospore and sheath to expand, which facilitates morphological examinations.

Most isolates of *T. tritici* and *T. controversa* can be identified by their teliospore morphology as discussed in the morphology section. However, two techniques can aid differentiation of the species. First, when teliospores are mounted in nonfluorescing immersion oil and epi-illuminated with blue light (485 nm excitation, 520 nm barrier filters) for approximately two minutes, the reticulate wall layer of most *T. tritici* teliospores does not autofluoresce, whereas the reticulate wall layer of most *T. controversa* teliospores autofluoresces (Stockwell and Trione 1986). Second, the walls of *T. tritici* teliospores frequently collapse inward when mounted in immersion oil, while wall collapse in *T. controversa* is infrequent (Stockwell and Trione 1986). The percentage of teliospores that autofluoresce or that collapse varies considerably among different isolates of the same species.

The only positive way to distinguish *T. controversa* from *T. tritici* is to germinate the teliospores. Teliospores of *T. tritici* germinate in 3-5 days at 17°C, whereas those of *T. controversa* will not germinate at this temperature. *T. controversa* typically germinates in 3-6 weeks at 5°C under low light intensity.

Germination Media

Teliospores of bunt fungi germinate on a variety of substrates. For CB fungi, each of the media described below—water agar, soil extract agar, nutrient agar, and soil plates—makes satisfactory germination substrates. For *T. controversa*, soil extract agar is superior to water agar or nutrient agar (Meiners and Waldher 1959). On nutrient agars, contaminating organisms may be a problem.

Water agar. For 2% water agar, suspend 20 g of Difco bacto-agar in 1 L of distilled or deionized water in a 2-L flask and autoclave.

Soil extract agar. For 2% soil extract agar, filter 500 ml of boiling water through 75 g of soil and bring the volume of the filtrate up to 1 L with fresh water in a 2-L flask; add 20 g of Difco bacto-agar and autoclave. Pour the agars relatively deep (approximately 20 ml of medium per 9-cm dish) when preparing petri dishes for *T. controversa* to prevent the agar from drying out during the extended period required for germination.

Soil plates. Mix a fine-textured soil with water to form a thick slurry and then pour it into petri dishes. Leave the dishes open until the free water evaporates from the soil surface. Spray a water suspension of teliospores as a fine mist onto the surface of the soil. Because the soil tends to dry out during long periods of incubation, occasionally add water to the plates without disturbing the soil surface. Add water through a short section of plastic straw that is placed upright in the center of the plate before adding the soil slurry.

Growth Media

Extended culture after teliospore germination requires solid or liquid nutrient substrates like potato sucrose agar and T-19 media described below. Generally, cultures of the CB fungi consist primarily of secondary sporidia, whereas those of *T. controversa* consist primarily of mycelium. Usually, in liquid media, filiform secondary sporidia are produced, and on solid media, allantoid secondary sporidia predominate. Isolates and monosporidial lines differ in production of hyphae or secondary sporidia. Cultures are routinely incubated at 15-20°C.

Potato sucrose agar and broth. Autoclave 200 g of diced potatoes in 500 ml distilled or deionized water and filter through two layers of cheese cloth into a 2-L flask; add 20 g of sucrose and 20 g of Difco bacto-agar; bring the volume up to 1 L and autoclave. Potato sucrose broth for liquid culture is made in the same manner, but the agar is excluded.

T-19. This medium has been used to grow bunt fungi through their entire life cycle in culture (Trione 1964). However, for an unknown reason, teliosporogenesis does not always occur after culture of dikaryons on this medium. For 1 L of medium, add the chemicals listed below to 1 L of water in a 2-L flask, adjust the pH to 6.0 and autoclave the solution. For a solid medium, add 20 g of Difco bacto-agar before autoclaving. A modified version of this medium includes additional amino acids, vitamins, and nucleic acid bases (Mills and Churchill 1988).

Chemicals and amounts for preparing T-19 media:

KH_2PO_4	613 mg
$MgSO_4 \cdot 7H_2O$	246 mg
$K_2HPO_4 \cdot 3H_2O$	114 mg
$CaCl_2$	55.5 mg
Chelated Fe	20 mg
$ZnSO_4 \cdot 7H_2O$	3.52 mg
$CuSO_4 \cdot 5H_2O$	0.38 mg
$MnSO_4 \cdot H_2O$	0.031 mg
$Na_2MoO_4 \cdot 2H_2O$	0.025 mg
Thiamin-HCl	5 mg
L-Asparagine	3 g
Sucrose	20 g

Plating Teliospores

Teliospores can be dissected from the interior of sori (bunt balls) without contamination, but teliospores are routinely

surface-sterilized before plating. Suspend the teliospores in water after crushing the sori with a glass rod. Filter the suspension through cheese cloth to remove debris and centrifuge. Teliospores settle rapidly with minimal centrifugation. Place the teliospores in 0.25% aqueous solution of sodium hypochlorite (5% solution of household bleach) for 1 minute at room temperature and then immediately rinse twice in sterile water using centrifugation to remove the disinfectant and rinse water. Suspend the clean teliospores in sterile water to the desired concentration and spread (0.25-0.50 ml/9-cm petri dish) evenly with a sterile glass rod on the agar surface. Allow excess water on the agar surface to evaporate before incubating the plates. Problems associated with contamination by fungal spores can be reduced or eliminated by keeping the water-teliospore suspension at room temperature for 24 hours prior to the surface sterilization procedure. Most fungal spore contaminants will germinate in the water and be killed by the disinfectant. Typically, *T. laevis* and *T. tritici* are incubated at 15ºC; *T. controversa* is incubated at 5ºC supplemented with continuous light.

Inoculation

Seed inoculation—common bunt. Mixing teliospores with seed prior to planting is a simple and effective inoculation method. To inoculate large seedlots, gently mix about 1 g of teliospores per 100 g of seed until the seeds are uniformly covered, then screen off excess spores. Vigorous mixing fractures a high percentage of the teliospores. To inoculate 5-20 g seedlots, place teliospores and seed in envelopes that can be clipped together and shaken by hand.

Cross contamination during planting can be minimized in studies with different isolates by sticking the spores to the seeds using an aqueous solution of methylcellulose. Dissolve methylcellulose that has a viscosity rating of 1500 to 4000 centipoises in water and then dilute the solution until it is slightly more viscous than water but still sticky. Crush the sori in the methylcellulose solution to form a highly concentrated inky black teliospore suspension. Approximately 10 medium-sized sori are sufficient to inoculate 30 5-g seedlots. Place approximately 5 g of seed and two drops of the spore suspension in a 20- x 70-mm glass vial and mix it with a vortex test tube mixer until the seed is uniformly darkened. As an alternative, use a water suspension of teliospores to apply the spores to the seed, but spores will stick better with methylcellulose.

After inoculation, sow the seeds 4-7 cm deep in soil at 5-10ºC. If a single isolate or a composite of isolates is used, seed can be machine-drilled in the field. Otherwise, seeding by hand is required. Bunt incidence of more than 90% is not uncommon when highly susceptible genotypes are inoculated and grown in an ideal environment. Infection is significantly reduced if the seeds are sown shallow in soil warmer than 15ºC.

Soil inoculation—common bunt. Soil is inoculated primarily to evaluate fungicide seed treatments. To properly evaluate the efficacy of fungicides, both the seed and the soil must be inoculated because many fungicides that are effective against seedborne inoculum are not effective against soilborne inoculum (Hoffmann and Waldher 1981). Inoculate the soil by spraying a teliospore suspension into an open furrow prior to hand-seeding. Prepare the teliospore suspension as described in the DB section. Alternatively, mix the teliospores with soil (4 g spores/100 cc soil) and apply at a rate of 4 cc per meter of row (Gaudet et al. 1989). Fungicide studies are typically arranged so that half of the replicates have seedborne inoculum only and half have both seedborne and soilborne inoculum.

Field inoculation—dwarf bunt. To promote high and uniform levels of DB in field-planted nurseries, plant seed about 2 cm deep in the bottom of an 8-10 cm deep furrow with a deep-furrow drill equipped with weighted packer wheels. The seeding date is timed so that plants are in the susceptible 2-3 leaf stage when they become dormant in late fall. Nurseries are inoculated either immediately after seeding or after emergence. The inoculum is a water suspension of teliospores that is prepared by grinding bunted spikes, suspending the material in water, and then filtering through cheese cloth or wire mesh. Dilute the inoculum to a final concentration of 15-25 g spores/L and spray it onto the soil over the row at about 25 ml of inoculum per 1.5 m of row. Periodic agitation is necessary to keep the teliospores from settling. Under ideal conditions, more than 90% of the spikes of highly susceptible genotypes will be diseased.

When there is insufficient snowcover to promote infection, place a cover of vermiculite or straw over the plants to simulate snow (Dewey 1961, 1963). The cover must be removed in early spring to prevent plants from rotting.

Deep snow that lasts into early spring promotes snow mold disease caused by species of *Typhula* and *Microdochium* (= *Fusarium*) that can destroy nurseries. If snow is present in mid-March, it can be melted by dusting furnace ash or other dark colored substances onto the snow. Also, graphite suspended in either water or liquid fertilizer solutions can be sprayed onto the snow. The dark substance absorbs heat from the sun and causes relatively rapid snow melt.

Inoculation of DB in the laboratory. For pathogenic race studies or for increasing DB isolates, laboratory inoculation is required to prevent contamination from teliospores that may be present in the field or adjacent rows.

The inoculation of seed with germinated teliospores is effective but labor-intensive (Meiners 1959). Plate the teliospores on soil extract agar in 9-cm petri dishes at a

rate that covers approximately 10% of the agar surface with teliospores. When approximately 70% of the teliospores have germinated, place 4-5 g of seed and 5 ml of cold water (about 5ºC) in the petri dish and mix it with a sanitized finger or sterile glass rod until the seed is wet and evenly covered with the fungus. The inoculum must be used before sporidia begin to lyse, which can be noted by the beaded appearance of individual sporidia. Sow the inoculated seed approximately 5 cm deep in moist vermiculite in a 12-cm diameter plastic pot and cover it with foil or plastic. Incubate pots at 10ºC until the seedlings are well emerged. Divide the seedlings from each pot into four groups, transplant them into pots containing soil and grow them in a cool (10-15ºC) greenhouse until the plants are established. Either grow the plants to maturity in the greenhouse or transplant them into the field. If using winter wheat, vernalize plants for 7-8 weeks at 5ºC after transplanting into pots. Time the inoculation procedure so that transplanting into the field takes place during early spring.

Seedlings can be inoculated using a variation of the above method. Wash germinating teliospores from the petri dishes with a minimum amount of water and spray them onto seedlings that are in the one- to two-leaf stage. Immediately enclose the plants in a plastic bag to maintain high humidity and incubate them at 10-15ºC for five days. Although this method is less labor-intensive than the seed inoculation technique, it usually results in less infection.

Hypodermic injection. A new crop of teliospores can be rapidly produced by hypodermically injecting older plants with a water suspension of germinating teliospores. Inject the suspension into the boot approximately 2.5 cm above the uppermost node in the region of the developing spike at stage 37 (Zadoks et al. 1974) when the flag leaf is just emerging (Fernández and Durán 1978). Grow the plants to maturity in a greenhouse. This method produces local infection and partially bunted heads with partially bunted kernels after 4-5 weeks. Do not use this method to determine host resistance because resistant genotypes may be susceptible. In a similar method, inject plants 2-3 mm above each node at Zadoks stages 37-39. This induces the formation of bunted adventitious shoots (Kawchuk and Nielsen 1987). Injection with sexually compatible, haploid monosporidial lines from liquid cultures will also result in infection (Trial and Mills 1990).

Race Tests and Increasing Bunt Races

Maintenance of pathogenic races that are highly viable and free of mixtures is essential if specific resistance genes are to be targeted. Increase races using monogenic differential cultivars to insure the presence of the proper virulence genes. Also, use seed that is free of contaminating CB spores. To disinfest, immerse seed in an aqueous solution of formaldehyde (3 parts/thousand solution of 37% formalin) for 10 minutes, and then rinse it thoroughly in running water for 30 minutes. Dry the seed on cloth or paper in a warm greenhouse. Since formaldehyde is a carcinogen, the following method may be preferred. Immerse the seed in a 5% aqueous solution of laundry bleach (0.25% sodium hypochlorite) at 45ºC for 6 minutes and then thoroughly rinse in running water and dry (J.L. Smilanick, pers. comm.). It is important to maintain the temperature during treatment.

For the purpose of race identification, an infection incidence above 10% of the spikes indicates virulence. Infection of the universally susceptible (*Bt-0*) line should be above 70% for adequate evaluation of the bunt isolate. When plants are mature, harvest diseased spikes from the differential cultivar that best discriminates the race from others.

CB or DB race tests are usually conducted in field nurseries in 1.5-m rows for each entry. Artificially inoculate and plant the differential cultivars as described in the inoculation section.

The soil used for CB race tests must be free of CB spores. Achieve this by using a field that has been out of wheat production for at least three years. CB teliospores are typically not viable after two years in the field under natural conditions.

When testing or increasing DB races, use the seed or seedling inoculation techniques described above because of the longevity of DB teliospores under natural field conditions, and also because of potential cross contamination when several races are tested adjacent to each other.

Inoculum Maintenance

For general disease screening, inoculum consists of a composite of races that are representative of those present in a particular area. When the crop is mature, bunted spikes are collected from susceptible and resistant cultivars to maintain composite variability. Store bunted spikes in a cool, dry place in the laboratory. Teliospores usually maintain high viability for 5-10 or more years. Spikes can be ground and sieved to remove most plant debris before storage, without loss of teliospore viability or longevity.

Data Collection

Common bunt. Disease incidence is usually expressed as the percentage of bunted spikes within a row. In some studies, it may be appropriate to express infection as the percentage of plants that show any infection. However, this may not be practical for routine disease screening. Recording the level of infection of individual spikes is useful to differentiate genotypes that have only a few infected florets per spike.

Dwarf bunt. Data are collected in the same manner as described above for CB. Although DB increases the total number of tillers, the percentage of bunted spikes provides a good indication of genotype reaction.

CHAPTER 3

Karnal Bunt of Wheat

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History

Karnal bunt (KB) or partial bunt is the most recently described smut disease of wheat (Mitra 1931, Mundkur 1943b, Bedi et al. 1949). It was first reported in 1931 in experimental wheats at the Botanical Station at Karnal, India (Mitra 1931) and was for many years known only in the plains of India and Pakistan. However, since 1974 it has been noted in many locations across northern India.

KB differs from other smuts of wheat in that the pathogen, *Tilletia indica*, infects during anthesis, unlike *T. caries*, *T. foetida*, *T. controversa*, and *Urocystis agropyri*, and it sporulates on the same generation of the host that it infects, unlike *Ustilago tritici*.

Distribution and Importance

Besides India and Pakistan, KB is known to occur in Mexico (Durán 1972) and Nepal (Singh et al. 1989; **Figure 1.3**). Although it is reported from Iraq (Mathur 1968), this report has not been confirmed by field incidence. A similar situation occurs for Afghanistan and Lebanon where the pathogen was found in wheat samples imported into the USA and India, respectively (Locke and Watson 1955, Nath et al. 1981).

Kernels with small lesions produce normal seedlings, whereas those with severe infection have poor germination and produce weak, distorted seedlings (Rai and Singh 1978, Singh 1980, Bansal et al. 1984b). Despite the fact that increases in disease severity result in proportional decreases in seed weight (Singh 1980, Bedi et al. 1981), yield losses are generally light (Munjal 1975, Brennan et al. 1990). Even during years when epidemics were most severe in India (some seed samples had about 89% infected kernels), losses were only 0.3 to 0.5% of production (Joshi et al. 1983).

KB may also reduce flour quality. In view of the importance of color, odor, and palatability of whole meal and chapaties, 1-4% infected kernels may be sufficient to render wheat grain unacceptable for human consumption (Mehdi et al. 1973, Sekhon et al. 1980, Amaya 1982, Medina 1985, Hussain et al. 1988). At 5% infection, quality distinctly deteriorates (Sekhon et al. 1980). There is also a loss in flour recovery and chemical changes in composition of flour and gluten content cause poor dough strength (Gopal and Sekhon 1988). If grains are washed and steeped, wheat lots with 7-10% infected grains are acceptable for consumption (Sekhon et al. 1981, Medina 1985, Hussain et al. 1988).

In northwestern Mexico, lots of grain with more than 3% infected kernels are rejected by the milling industry. However, they can be used for animal feed (Anon. 1989b). Further, a federal quarantine prohibits growing bread wheat in fields that have more than 2% KB infection. The movement of wheat grain from affected counties to other parts of Mexico is prohibited unless fumigated with methyl bromide. Seed grown for certification has a 0% infection tolerance and must be treated with a fungicide. Trucks, combines, and agricultural machinery cannot leave the quarantined areas unless cleaned and treated as determined by the Department of Agriculture. Movement of germplasm, however, is allowed under certain, but strict quarantine rules (SARH 1987).

Quarantines for KB have been established by many countries to exclude the disease from entry. For example, the USA prohibits importation of all wheat from Mexico, allowing entry only of experimental seed under permits requiring strict safeguards and handling procedures (Anon. 1983, Cooper 1983).

Approximate losses in northwestern Mexico due to KB, including losses in yield and quality, loss of seed and grain export markets, and costs associated with control measures, are estimated at about $US7 million annually (Brennan et al. 1990).

Hosts

KB occurs naturally on bread wheat (*Triticum aestivum*; Mitra 1931), durum wheat (*T. turgidum*), and triticale (X *Triticosecale*; Agarwal et al. 1977). According to Aujla et al. (1985), *T. indica* also occurs on *Triticum shareonensis*, *T. variabilis*, *T. ovatum*, and *T. scerrit*. Although inoculation has infected accessions of the following species, it is not known whether they can be infected under natural conditions: *Oryzopsis miliacea*, *Bromus ciliatus*, *B. tectorum*, *Lolium multiflorum*, *L. perenne*, *Triticum monoccocum*, *T. timopheevi*, *T. tauschii*, *Aegilops triuncialis*, *Ae. mutica*, *Ae. columnaris*, *Ae. caudata*, *Ae. sharonensis*, *Ae. cylindrica*, *Ae. bicornis*, *Ae. comosa*, *Ae. searsii*, *Ae. tauschii*, and *Ae. triaristata* (Royer and Rytter 1988).

Pathogen

Taxonomy

The KB fungus was originally classified as *Tilletia indica* (Mitra 1931) and was later placed in *Neovossia* on the basis of a long promycelium with a whorl of 32-128 nonfusing conidia at the apex (Mundkur

1940). However, others now consider *Tilletia* to be the appropriate genus (Fischer 1953, Durán and Fischer 1961, Waller and Mordue 1983).

Pathogenic Specialization

Mitra (1935) reported two physiologic forms of *T. indica* on the basis of spore size. However, significant differences in teliospore size were not found in collections from five Indian states (Bansal et al. 1984a). Recently, three distinct pathotypes of *T. indica*, which differed in virulence, were present in Punjab (Dhiman 1982). Aujla et al. (1987) reported pathotypes K1, K2, K3, and K4 in 21 collections of *T. indica* from different regions of Punjab and Himachal Pradesh. Their pathotypes were single teliospore isolates from single kernels and were identified on the basis of pathogenicity to genotypes of *T. aestivum*, *T. durum*, X *Triticosecale*, and *Secale cereale*. The pathotypes were morphologically alike and all were virulent on the wheat cultivars used. Pairs of pathogenic monosporidial cultures, each isolated from different teliospores, generally infect greater numbers of spikes than pathogenic pairs from the same teliospore (Fuentes-Davila 1989). Crosses within monosporidial lines from India cause more diseased kernels and spikes than crosses between Mexican and Indian lines (Royer and Rytter 1985). More research is needed to confirm the existence of *T. indica* races and to determine their importance.

Teliospore Morphology, Germination, and Physiology

Teliospores are brown to dark brown, spherical or subspherical or oval; 22-42 x 25-40 μm in diameter, average 35.5 μm; some may be 55 μm (**Figure 3.1**). They occasionally have an apiculus (Roberson and Lutrell 1987), papilla (Mitra 1931) or a vestige of attached mycelium (Durán and Fischer 1961). Teliospores have three wall layers (Khanna et al. 1966; Khanna and Payak 1968; Roberson and Lutrell 1987; Gardner et al. 1983a,b). The primary wall is continuous with the apiculus wall (Roberson and Lutrell 1987). Immature teliospores are sterile, numerous, yellowish or sub-hyaline, rounded, angular or lacrimiform, 10-28 μm in diameter, and have thin, laminated walls (Mitra 1931, Durán and Fischer 1961).

Though each teliospore generally produces one promycelium (Mitra 1931), several promycelia may arise from a single teliospore (Krishna and Singh 1981, Warham 1988). Promycelia vary in length up to 1500 μm (Mitra 1931, Holton 1949) and bear at the apex a whorl of 32 to 128 or more primary sporidia (**Figure 3.2**) (Mitra 1931, Holton 1949). The promycelium may branch (Mitra 1931, Krishna and Singh 1981). Variation in the enlargement of promycelial tips may occur. Primary sporidia are filiform, 64.4-78.8 μm long and 1.6-1.8 μm wide (Peterson et al. 1984).

Germination. Primary sporidia germinate terminally or laterally to produce hyphae or sterigmata from which secondary falcate sporidia, 11.9-13 μm long and 2-2.03 μm wide (Peterson et al. 1984), are formed and forcibly discharged (Fuentes-Davila 1984). Falcate secondary sporidia produce hyphae or other sporidia by repetition. Hyphae that originate from primary or secondary sporidia produce large numbers of secondary, mononucleate falcate sporidia and somewhat fewer secondary filiform sporidia (Fuentes-Davila 1984). H-bodies are not formed by *T. indica* (Mitra 1931), although Holton (1949) reported seeing one H-body. Primary sporidia are initiated by the promycelium 6 to 44 hours after its extension, and production of secondary sporidia may require an additional 128 hours (Warham 1988).

During teliospore germination, meiosis occurs and the haploid nuclei migrate into the promycelium and primary sporidia, each of which receives one nucleus (Fuentes-Davila and Durán 1986). After one or two mitoses, most sporidia become septate forming two to four monokaryotic cells. Most secondary sporidia are mononucleate. Mycelial cells that originate from either type of sporidia are also mononucleate. After anastomosis, the dikaryotic sporogenous mycelium bears intercalated Y-shaped septa formed at the base of the probasidial initials. Nuclei migrate to the teliospore initials, which enlarge to form the teliospores, and the nuclei presumably fuse to form a diploid

Figure 3.1. Teliospores and sterile cells of *T. indica* Mean diameter about 36 μm. Sterile cells lack pigment.

Figure 3.2. Germinated teliospore of *T. indica* with promycelium and primary sporidia.

nucleus (Fuentes-Davila and Durán 1986, Roberson and Lutrell 1987).

T. indica is heterothallic (Durán and Cromarty 1977, Fuentes-Davila 1989). Heterothallism and pathogenicity are controlled by four alleles at one locus (Durán and Cromarty 1977, Fuentes-Davila 1989). Solopathogenic lines have not been found.

Variation in germination is common in reports from different researchers. Freshly collected teliospores are dormant, as indicated by failure of fresh teliospores to germinate (Mitra 1931, 1935). Others report low germination percentages of fresh teliospores (Bansal et al. 1983, Smilanick et al. 1985b). The highest germination occurs with year-old teliospores (Mathur and Ram 1963, Kiryukhina and Shcherbakova 1976, Bansal et al. 1983).

Teliospores of *T. indica* germinate at 5-30°C (Mitra 1935, Bansal et al. 1983, Krishna and Singh 1982a, Zhang et al. 1984, Smilanick et al. 1985b, Dupler et al. 1987). However, germination has been reported after 10 weeks at -18°C (Zhang et al. 1984) or after 4-12 weeks at -30°C. Optimum temperature for teliospore germination is between 15 and 25°C (Mitra 1935, Mundkur 1943b, Holton 1949, Mathur and Ram 1963, Durán and Cromarty 1977, Krishna and Singh 1982a, Bansal et al. 1983, Zhang et al. 1984, Smilanick et al. 1985b). It is better in alternating light (Krishna and Singh 1982a, Zhang et al. 1984) than in darkness or near UV light, but germination in continuous light has been reported (Smilanick et al. 1985b).

Teliospores germinate between pH 4 and 11, the optimum being pH 6-9.5 (Krishna and Singh 1982a, Smilanick et al. 1985b). Under different osmotic and matric potentials, teliospore germination is delayed (Dupler et al. 1987) and both rate and percentage decrease with decreasing water potential. The highest percentage germination occurs at the highest potential tested, -1.4 bars.

Viability. Teliospores are viable in the laboratory for 5-7 years (Mathur and Ram 1963, Kiryukhina and Shcherbakova 1976, Zhang et al. 1984, Krishna and Singh 1983a). Teliospores in unbroken sori and buried 3 or 6 inches in field soil or left on the soil surface can remain viable for 27-45 months (Krishna and Singh 1982b).

Dissemination. Before harvest, dissemination of teliospores is limited, unless the sori break, which occurs infrequently. However, during harvest, sori may be broken and teliospores may contaminate healthy seed, soil, machinery, or vehicles and may be blown by the wind for long distances. Bonde et al. (1987) found viable teliospores up to 3000 m over burning wheat fields, suggesting the possibility of wind dissemination. Teliospores also germinate after ingestion by livestock and grasshoppers, providing another means of dissemination (Smilanick et al. 1986).

Infection and Disease Cycle

The disease cycle of Karnal bunt is illustrated in **Figure 1.7**. Moderate temperatures, high relative humidity or free moisture, cloudiness, and rainfall during anthesis favor disease development (Mundkur 1943a, Bedi et al. 1949, Sattar and Hafiz 1952c, Agarwal et al. 1976, Aujla et al. 1977, Singh and Prasad 1978, Khetarpal et al. 1980, Krishna and Singh 1982c). Disease development was favored in soil at 17-21°C (Aujla et al. 1977). High rates of nitrogen applications, as well as heavy manuring, may increase disease incidence (Bedi et al. 1949, Aujla et al. 1981, Ortiz-Monasterio et al. 1993). In India, cultivars sown late (November 25-December 15) tend to have greater KB incidence than when sown earlier (October 25-November 24; Singh and Prasad 1978, Aujla et al. 1981).

Symptoms

Not all spikes of a plant are affected by KB (Mitra 1935, Bedi et al. 1949), and usually only a few irregularly distributed kernels are bunted. Furthermore, infection of individual kernels varies from small points of infection to completely bunted kernels. Affected kernels are usually partially infected (**Figure 3.3**), and completely infected ones are rare (Mitra 1935, Bedi et al. 1949, Chona et al. 1961). The embryo is largely undamaged except when infection is severe. In infected spikelets, the glumes may be flared to expose bunted kernels (**Figure 3.4**), which reek of an odor similar to rotten fish caused by trimethylamine (Mitra 1935). The spikes of infected plants generally are reduced in length and in number of spikelets (Mitra 1937).

Penetration

The site of dikaryotization in *T. indica* is not known, although apparent hyphal anastomosis has been observed on glume surfaces. Conclusive evidence of the initiation of the dikaryotic phase will require

Figure 3.3. Healthy and affected kernels of wheat showing different levels of infection.

elucidation of the nuclear condition of hyphae during the infection process (Goates 1988).

Sporidial germ tubes penetrate stomata in the rachis (Dhaliwal et al. 1989), glumes, lemma, and palea (Goates 1988, Salazar-Huerta et al. 1990). Growth of germ tubes towards stomata is common and, although germ tubes often penetrate beyond the stomatal ledges, they only occasionally pass into the substomatal chamber.

During early stages of infection, hyphae are intercellular among parenchyma and chlorenchyma cells in the distal to midportions of the glume, lemma, and palea, but not in basal portions. Later hyphae grow intercellularly toward the ovary, subovarian tissue, and rachis. After subovarian tissue is infected, the pathogen enters the pericarp through the funiculus. Hyphae have been seen in the rachis only during the later stages of infection. The epidermis of the ovary wall is not penetrated, even after prolonged contact with germinating secondary sporidia (Dhaliwal et al. 1983, Goates 1988).

Figure 3.4. Wheat spike infected by *T. indica*.

During kernel development, intercellular hyphae proliferate in the middle layers of cells of the pericarp and form a compact, hymenium-like layer (Cashion and Luttrell 1988). These hyphae prevent fusion of the outer and inner layers of the pericarp and fusion with the seed coat. Their terminal cells give rise to the teliospores.

As the fungus grows, it ruptures the pericarp tissues in the bottom of the adaxial groove along the length of the developing kernel. This disrupts the flow of nutrients from the pericarp causing the endosperm to shrink and become cartilaginous. The embryo with attached endosperm may be easily removed and it germinates before or after removal. In the most severe infections, the kernel is reduced to a black membranous sorus and the embryo is killed.

Control Strategies

Resistance

Since the 1940s, cultivars of *T. aestivum*, *T. durum*, and *T. dicoccum* have been reported to be resistant to KB under field conditions in India (Anon. 1943; Bedi et al. 1949; Gautam et al. 1977; Singh et al. 1986, 1988; Singh and Srivastava 1990). X *Triticosecale* cultivars exhibited near immunity to KB in field trials (Meeta et al.1980). Inoculation indicated resistance among experimental lines and cultivars of *Triticum*, *Triticosecale*, and grass species (Aujla et al. 1980, Krishna and Singh 1983c, Warham et al. 1986, Gill and Aujla 1987, Royer and Rytter 1988, Singh et al. 1988, Singh and Srivastava 1990, Fuentes-Davila et al. 1992).

The reactions of lines or cultivars to KB are determined by percent infected kernels per spike (Fuentes-Davila and Rajaram 1994) or as coefficient of infection (Aujla et al. 1989). After a line or cultivar has been evaluated under inoculated and natural conditions in more than 12 tests, it is considered to be resistant if disease incidence averages 5% or less (Gill 1990, Fuentes-Davila and Rodriguez-Ramos 1993, Fuentes-Davila and Rajaram 1994). In CIMMYT's Wheat Program, after five years of testing, 98 lines and cultivars of bread wheat have been identified as resistant, while at Punjab Agricultural University, 68 resistant lines have been identified. Most of these lines trace to germplasm from China, India, and Brazil. In 1992, KB resistant bread wheat cultivar Arivechi was released for commercial use by the Mexican national program for northwestern Mexico.

Inheritance. Studies on the mode of inheritance and allelic relationship among genes conferring KB resistance in bread wheat have indicated two partially recessive and four partially dominant genes (Fuentes-Davila et al. 1995). Other studies indicate polygenic and partially dominant genes; resistance genes are dispersed on chromosomes 1D, 2A, 3B, 3D, 5D, and 7A (Gill et al. 1993).

Impact of Cultivars

The importance and distribution of KB in India appear to be related to periodic widespread cultivation of susceptible or tolerant cultivars, although the impact of weather during these periods is also recognized (Gill et al. 1993). Prior to about 1968, indigenous tall wheats grown in the main wheat belt were KB susceptible, and the disease was often widespread, although usually not severe. The semidwarf wheats Kalyansona, PV18, and Sonalika (introduced in the late 1960s) were more resistant than indigenous wheats and KB decreased somewhat in importance. In 1975, high yielding but KB susceptible cultivars were released, and as their popularity grew, disease severity increased throughout northern India. After 1982, cultivars with KB tolerance were introduced and efforts were made to diversify the cultivars grown by farmers. These changes coincided with a reduction in disease incidence up to 1989. However, the situation changed again with the widespread use of KB susceptible cultivars.

Cultural Practices

Cultural practices appear to have been of little practical value because most recommendations have been based on observation rather than long-term experiments (**Table 3.1**).

Warham and Flores (1988) surveyed farmers in the Yaqui Valley, Sonora, Mexico, from 1983 to 1985 to learn whether cultural practices to control KB were useful. They reported limited use involving soil type, land preparation, origin of seed, irrigation, nitrogen and other fertilizers, weed control, and crop rotation. Rainfall or high humidity at flowering were more important than any of the cultural practices for conditioning KB. Adjustment of sowing dates did not appear to be beneficial because weather fluctuations at anthesis were probably more important. Limiting irrigation during flowering would have adverse effects on yield. A three-year rotation did not appear to be useful because it is too short to affect teliospores in the soil and because inoculum blew into the fields from adjacent areas.

In repeated experiments, Ortiz-Monasterio et al. (1993) found that disease incidence would increase with greater rates of nitrogen. Similar results were obtained with nitrogen applications during sowing compared to split applications. KB incidence was also greater when wheat cultivars were sown in flats instead of beds, and some cultivars showed a direct correlation between plant density and disease incidence.

Table 3.1. Cultural practices suggested by various researchers for Karnal bunt control.

Cultural practice	Reference
Crop rotation	Mitra (1935), Padwick (1939), Singh and Mathur (1953), Singh et al. (1979)
Reduced irrigation and/or fertilizer	Bedi et al. (1949), Padwick (1939), Singh et al. (1979), Dhimann (1982), Ortiz-Monasterio et al. (1993)
Avoid continuous wheat	Singh et al. (1979)
Use disease-free clean seed, avoid late planting	Singh et al. (1979), Dhimann (1982), Lopez-Lugo (1986)
Use deep plowing, fallow after harvest	Dhimann (1982)
Reduce stand density	Lopez-Lugo, (1986), Ortiz-Monasterio et al. (1993)
Plant on light soils	Aujla et al. (1986a)

Seed Treatment

Hot water and solar energy treatments have been applied to KB-infected seeds. However, they have had limited application. These treatments inhibit teliospore germination, but not as much as fungicide treatments (Mitra 1937). Fungicide seed treatments have been investigated for KB control since 1933-34 (Mitra 1935). Although many fungicides have been tested for effectiveness (**Table 3.2**), results have not been satisfactory.

Smilanick et al. (1987) applied seven systemic fungicides to seed (Benomyl, Bitertanol, Campogran, Trimidol, Propiconazole, Thiabendazole LSP, and Triadimefon), planted the seed, and inoculated spikes on some tillers. KB incidence was evaluated on inoculated and naturally infected spikes. None of the fungicides controlled development of the disease.

Some fungicides applied to infected kernels, which were then stored for various periods, inhibited teliospore germination, but others had little or no effect. Most of the fungicides reported to be effective inhibitors of teliospore germination have not been tested for germicidal properties. However, the fact that they inhibit teliospore germination after months of storage suggests they might be useful to control KB or to eradicate the pathogen from infected seed lots. For example, in one test, pentachloronitro-benzene in liquid or wettable formulations applied to wheat seeds inhibited teliospore germination up to two months (Fuentes et al. 1983).

Chlorothalonil, as emulsifiable concentrate and wettable powder applied to infected kernels, inhibited teliospore germination up to eight months (Figueroa-Lopez and Espinoza-Salazar 1988). Triphenyltin hydroxide, methoxyethylmercury acetate, and ethylmercury chloride inhibited teliospore germination for 18 months (Warham and Prescott 1989). Chorothalonil (powder), Mancozeb, and Carbendazim + Mancozeb on infected seed for 10 months resulted in about 97% inhibition of teliospore germination (Salazar-Huerta et al. 1986a,b).

Table 3.2. Chemicals evaluated in wheat seed treatments for Karnal bunt control.

Aujla et al. (1981)
Ethylmercury chloride, Thiram

Aujla et al. (1986b)
Agrozim, Benomyl, Bitertanol, Butrizol, Captan, Carbendazim, Carboxin, ethylmercury chloride, Fenfuram, Fuberidazole, Furavax, Mancozeb, oxycarboxin, pentachloronitrobenzene, thiophanate-methyl, triadimefon, triadimenol

Figueroa-Lopez and Espinoza-Salazar (1988)
Chlorothalonil (EC), Chlorothalonil (WP)

Fuentes et al. (1982)
Benomyl, Carboxin, Corbel, 2 methoxyethyl mercury chloride, hexachlorobenzene, Mist-o-matic, oxycarboxin, phenylmercury acetate, propiconazole, RH 5871, thiabendazole

Fuentes et al. (1983)
Methylmercury guanadine, pentachloronitrobenzene

Mitra (1935, 1937)
Phenylmercury acetate, ethylmercury chloride, charcoal formaldehyde, copper carbonate, Formalin, Hortisan A, sulfur, Uspulum

Rai and Singh (1979)
Triphenyltin acetate, Brestanol, triphenyltin hydroxide, oxycarboxin, Butrizol

Salazar-Huerta et al. (1986)
Chlorothalonil, Mancozeb, Carbendazim + Mancozeb

Singh et al. (1979)
Phenylmercury acetate, ethylmercury chloride, improved ethylmercury chloride

Smilanick et al. (1985a)
Chlorine dioxide, ethanol 40%, formaldehyde, sodium hypochlorite, hot water (54^{o}C), cupric acetate, Chloro-picrin, sulfur dioxide, methyl bromide

Smilanick et al. (1987)
Triadimefon, Bitertanol, benomyl, Campogran, thiabendazole, propiconazole, Trimidol

Valenzuela-Rodriguez and Navarro-Soto (1985)
Chlorothalonil (WP), Maneb, Carbendazim + Mancozeb
Carbendazim + Mancozeb, Maneb

Warham and Prescott (1989)
Triphenyltin hydroxide, methoxyethylmercury acetate, ethylmercury chloride

On the other hand, Aujla et al. (1986b) treated infected seed with 17 protectant and systemic fungicides and buried the seed in soil for 16 days. None of these fungicides inhibited germination of teliospores that were recovered from the buried seed.

Fungicides that had little or no effect on teliospore germination have been reported in other experiments. Examples are: ethyl-mercury chloride, oxycarboxin, 4-butyl-1,2,4-triazole, chlorothalonil, and propiconazole, Mancozeb, tridimefon, and carboxin.

In vitro teliospore germination tests, in the absence of seeds, provide little information directly related to KB control, but they might be useful in preliminary identification of chemicals for later study. Such screenings were made by Krishna and Singh (1983b) with Carbendazim, copper oxychloride, thiophanate, Mancozeb M45, Mancozeb 78, triphenyltin hydroxide, edifenphos, Dinocap, Iprobenfos, Thiram, and carboxin. All of these fungicides inhibited teliospore germination to some extent.

Foliar Treatments

A number of experiments with foliar applications of fungicides have been reported since the late 1970s and all have had positive results (**Table 3.3**). Some studies reported low disease incidence in the checks (Singh and Prasad 1980, Quiñones-Leyva 1984), suggesting that future work on foliar applications should be done in conjunction with inoculations to assure adequate levels of disease in the tests.

Soil Fumigation

Soil fumigation to control teliospore germination has been attempted with some success. In wet soil, methyl bromide reduced teliospore germination 98% when they were buried at depths up to 10 cm. Metam-sodium (Vapam) and formaldehyde were effective only on the soil surface. In dry soil, Vapam reduced germination 57-99% when spores were buried at depths up to 10 cm. Methyl bromide and formaldehyde were less effective (Smilanick and Prescott 1986). In other experiments, teliospore germination in samples buried at depths of 5 and 10 cm was 0, 0.2, and 2.4, and 0.6% in plots treated with methyl bromide and Dazomet, respectively, while in the untreated check soil, germination was 7-10% (Fuentes-Davila and Lawn 1992)

Combined Fungicide Application

During 1985 to 1987, the combination of soil fumigation with Brassicol (pentachloro-nitrobenzene), seed treatment with Bavistin (Carbendazim), and foliar sprays with propiconazole has shown a range of 50 to 80% control in experiments in India.

Table 3.3. Fungicides applied to foliage for KB control.

Reference/ fungicide	Percentage control
Krishna and Singh (1982b)	
Carboxin Carbendazim Oxycarboxin Triadimefon	82-87% control in greenhouse
Quiñones-Leyva (1984)	
Triadimenol Propiconazole	Reduced infection in field
Salazar-Huerta et al. (1986a), Salazar-Huerta and Prescott (1986, 1987)	
Propiconazole	93-98% control in field
Singh et al. (1985a)	
Carbendazim Fentin hydroxide Mancozeb	Reduced infection in field
Singh and Prasad (1980)	
Benomyl Carbendazim Mancozeb Triphenyltin hydroxide	Reduced infection in field
Singh and Singh (1985)	
Triadimefon Triadimenol	Reduced disease incidence in field
Singh et al. (1985b)	
Bitertanol	64% control in field
Smilanick et al. (1987)	
Copper hydroxide Etaconazole Mancozeb Propiconazole	80% control in field

Combinations of foliar sprays with seed treatment or with soil fumigation also reduce KB incidence (Gill et al. 1993).

Soil Drench

Applications of chlorothalonil (WP) and Mancozeb in irrigation water to 10 wheat cultivars did not significantly reduce KB incidence (Valenzuela-Rodriguez 1985).

Techniques for Study

Culture Techniques

Teliospores of *T. indica* can be isolated by making a fine hole in an intact sorus and dusting the spores onto water agar in a petri plate. Alternatively, a teliospore suspension may be prepared by shaking sori in water containing a surfactant. Then sieve the suspension to remove debris and centrifuge to pellet spores, re-suspend the spores in water containing sodium hypochlorite (0.5%) and centrifuge; rinse in sterile water and centrifuge (repeat this step 2-3 times to remove disinfectant).

Plate teliospores on 1.5% water agar and incubate at room temperature (20-22^{o}C) and light; germination begins in 7-10 days. At this stage, single sporidia can be isolated by micromanipulation. Masses of sporidia from single teliospores can be isolated or pieces of agar bearing the fungus can be transferred to potato dextrose agar slants or petri plates. After 4-6 days, add sterile water and scrape the cultures into additional PDA test tubes or plates.

Inoculation Techniques

Moore's vacuum method. Wet the spikes during anthesis with a teliospore or sporidial suspension. Insert the stems through a rubber stopper cut along the radius and fit the stopper to the inferior end of a cylinder connected by a hose to a water suspension of inoculum. Pull the inoculum into the cylinder by vacuum.

After spike exposure to the inoculum for about 1-2 minutes, release the vacuum by a valve located between the cylinder and the vacuum pump. The inoculum suspension returns to the repository by gravity (Moore 1936). A range of 11-100% infection has been obtained when inoculation is done with sporidial suspensions (Bedi et al. 1949, Chona et al. 1961). A number of factors may account for the range of infection, including: 1) viability of inoculum, 2) different cultivars, 3) varying environmental conditions, and 4) variation in efficient use of the inoculation technique.

Dropper method. Open the florets at anthesis and add a drop of sporidial suspension with an eye dropper. Chona et al. (1961) obtained 18 to 45% infection with this method.

Injection technique. Inject the inoculum with a hypodermic syringe into the boot just as awns emerge (**Figure 3.5**). High percentages of infection can be obtained with this technique (Chona et al. 1961, Durán and Cromarty 1977, Singh and Krishna 1982, Aujla et al. 1980). Mist-spraying inoculated plants also helps to obtain high percentages of infection (Aujla et al. 1982).

Go-go injection technique. Remove the central floret of an individual flower and clip off the awns with scissors. Then inject one or two drops of inoculum with a syringe into the remaining florets. This method gives satisfactory infection, but is more time consuming and less successful than the boot inoculation technique (Aujla et al. 1982, 1983).

Figure 3.5. Injection of *T. indica* inoculum into the boot of a wheat plant.

Other techniques that involve spraying spikes with an aqueous suspension of inoculum (Durán and Cromarty 1977) or applying sporidia with a small piece of cotton wool inside florets (Warham 1990) have given minimal infection.

Goates' inoculation method. Inoculate spikes inside an incubator. Lay pots and plants on their sides and place the spikes on fresh water agar in a plastic petri dish. Make a slit in the side of the petri dish to accommodate each stem. Then invert a petri plate containing the fungal colony over the spikes. Sporidia will shower from the culture onto the spikes. Inoculate the spikes in this fashion for 24-48 hours at 20°C under continuous light (Goates 1988). This method is more suitable to screen for morphological resistance than the boot inoculation technique and the percentage of infection is higher than with the boot technique (Salazar-Huerta et al. 1990).

Disease Scoring

Disease scoring has been based on the number of healthy and infected spikes (Mitra 1935, 1937; Mundkur 1943a; Bedi et al. 1949; Chona et al. 1961). However, this classification gives equal weight to the presence of one infected kernel per head and to many infected kernels per head. Today, disease scoring is primarily based on the percentage of infected kernels (Singh and Krishna 1982, Aujla et al. 1982, Fuentes-Davila and Rajaram 1994).

Lines are considered to be resistant when kernel infection percentage is below 5% in 10 inoculated spikes after several tests (Fuentes-Davila and Rajaram 1994). Also used is a rating scale that considers the size of the lesion in the kernel, the number of kernels in each category, and the total number of kernels (Aujla et al. 1989).

Loose Smut

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History

The first written account of the cereal smuts comes from Theophrastus (384-332 BC). Smut was known to the Romans, who named it Ustilago, which comes from the Latin word for burn. This term was later used in many languages as the common name for smut (e.g., carbon, carbone, sot, brand). Loose smut (LS) of wheat was illustrated in 1556 in Hieronymus Bock's Herbal (**Figure 4.1**), and an accurate symptomology is given in Fabricius' text of 1774.

The accurate illustrations and descriptions of symptoms contrast with early views on the possible causes of cereal LS. Among them were: a superabundance of sap that fermented and dried up and was thought to be favored by certain soils and weather; the wrath of the gods or acts of the devil; a curse from malevolent neighbors; ill-boding solar, lunar, or stellar positions; and spontaneous generation, perhaps through previously disarranged plant tissue. These causes were held to be valid until about 1800, when the true causes of the smut diseases of cereals began to be uncovered. By 1890, three distinct fungal species had been shown to cause the LSs of wheat, barley, and oat, and it was soon learned that wheat is infected via the ovary (Maddox 1896).

Figure 4.1. An illustration of loose smut from Hieronymus Bock's Herbal, published in 1556.

Distribution and Importance

Loose smut [*Ustilago tritici* (Persoon) Rostrup] of wheat occurs wherever cultivated wheat, *Triticum aestivum* and *T. turgidum*, is grown (Anon. 1982; **Figure 1.4**). Since the pathogen is seedborne, it will be spread from place to place by man; rarely is it disseminated over long distances through the air. *U. tritici* did not occur in the Americas, Australia, or South Africa before European settlers introduced it when they brought wheat seed with them. In North America, LS was present on wheat at least as early as 1832 (Hendry and Hansen 1934). Resistant cultivars impede, but do not prevent, dissemination. When breeding resistant cultivars, it is essential to test with races that occur in the area of adaptation of the cultivar, and to have resistance available to races with wider virulence from other parts of the world.

LS is never devastating, but causes low to moderate annual losses. It is more common in regions with a cool, moist climate during flowering of the host. Yet, even in dry warm climates, economic losses occur. Since percentage infection equals loss in yield, and since most of the monetary return from any one field goes into cost of production, even 1-2% infection can reduce profit to the farmer by 5-20%.

Hosts

Wheat species are the principal hosts of *U. tritici*, which has been found on all *Triticum* spp. except *T. timopheevi*. LS occurs on *Aegilops* spp. and is likely to have parasitized *Aegilops* before cultivated wheat evolved. Some races that are specialized on bread or durum wheats are pathogenic on some *Aegilops* spp., while most races on *Aegilops* spp. are not pathogenic on *Triticum* spp. (Nielsen 1985).

The LS fungus is pathogenic on rye (*Secale cereale* L.) and was at one time economically significant on this crop (Humphrey and Tapke 1925). Races that specialize on either bread or durum wheats can attack rye, but the level of infection is low, and in rye resistance is more frequent than in wheat. Cultivars of triticale (X *Triticosecale*) can be susceptible to any race that attacks the parental wheat or rye (Nielsen 1973).

In nature, *U. tritici* is rare on wild grasses other than *Aegilops* spp. There are two reports of natural occurrence on *Agropyron* spp. in North America, and one report each on *Elymus* and *Taeniatherum* spp. in Morocco (Nielsen 1978). *U. tritici* is also pathogenic on *Haynaldia*, *Hordeum*, and *Secale* spp. Within many of the wild species, there are both susceptible and resistant strains; specialization by the fungus is thus not restricted to cultivated wheat. Likewise, secondary symptoms, such as the incompatibility reaction of seedlings and shorter plants, are found within all host species.

Most wild hosts have no role in the epidemiology of wheat LS. The possible exceptions are races found on *Aegilops* spp., but only eight such races have been tested for virulence/avirulence on accessions of bread and durum wheat. Seven races were avirulent and one was virulent on certain bread and durum wheats (Nielsen 1985). The virulent race originated from *T. tauschii* (*Ae. squarrosa*) from an area of Iran where little wheat is grown. This supports the view of a phylogenetic relationship among races from *T. tauschii* and *T. aestivum* (Krivchenko 1984). Races on *T. tauschii* may harbor genes for virulence not yet found in races on *T. aestivum*. Likewise, races found on wild diploid and tetraploid relatives of *T. turgidum* may carry genes for virulence on durum wheat.

Pathogen

Taxonomy

LS of wheat is caused by the hetero-basidiomycetous fungus *Ustilago tritici* (Persoon) Rostrup. It is the only *Ustilago* sp. that occurs on wheat. A trinomial system to designate strains that are specialized on certain host species is impractical because of the multitude of possible *formae speciales* and the overlapping pathogenicity of each strain on several host species or genera (Nielsen 1985).

Because *U. nuda*, the cause of LS of barley, and *U. tritici* have identical spores and a similar biology, the two diseases have been claimed to be caused by the same organism. This taxonomic treatment, based solely on spore morphology, is untenable because it ignores the following important differences (Kellerman and Swingle 1889, Kim et al. 1984, Kanehira and Shinohara 1986, Vánky 1994):

- The morphology of the promycelium and of the monokaryotic and dikaryotic hyphae after germination is different in the two fungi.
- In *U. nuda*, all monokaryotic haploid isolates of the *MAT-2* mating type are proline auxotrophs, and those of the *MAT-1* mating type are temperature-sensitive; no such properties are found in the isolates of *U. tritici*
- The sori of *U. nuda* are initially covered by a thin membrane when the infected spike emerges, whereas those of *U. tritici* are naked (**Figure 4.2**).
- There are pronounced differences in polypeptides and isozymes between the two species, suggesting genetic differences.
- Only selected races of the two organisms can be hybridized, but the offspring are not viable (J. Nielsen, unpubl.).

These differences justify *U. nuda* and *U. tritici* as valid, though related species. *U. nuda* may have evolved from *U. tritici* (Nielsen 1987b).

Figure 4.2. Loose smut sporulating on spikes of bread wheat. Normal spike at left.

Physiologic Specialization

The study of races of *U. tritici* began about 1930 (Tiemann 1925, Piekenbrock 1927, Grevel 1930). Principles evolved from these early studies that are valid today:

- Resistance to each race is monofactorial and the genes can be readily transferred or combined, enabling planned breeding for resistance.
- The virulence pattern of a race does not depend on its geographic origin, but is determined by the cultivar on which it occurred.
- Cultivars select the races that are virulent on them.
- The same race may occur in different geographic areas.

Origin of races. Many races, T1 for example, occur in more than one country (**Table 4.1**). That some races seem to be restricted to one country is in all likelihood more apparent than real. Analysis of more collections on the standard set of differentials may show that the distribution of most races is not restricted because *U. tritici* is disseminated in seed by man. Further, new races of *U. tritici* appear to arise infrequently, spread slowly, and are found nearly exclusively on cultivated wheat.

A new race may arise by recombination of pre-existing virulence genes or by mutations at loci responsible for virulence. It is possible for new races to arise by recombination but this possibility is now greatly reduced because of widespread use of the same cultivars and of pedigreed seed. Further, LS is a monocyclic disease that has only a limited number of infection sites in any one field. Therefore, the role of new races has been greatly overrated. In fact, all past so-called “breakdowns” of resistance of wheat to LS can be

explained by existing races that escaped detection by race surveys that are necessarily restricted in geographic coverage and in the number of collections examined. Such so-called "new" races can become prominent after being exposed to hosts that select for them.

Differential cultivars. Races of the LS fungus are determined by inoculating a series of differential cultivars, and noting the incidence of sporulation in the next generation. This method is time-consuming and labor-intensive because of the biology of the pathogen. Methods that require less time are desired, as are single-gene differentials.

Several sets of differential cultivars have been used in different countries (Tiemann 1925, Piekenbrock 1927, Grevel 1930, Oort 1947, Bever 1953), based on reactions of local cultivars to local collections of the pathogen. These sets precluded any comparison with races detected elsewhere. This was partially alleviated when sets of differentials used in one country were also used in other countries, often after supplementing them with local cultivars. The set most widely used, and most modified, is that of Oort (1947). The original set of 10 cultivars or a supplemented version was employed in Great Britain (Batts 1955a), South Africa (Gorter 1964a), India (Rewal and Jhooty 1986a), and Canada (Cherewick 1953).

The present set of spring wheat differentials (Nielsen 1987a, Nielsen and Dyck 1988) contains 19 cultivars or lines (**Table 4.2**), of which only five were originally proposed by Oort (1947). The numbering of the differentials (TD = *U. tritici*Differential) is arbitrary; TD-1 to 11 were designated by Cherewick (1953), and TD-12 to 19 by J. Nielsen. Each of these differentials identifies either different combinations of known genes or a previously undetected gene for virulence. TD-13 is the first known universal suscept for all races of LS from *Triticum* spp., but it is resistant to races collected on *Aegilops* spp. (Nielsen 1985).

A new race is recognized if teliospores from a single spike of a field collection give a pattern of virulence that differs from any known race in at least three consecutive tests on the differentials, or if

Table 4.1. Occurrence of races of *U. tritici* worldwide (analyses of J. Nielsen to 1989 and P. Thomas to 1994).

Race designation	First identified in collection from	Later identified in collection from
T1	Canada	Afghanistan, Yugoslavia, Algeria, Kenya, Iran, Iraq, Ethiopia, Tunisia, Turkey, Poland, Germany, Australia, USSR, India, USA, Denmark, China, Pakistan, Nepal, Egypt, Mexico
T2	Canada	Sweden, Germany, Tunisia, Kenya, Denmark, South Africa, Brazil, Great Britain, China, USA, Turkey
T3	Canada	USSR, USA, Italy
T4	Canada	Algeria, USA, Italy, Mexico
T5	Canada	
T6	Sweden	Ireland, USSR
T7	Denmark	Canada, New Zealand, Australia
T8	Germany	Brazil, Poland, Australia, USSR, USA
T9	Czechoslovakia	Canada
T10	Canada	India, USA
T11	India	Ethiopia, Pakistan, Iran, Poland, Turkey, Canada, USA, Iraq, Nepal
T12	Argentina	Canada, USA, Uruguay
T13	USSR	
T14	Tunisia	Canada
T15	Canada	
T16	Canada	
T17	Canada	Poland, China, Egypt
T18	Canada	USSR, Turkey, USA, Uruguay
T19	Canada	
T20	Canada	
T21	Brazil	
T22	Brazil	
T23	Brazil	
T24	Brazil	
T25	Brazil	
T26	Turkey	Canada, USA
T27	Turkey	Poland
T28	USSR	Australia, China, Uruguay
T29	Poland	
T30	Australia	
T31	Poland	
T32	Canada	Italy
T33	Canada	
T34	USSR	China, Egypt*
T35	USSR	
T36	USSR	
T37	China	Turkey, Canada
T38	Israel	Egypt, Italy
T39	USA	Canada
T40	Reaction could not be confirmed	Deleted from collection
T41	China	
T42	Russia	
T43	Russia	
T44	Canada	

* Race T34 detected in Egypt by Dr. Abu El-Naga, Sakha.

Table 4.2. Lines or cultivars of spring wheat to differentiate races of *U. tritici*.

Differential designation	Cultivar, line or pedigree	CI or PI *	CN **
TD-1	Mindum		1795
TD-2	Renfrew	8194	1796
TD-3	Florence/Aurore	150111	1797
TD-4	Kota	5878	1798
TD-5A	Little Club/Reward		18129
TD-6	PI 69282	69282	
TD-7	Reward	8182	1801
TD-8A	Carma/Reward		18130
TD-9	Kearney	6585	1803
TD-10	Red Bobs	6255	1804
TD-11	Pentad	3322	1811
TD-12A	Thatcher/Regent//Reward		18131
TD-13	PI 298554/CI 7795		
TD-14	Sonop	227060	1814
TD-15	H44/Marquis	11782	1815
TD-16	Marroqui 588		1816
TD-17	Marquillo/Waratah		
TD-18	Manitou*2/Giza 144		1818
TD-19	Wakooma		1819

* CI = Cereal investigation number; PI = USDA plant introduction number.
** CN = Agriculture Canada number.

it is virulent on a line or cultivar that has been resistant to all known races (Nielsen 1983). The reactions of the differentials to 40 races that have been identified in collections from around the world are listed in **Table 4.3**. The countries from which each race was first and subsequently identified are given in Table 4.1 (Nielsen 1987a, Nielsen and Tikhomirov 1993).

Inheritance of virulence. There have been limited genetic studies since the development of techniques to isolate and culture monokaryotic haplonts (see page 46 in the "Techniques for Study" section; Nielsen 1972). To date, five genes for virulence have been identified (Nielsen 1977, 1982, unpubl.). Each is recessive, independently inherited, and not linked to the mating type locus. The five virulence genes and the cultivars they attack are shown below:

Virulence gene	Imparts virulence on cultivar
Utv1	Renfrew, Florence/Aurore, Red Bobs
Utv2	Kota, Little Club
Utv3	Carma
Utv4	Thatcher/Regent
Utv5	Sonop

Teliospore Germination and Culture of Isolates

The normal spore mass of *U. tritici* is dark olivaceous brown. Under the light microscope, single teliospores in water are light olivaceous brown, with one side lighter since the wall on this side is thin and contains less pigment. Dry, mature spores are raspberry shaped. In water they become globose to ovoid, with a diameter of 4-6 µm. The exospore is finely echinulate. Buff spore color has been reported only once (Campagna 1926). As in other *Ustilago* spp. that attack cereals, the normal dark color of wild-type *U. tritici* is likely controlled by one dominant gene that, when homozygous recessive for the buff allele, is epistatic to echinulation and results in smooth spores.

Teliospores do not exhibit dormancy. At spore germination, the single diploid nucleus of the spore divides meiotically, then mitotically, and a slender, slightly curved promycelium emerges on the lighter colored side of the spore. The promycelium consists of four cells, each with a single haploid nucleus. These cells may divide before producing a short hypha within 18 hours at 20°C, the optimum temperature for germination, in either light or darkness (**Figure 4.3**).

Ustilago tritici is heterothallic and bipolar. A haploid monokaryotic hypha, or a mycelium developed from it, can be either *MAT-1* or *MAT-2* mating type. This mating system is identical to that of *Ustilago* spp. parasitic on barley, oat, and several grasses; isolates of these several fungi are compatible with each other. Mating type is determined in a fusion test with two standards of known mating type, on a medium low in nutrients such as potato-dextrose agar at 1/10 normal

Table 4.3. Reaction of differentials to races of *U. tritici.*

	Differential (TD-)																		
Race (T-)	**1**	**2**	**3**	**4**	**5A**	**6**	**7**	**8A**	**9**	**10**	**11**	**12A**	**13**	**14**	**15**	**16**	**17**	**18**	**19**
1				S	S		S						S						
2		S	S			S	S	S		S			S						
3	S												S						
4	S										S		S						
5							S					S	S						
6				S									S	S					S
7				S	S		S						S		S				
8				S	S		S	S	S				S		S				
9			S	S	S		S			S			S		S	S			
10				S	S		S	S	S			S	S					S	
11							S						S						
12				S	S		S					S	S					S	
13			S	S	S	S	S	S	S	S	S		S		S	S			
14											S		S						S
15		S	S	S	S		S			S	S		S			S			
16							S	S					S						
17		S	S				S			S			S						
18				S	S		S	S	S				S						
19			S		S	S	S	S	S	S	S	S	S			S	S		
20			S	S	S		S			S	S		S			S			
21					S		S	S	S		S		S		S				
22			S	S	S	S	S	S	S	S	S		S			S			
23					S		S	S	S		S		S						
24*																			
25*																			
26													S						S
27				S									S						
28					S		S						S		S				
29				S	S		S						S						S
30					S		S	S	S				S		S				
31		S					S			S		S	S						
32	S												S						S
33	S										S		S						S
34					S		S						S						
35					S		S	S					S						
36*																			
37			S			S	S			S			S						
38			S		S		S			S			S			S			
39			S	S	S	S	S	S	S	S		S	S			S	S	S	
40**																			
41			S			S	S	S		S			S						
42													S						
43				S	S		S		S				S						
44				S	S		S					S	S		S			S	

* The following pairs of previously reported (Nielsen 1987a) races were combined under one designation since they differ only by symptoms of incompatibility on one or more differentials: T2/T25, T8/T24, T27/T36.

** The virulence pattern previously reported (Nielsen 1987a) for race T40 could not be repeated in subsequent tests; it was deleted from the collections.

concentration. Hyphae of opposite compatible mating type readily fuse to form a haploid dikaryotic hypha, which is slightly thicker than a monokaryotic hypha. In axenic culture, a dikaryotic cell usually soon divides into one dikaryotic and two monokaryotic cells, each of the latter of opposite mating type. A germinated spore will thus be surrounded by a mixture of monokaryotic and dikaryotic hyphae, both slightly curved.

Infection and Disease Cycle

Infection

Infection by *U. tritici* occurs only on the ovary. Spores enter the floret, germinate, and form dikaryotic hyphae that infect the ovary, usually at the brush end (Batts 1955b, Shinohara 1976). The process requires 5 to 7 days. Once in the testa, the hyphae grow intracellularly; but in the integument and nucellus, the fungus grows intercellularly, mainly on the dorsal side of the developing caryopsis. The mycelium enters the upper and side parts of the scutellum 10 to 15 days after penetration and grows through the hypocotyl into the plumular bud, or growing point, of the embryo (**Figure 4.4**). Thus, about 3 weeks are required for the fungus to reach the growing point, where it will lie dormant in the mature seed. Infected seeds do not differ outwardly from healthy seed.

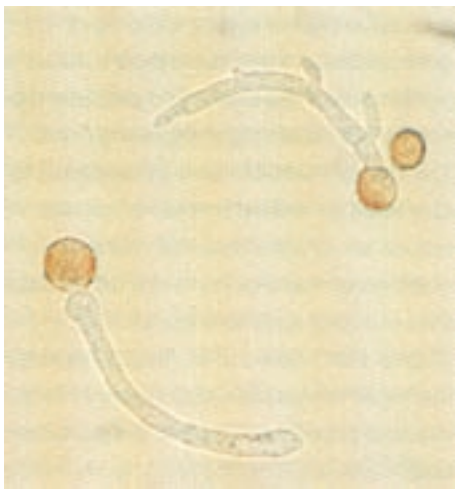

Figure 4.3. Teliospores of *U. tritici* germinating to form curved promycelia in which meiosis takes place (400x). The resulting haploid hyphae will subsequently fuse to produce dikaryons.

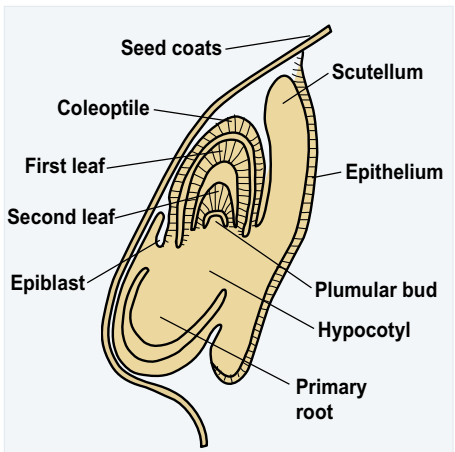

Figure 4.4. Longitudinal section of a wheat embryo.

When an infected seed germinates, the mycelium in the growing point is revitalized and carried in the crown node as the subcrown internode elongates. The fungus permeates crown tissues and enters the initials of the inflorescence. As the developing spike is carried upward by elongation of the internodes, the mycelium is also carried upward and proliferates in the developing spike (Batts and Jeater 1958a).

The dikaryotic nuclei fuse to form a diploid nucleus as the teliospores mature. At emergence of the spike, the teliospores are readily carried by the wind to nearby florets, where they germinate and cause infection, closing the life cycle of the fungus (**Figure 1.8**).

Symptoms

Masses of dark olivaceous brown spores are seen when the spike emerges from the boot with nearly all tissue of the spikelets affected (Figure 4.2). Only the rachis is intact, but it may be slightly shorter than the rachis of a healthy tiller. The dark spore mass can be seen through the wall of the boot several days before heading. Rain or heavy dew can cause the spores to cake into a black, hardened mass as does a severe drought in the late stages of spore formation. Hyperparasites, usually *Fusarium* spp., may cover and permeate the sori with whitish to pinkish mycelium. Yellow streaks appear on the flag leaf of certain genotypes (**Figure 4.5**). Narrow linear sori form only rarely on the flag leaf (**Figure 4.6**), the leaf sheath, or the peduncle (Klushnikova 1928, Batts and Jeater 1958a) under conditions such as those in the greenhouse. The mycelium of the LS fungus can be found in each node, but not the internodes, and is scattered in leaves of adult plants.

The physiological response of a susceptible host to infection includes increases in respiration; in catalase, peroxidase, and polyphenoloxidase

Figure 4.5. Yellow streaks on flag leaf of wheat cultivar Sonalika infected with *U. tritici*.

Figure 4.6. Linear sori of loose smut on the flag leaves of wheat plants, an unusual symptom of the disease.

activity; and in glucose and saccharose content (Stanescu 1961). The fungus produces trehalose, mannitol, and erythritol in liquid culture and in the host, but these compounds do not appear to be translocated. A reciprocal flow of carbohydrates between host and fungus is suggested (Gaunt and Manners 1971b, 1973).

In plants with sporulation, the number and dry weight of roots are reduced along with number, height, and dry weight of tillers. After heading, plants with sporulation stop growing. The lower internodes are usually longer and the upper ones shorter than in healthy plants, but the peduncle of the spike with sporulation is much shorter. The leaf-sheaths of some infected cultivars are grayish-purple; the leaves, particularly the flag leaf, are reduced in size, often yellowed and senesce early (Tingey and Tolman 1934, Mather and Hansing 1960, Gaunt and Manners 1971a, Gothwal 1972b). Under some environmental conditions, sporulation may be confined to the lower part of the spike.

Certain cultivars respond to infection by some races with hypersensitive (Oort 1944) or incompatible (Mantle 1961c) reactions. In this type of reaction, some seedlings die before emergence; others emerge but are stunted and have leaves that are brittle, often distorted, dark green, and with necrotic tips; such seedlings succumb easily to root rot and seedling blights. If a secondary tiller is produced, it usually appears to be normal and will bear a healthy spike. This tiller develops from the coleoptile bud, which has escaped invasion by the mycelium (Mantle 1961c). These symptoms are obvious in the greenhouse (**Figure 4.7**), but less so in the field, where most infected seedlings die before emergence. This resistance reaction will not be seen in commercial fields because no spores of the causative race are produced.

Association with Other Diseases

If a spike is partially infected, the upper florets can be infected by ergot (*Claviceps purpurea*), head blight (*Fusarium* spp.), Karnal bunt (*Tilletia indica*) or other floral infecting organisms. LS and common bunt (caused by *T. tritici* and *T. laevis*) may sporulate on the same spike, with sori of LS on the lower part of the spike clearly demarked from those of bunt in the upper florets. If seed that is infected with LS is inoculated with spores of common bunt, the interaction between the two organisms depends on the race of the pathogen and host cultivar. There can be complete compatibility, with both fungi on the same spike as just described, or infection by bunt is suppressed (Hanna 1938, Mantle 1962). Both flag smut (*Urocystis agropyri*) and LS have been observed on the same plant or culm (Aujla and Sharma 1977).

Influence of Environment

The environment influences development of LS soon after infection while the pathogen spreads through the embryo and also during growth of the infected plant in the next generation.

Because several days are required to penetrate the ovary wall, the first few days after inoculation are critical. The optimum for teliospore germination and further growth is 95% relative humidity and 20-25ºC (Danko and Michalikova 1969). Excessive heat or dry air will lower germination and germtube growth, delay penetration of the ovary, and preclude the fungus from reaching the growing point. The environment can also cause florets to stay open for a shorter time, which will reduce spore entry. Less LS has been noted on cultivars in warm and dry climates than in cooler and more humid ones (Tapke 1931, Atkins et al. 1963). It is likely that rapid dehydration of the embryo may inhibit or prevent infection of the plumular bud.

Seedlings developing from infected kernels are often weak and succumb sooner to adverse environmental conditions such as low soil temperature, drought, compacted soil, deep seeding, and waterlogging. Vernalization under normal conditions does not influence the level of sporulation in the next generation (Mantle 1961b). However, infected seedlings are more susceptible to severe frost (Tapke 1929), an effect that could suppress the disease in winter wheat

Figure 4.7. The short plants show the typical hypersensitive or incompatible reaction exhibited by some race/cultivar combinations. The tall plants are infected by a race that does not cause the reaction.

in areas where winter temperatures are already close to the climatic limit of the crop. It is likely that seedlots containing LS-infected kernels, treated with a fungicide to protect against soilborne diseases, will have a higher incidence of infection by LS than untreated seedlots. Soil fertility does not affect infection. Furthermore, lighter kernels have a higher incidence of infection than larger, heavier kernels (Taylor 1927, Niemann 1955). The reason for a higher incidence of infection in the smaller seed class is because the florets of the uppermost spikelets, and the third or fourth florets of the other spikelets, tend to produce smaller kernels; these florets also tend to open wider for a longer time, allowing more spores to enter and infect the embryo.

Adverse environmental conditions for wheat could lower LS incidence in the field because fewer infected seedlings survive. Those that survive may grow to maturity, but environmental conditions in later stages of wheat development can also influence infection. In one study (Kavanagh 1961b), infected plants were raised at one of three continuous temperatures. At 19°C, spikes with normal symptoms were produced; at 24°C, both normal spikes and spikes with sori only on glumes or in ovaries developed; at 29°C, there were no spores and the spikes were very slender. A week of continuous 29°C between internode elongation and boot stage almost completely inhibited spore formation. Cultivars differed in their response to the temperatures used (Kavanagh 1961b). It is conceivable that shorter or interrupted exposure to high temperature at certain growth stages may prevent or reduce sporulation in some plants.

In another study (Dean 1969), sporulation was maximum at continuous 23°C, less at continuous 20°C, and even less at 15°C. Seedlings kept at 6°C for 2 months and then at 15°C developed into plants that had only one third the level of infection observed at 23°C. Differences in response to temperature were observed between different race/cultivar combinations. Low temperature may reduce the growth rate of the fungus in the later stages of host development when growth rate of the spike increases. These experiments partly explain why late seeding of winter wheat and early seeding of spring wheat may reduce LS incidence (Freeman and Johnson 1909, Tiemann 1925).

Control Strategies

Physiologic Resistance

Resistance may be recognized by the absence of sporulation due to the following according to Batts and Jeater (1958a,b), Mantle (1961a,d), and Gaskin and Schafer (1962):

A) There are only a few penetrations of the ovary wall and little or no mycelium in the pericarp.

B) The mycelium is widely established in the pericarp, but there are few or no hyphae in the scutellum.

C) Hyphae permeate the scutellum, may be found in the hypocotyl, but are absent in the growing point.

D) When mycelium is found in scutellum, hypocotyl, and growing point, it indicates one of three interactions:
   I. a compatible host/ pathogen interaction that results in sporulation;
   II. an incompatible reaction as described earlier under “Symptoms” on page 36 (if a tiller is produced, it bears a healthy spike), or
   III. the crown node is infected, but the fungus rarely reaches the initials of the inflorescence and there is no sporulation.

Each of these interactions is specific for each race/cultivar combination and is determined by genotypes of both host and pathogen. Intermediate reactions are frequent and are affected by environmental conditions. The physiological principles that determine these interactions are unknown.

The resistance of a cultivar—if it is based on interactions A, B, or C in which the fungus does not reach the growing point—can be accurately assessed by the embryo or crown node tests. These tests, however, cannot distinguish between susceptibility based on interaction D-I and resistance based on interactions D-II or D-III. With these three interactions, there is mycelium in the embryo and crown node, making it necessary to plant inoculated seeds to determine whether or not there is sporulation on the adult plant (Gaskin and Schafer 1962, do Valle Ribeiro 1963a, Gorter 1964b).

Breeding for physiological resistance. Large differences in susceptibility are governed by the interaction between the genotypes of host and pathogen, and are thus specific for each cultivar/race combination. There is a correlation between the incidence of plants with sporulation in a cultivar and the proportion of tillers with sporulation on a single plant (Oort 1947, Batts and Jeater 1958a). Therefore, in moderately resistant cultivars, there is an additional factor that reduces spore production in the field, and with it, the chance and level of re-infection of the next generation.

Detection of sources of resistance. Resistance to LS has been demonstrated many times and remains the most economical and environmentally sound way to control the disease (Coons and Spragg 1918, Nielsen 1983). Unfortunately, most resistance has been detected with undefined field collections, which may have been extremely narrow in virulence, perhaps not even reflecting the virulence actually present in an area.

Breeders and pathologists must be aware of the races as they occur worldwide, but in particular the races of neighboring countries and countries with which cereals are traded. When searching for enduring resistance to LS, it is imperative that the lines or cultivars be tested with as wide a

spectrum as possible. An entry that passes such a screening has a good chance of being resistant to most races of the pathogen. Races with wider virulence should not be introduced for the sole purpose of testing in the field. Rather, their accidental introduction should be prevented by mandatory fungicidal treatment of all seed imports. If it is deemed necessary to use foreign races with wider virulence in evaluating resistance to LS, the tests should be made in controlled environments during seasons when wheat is not grown. When tests are completed, the test area should be sanitized, infected hosts should be autoclaved, and the pathogen should be destroyed or placed in safe storage.

The search for sources of resistance and their utilization in breeding programs is facilitated by use of high pressure spray or partial vacuum inoculation methods. The high pressure spray method (Moore and Munnecke 1954) is fast but uses large quantities of inoculum and may be less reliable than the partial vacuum method (Cherewick and Cunningham 1956, Nielsen 1983). However, the high pressure spray method enables one person to efficiently inoculate large numbers of entries.

For field tests, 10 to 12 seeds of each entry are sown in one hill. Ten hills are arranged 30 cm apart in a row. The spacing between rows ranges up to 90 cm to allow movement between two rows of hills. Blocks of several hundred hills are surrounded by a border of a resistant cultivar sown 30 cm away from the hills. Several highly susceptible checks should be included in each test to ascertain success of inoculation.

Since a single race rarely carries all genes for virulence that are needed to test for resistance, inoculum usually consists of a mixture of defined races. Each component of a mixture of up to three races should be present at a concentration of approximately 500 mg/L of water, an amount that represents the spores from one-half to all of a spike with sporulation. Since even a dilute inoculum will result in a high level of infection, there is no need to fine-adjust the concentration. An inoculum prepared in the afternoon and not used can be stored at 3-5^{o}C until the next morning.

A diverse collection of resistance sources of bread and durum wheats of spring habit (Nielsen 1983) were identified using most of the 40 races listed in Table 4.3 and the vacuum inoculation procedure. Since these races were collected from many countries, these resistance sources will likely be resistant in many countries. They are available on request from the Research Centre, Agriculture and Agri-Food Canada, Winnipeg, Manitoba, or from Plant Gene Resources of Canada, Agriculture and Agri-Food Canada, Ottawa, Ontario. In addition, many sources of resistance may be identified in winter wheats and in collections of diploid and tetraploid *Triticum* and *Aegilops* spp. (Nielsen 1985).

Inheritance of resistance. Krivchenko (1984) listed 52 genes for resistance to LS in 34 bread wheat cultivars or lines; some carried up to three genes. Most genes were dominant, 11 were recessive. Unfortunately, summation of resistance genes reported by different workers is likely to overestimate the number of genes. Future studies of the inheritance of resistance should be made with defined races and should include some hosts with known genotype.

Studies on inheritance of resistance are relatively easy if they involve a highly susceptible parent, a resistant parent that carries one or a few genes for resistance, and races with narrow virulence. When more than two genes are involved in the host/pathogen interaction, it is difficult to interpret results. The greatest difficulty lies in classifying the segregating progenies into homozygous resistant-heterozygous-homozygous susceptible classes to establish ratios for interpretation of the genetics. The susceptible parent should be used as a check at each step of the study to establish a base for the incidence of infection to be expected in the homozygous susceptible class. Without the susceptible check as a benchmark it is difficult to delimit this class. Some misclassification is unavoidable due to variability among tests.

New techniques are evolving. Resistance to race T19 was located on the short arm of chromosome 6A, using a monoclonal antibody in conjunction with an alien substitution line (Knox and Howes 1994). Work involving various DNA mapping techniques is in progress.

Present knowledge of genes for resistance to LS is summarized below (Tingey and Tolman 1934, Heyne and Hansing 1955, Gaskin and Shafer 1962, do Valle Ribeiro 1963b, Heinrich 1970, McIntosh 1983, Dhitaphichit et al. 1989):

- Resistance can be dominant or recessive.
- A gene can impart complete resistance (immunity) or partial resistance.
- Effects may be additive.
- Different genes may condition resistance to a single race.
- Resistance to one race can be conditioned by different, single genes.
- Genes may stop pathogen development at one or several specific sites in the ovary, embryo, or seedling.
- The genes of the embryo usually determine whether the fungus is arrested in the ovary or embryo or later, resulting in resistance.
- The maternal tissue of the ovary blocks, in some cases, further penetration by the fungus, even though the embryo is genetically susceptible.
- Genes can by located with monosomic and disomic chromosome substitution lines.

- Cytoplasmic host genes can affect expression of nuclear genes.
- Gene symbols have been proposed.
- There is no known linkage between a gene for resistance to LS and genes that condition reaction to leaf rust and mildew, awns, color of glume, lemma or kernel, or other morphological traits.

It is now clear that the gene-for-gene relationship postulated by Flor (1947) exists in the *Ustilago tritici*/*Triticum* pathogen/host system (Oort 1963). It has been proposed, on the basis of the gene-for-gene model, that 11 genes for virulence correspond to 11 dominant genes for resistance (Tikhomirov 1983).

Breeding methods . Breeding for resistance should emphasize genes for virulence rather than races. Since many cultivars already carry one or more genes for resistance, it is not an overwhelming task to incorporate additional gene(s) because there is no shortage of resistance sources.

Several approaches can be followed when breeding for resistance, depending on priorities and local conditions. If both parents are agronomically acceptable, screening for resistance to LS can start as late as F5 to F7, after other traits have been selected. Backcrosses are employed if resistance is to be transferred from an agronomically inferior source into an already established line or cultivar. With both the pedigree and backcross approaches, the two parents may differ in more than one gene for resistance. Inoculum should be a mixture of races that carries all genes for virulence against which resistance is desired.

Inoculation of small numbers of lines in early generations can be handled by syringe, but for later generations the partial vacuum method is the preferred method of inoculation. The reaction of entries is rated by the incidence of infection based on infection of two highly susceptible checks that are planted for about every 300 entries. If these checks have at least 80-90% infected spikes (as they should), the reaction classes are usually as follows:

Infection incidence	Reaction class	
0-10%	R	resistant
11-30%	MR	moderately resistant
31-50%	MS	moderately susceptible
51-70%	S	susceptible
over 70%	HS	highly susceptible

When selecting lines, it should be remembered that infection resulted from an amount of inoculum greater than encountered under natural conditions. Therefore, about 30% infection in repeated tests is probably adequate to indicate sufficient resistance under field conditions to prevent economic losses. Although immunity or high levels of resistance may be ideal for a cultivar, it is difficult to achieve, especially at the outset of a breeding program. Initially, whatever level of resistance is achievable should be used, thus preventing release of susceptible cultivars.

Morphological Resistance

Cultivars may be susceptible to LS when inoculated, but resistant, or nearly resistant, under field conditions (Tyler 1965; Nielsen, unpubl.). Freeman and Johnson (1909) expected that the number of spores entering florets would vary among cultivars because of differing floral opening habits. Tavcar (1934) reported that, in field-resistant cultivars, there are smaller lodicules and a smaller angle of opening for the palea and lemma during flowering than in susceptible cultivars. Cultivars also differ in the length of time florets stay open, in the proportion of florets that do not open at all, and in the extrusion of anthers (Ryzhei 1960, Parii 1973, Loria et al. 1982, Pandey and Gautam 1988). Breeding programs that screen wheat cultivars for resistance to LS by placing spores directly into florets may overlook morphological resistance conferred by factors like limited floret opening. At present, however, morphological resistance cannot be easily used in the improvement of cultivars because of the difficulties associated with selection. Currently, there is no method to quickly test large numbers of entries for both physiological and morphological resistance, which together determine the field reaction of a cultivar.

Morphological resistance, based on exclusion of spores from the site of infection, is effective against all races, and cannot be endangered by new virulence. Furthermore, most cultivars have varying, though low levels of physiological resistance, which in combination with morphological resistance will depress infection to an acceptable level. Therefore, one of the most urgent areas of study in relation to LS resistance is the search for an easily recognizable trait that conditions morphological resistance, or that is correlated with it.

Seed Treatment

Hot water, anaerobic, and similar treatments. One hundred years ago, LS of wheat could not be controlled except by the use of new seed. When hot water treatment (Jensen 1888) proved effective in controlling LS of oat and barley, it was applied to LS of wheat (Swingle 1892, Freeman and Johnson 1909). Numerous subsequent contributions detailed the conditions, particularly the time and temperature required, and all agreed that temperature had to be exactly controlled (Larose et al. 1946, Tapke 1926). Although large-scale seed treatment is possible, the hot water method is laborious and demands expertise.

Other attempts to control wheat LS involved the use of warm water, alone or in combination (Tyner 1953, Weibel 1958) with ethanol (Gassner and Kirchhoff 1938), or mercurial seed treatment fungicides (Rodenhiser and Stakman 1925, Bever 1961). In time, anaerobic

treatments evolved (Zalesskij 1935, Zemanek and Bartos 1964) in which wet seed was deprived of air for up to a week. Phytotoxicity, although lower than with the hot water method, was common with all of these treatments. The treatment advocated by Luthra (1953) used the sun as the source of heat: seed was soaked in water at ambient temperature for 4 hours, then spread in the sun for 4 hours. However, the usefulness of the method was limited. Microwave heating of seed (Szepessy 1969) to control the fungus has never been put to practical use. Today, all these procedures are mostly of historical interest (Niemann 1962).

Fungicidal seed treatment. Any efficacious fungicide must reach the mycelium inside the embryo or young seedling without harming the host. Early attempts at chemotherapy, including some work with antibiotics (Paulus and Starr 1951), were not successful. However, in 1966, the 1,4-oxathiin derivatives were reported to be active against *U. nuda* in barley (von Schmeling and Kulka 1966), and later proved to be effective against *U. tritici* in wheat (Hansing 1967). These compounds heralded a revolution in the control of LSs of wheat and barley. They are systemic, absorbed by plant tissue, and translocated to the site of action on the mycelium of the pathogen. Applied as wet or dry formulations, even distribution throughout the seed sample is important. The compounds, which dissolve in soil moisture and penetrate and spread throughout the hydrated embryo and the very young seedling, interfere with essential metabolic processes in the mycelium. Fungicides efficacious against LS include carbathiin, carboxin, benomyl, difenoconazole, etaconazole, ethyltrianol, flutriafol, furmecyclox, myclobutanil, nuarimol, and triadimenol.

Although a seedling test has been proposed (Verma et al. 1984) to evaluate the efficacy of fungicides, testing is usually done in the field at several locations with different climatic and soil conditions. Possible phytotoxicity can then be assessed. There have been no reports of *U. tritici* developing resistance to any fungicide.

Other Methods

Cleaning seed. The smaller kernels in a seedlot have a higher incidence of infection (Taylor 1927, Niemann 1955). Proper and rigorous cleaning will remove many of the small kernels and the crop will have less infection.

Roguing. Roguing infected plants as early as possible has been recommended, particularly in small isolated seed increase plots (Maddox 1896, Freeman and Johnson 1909). This approach may be practical only where small areas of wheat are grown.

Certification of seed. Seed certification schemes specify the incidence of LS infected plants at inspection of a field producing pedigreed seed. The tolerance level varies between 0.01% and 0.05% infection, with different levels for different classes of seed.

Test were developed to gauge the percentage of infected seeds in a seed lot. One early test was based on the difference in specific gravity of healthy vs infected kernels. When the kernels were placed in a chloroform/ethanol mixture, the infected kernels floated in the upper layer. Other tests have involved the influorescence of whole or dissected kernels (Yablokova 1939, Woestmann 1942). Seed certification can be based on the embryo or crown test to determine the incidence of infection in a seed lot (Skvortzov 1937, Yablokova 1939, Woestmann 1942, Simmonds 1946, Popp 1958, Khanzada et al. 1980). These laboratory procedures predict, in susceptible cultivars, the incidence of sporulating plants in fields sown with naturally infected seed under favorable environmental conditions (Batts and Jeater 1958a, Popp 1959). The various embryo and crown tests use staining of whole embryos, (Skvortzov 1937, Simmonds 1946, Popp 1958, Agarwal et al. 1981) or longitudinal sections, to detect the mycelium of the pathogen (Yablokova 1939, Woestmann 1942). Khanzada et al. (1980) detail a procedure for staining whole embryos.

The embryo and crown node tests are reliable if properly done, but they are time- and labor-intensive and costly. Also, working with NaOH and phenol is neither pleasant nor completely safe. For these reasons, the tests are unlikely to become a standard method for all classes of pedigreed seed. A simpler technique is needed to identify seedlots that are likely to produce crops with reduced yield.

Techniques for Study

Evaluation of Fungicide Efficacy

A minimum of 95% control of smut is needed to avoid the necessity of treating seed every year. Do not inoculate the seeds for fungicide tests; they should carry natural infection that is about the maximum that occurs in commercial fields in the area; 10% is an appropriate level. For efficacy tests, plots with single rows 4-5 m long and replicated four times are sufficient. Evaluate the tests by calculating the percentage of infected tillers after counting infected and healthy tillers. Ignore the slight variability due to cultivar interactions.

Determination of Incidence in the Field

In fields where infection appears to be less than 1%, make a 100-m loop through the field counting the smutted plants in a 1-m wide swath. Determine stand density and calculate percentage incidence. In fields with 1%, or greater, count smutted plants among 200 plants and calculate the percentage incidence.

Inoculum Preparation

For race determinations, inoculum is often prepared from bulked collections of spikes with sporulation from susceptible cultivars in commercial fields and could therefore be a mixture of races of the pathogen. If used for repeated testing of differentials, or if passed repeatedly through one cultivar (Roemer and Kamlah 1932), such inoculum could give the impression that virulence of the original "race" is not stable. This is likely the reason for earlier claims of instability of races (Cherewick 1958). To greatly reduce the possibility of working with mixtures of races, only teliospores from one spike should be used for race identification.

A suspension of teliospores in tap water has, over the years, proven to be the most reliable and practical inoculum. Addition of nutrients is unnecessary. The suspension is prepared by rubbing spores from an infected spike under water and straining the suspension through folded nylon screen (mesh size 250 μm) into a beaker. Spores suspended in water will stay viable for up to 5 days at 2 to 4°C, even if the temperature of the inoculum rises to 20°C when inoculating once a day. For longer storage, a viable inoculum should be put in a small polyethylene bottle and frozen at -15 to -18°C (Nielsen 1987a).

The optimal concentration of spores is 1 g/L water (Oort 1939, Rod 1958), but it is not necessary to adjust each suspension to this optimum. Even at 1/10 of this concentration, the level of infection is not significantly reduced. The inoculum may be adjusted to optimal concentration by visually comparing its color to a "standard" suspension prepared beforehand. The standard may be preserved for years with addition of a few drops of isopropanol before it is stored.

Inoculation Procedures

For maximum infection, inoculate the host at early to mid-anthesis growth stage (Freeman and Johnson 1909, Piekenbrock 1927, Stringfield 1929, Tapke 1929, Oort 1939, Batts 1955a, Rod 1958, Gothwal 1972a). The long time required by the mycelium to reach the growing point in the maturing embryo, together with the thickened cuticle and epidermis, explains why inoculations later than the optimal time give progressively lower infection levels. Because it is impractical to inoculate only those florets on a spike that are at the optimal stage, usually all florets are inoculated at one time. The proper stage is readily recognized on most cultivars by extrusion of the first anthers until the extruded anthers of the florets in the middle of the spike start to turn white. This period is described by growth stages 60-65 (Zadoks et al. 1974). Despite all precautions, inoculation with the same race and the same host in separate tests or years often results in considerably different levels of infection. These differences (even in a homogeneous host) are attributed to environmental factors at the time of inoculation or when growing plants from inoculated plants. Moreover, inoculation procedures cannot be completely standardized.

Greenhouse. A standard procedure for inoculation in the greenhouse (Nielsen 1987a) may be as follows. Inoculate two spikes for each race/differential combination. Just prior to inoculation, clip off the uppermost 1 cm of the spikes to identify those that are inoculated; it is unnecessary to clip awns and tips of the lemma, palea, and glume of individual florets. Placing a tag around the two stems identifies the inoculum, of which about 5 ml are needed. Inject the inoculum with a 5- or 10-ml syringe with a 22- or 24-gauge hypodermic needle that is 10 to 20 mm long. Hold the syringe and needle at a slight angle to the rachis (**Figure 4.8**); push the needle through the upper third of the soft palea until some resistance is felt when it reaches the tougher lemma. Then inject a drop of inoculum; this causes the hue of the lemma to change as the floret fills with inoculum. Begin injection at a floret on one side of the lowest spikelet and proceed upwards on this side. Then move to the florets on the other side of the spikelets still on the same side of the spike, and finally to the florets of the opposite row of spikelets. Inject the central floret in a spikelet if it appears vigorous enough to produce a kernel. If the plants are grown under artificial light, keep the inoculated spikes at least 20 cm away from incandescent or fluorescent lamps. Leave the inoculated spikes uncovered; putting them into small paper bags is of doubtful value (Tapke 1931, Atkins et al. 1963, Danko and Michalikova 1969, Gothwal 1972a, Loria et al. 1982). Growing out at least 30 plants is required to reliably assess the reaction of a differential or line.

Field. Most field inoculation methods have been developed to simulate natural conditions. Dry spores are introduced into the florets with a forceps or a small brush, with a puff of air over a tiny piece of paper or on a ball of cotton containing spores, or

Figure 4.8. The most reliable and consistent method for inoculating wheat with *U. tritici*. The ideal stage for inoculation is from first extrusion of anthers until the anthers in the middle of the spike start to turn white (illustrated above). The inoculum is injected into the floret after the needle penetrates the palea.

by dusting spores over entire spikes (Maddox 1896, Brefeld and Falck 1905, Freeman and Johnson 1909, Grevel 1930, Mishra and Jain 1968). To provide better access to the ovary, the tips of the florets are clipped. Partial vacuum or high pressure jet spray methods have been developed and are preferred for testing large numbers of entries in a breeding program where a single inoculum consists of a mixture of races (Moore and Munnecke 1954, Cherewick and Cunningham 1956, Nielsen 1983).

Go-go technique. This method, which has been commonly used at CIMMYT and other institutions, is an adaptation of the "go-go" pollination method. Remove the central floret of each spikelet and clip the glumes of the two remaining florets to expose the stigma and anthers. However, do not remove the anthers. Cover the spike with a paper bag, and, before anthesis is complete, use a smutted spike to "dust" teliospores onto the clipped spike. This technique requires a supply of smutted spikes and is unsuitable for inocula of more than one race. The simplest way to assure an adequate supply of spikes is to sow susceptible inoculated cultivars at the same time as the lines to be tested are sown. Staggered sowings will usually provide a range of headings, which will provide fresh inoculum. Infected spikes can also be picked and stored for varying time periods. This technique can also be used in the greenhouse. Inoculum may be blown into the bag containing the spike to be inoculated, if desired.

Air blast. Although this procedure gives good results, you must give strict attention to the operation or you risk injuring the plants or improperly inoculating them. At about mid-anthesis, tag 10 spikes and blow the inoculum into the florets with a jet of air (**Figure 4.9**). Direct the inoculum-laden air along each side of the floret to complete inoculation. The air jet is produced by a pressure pump that operates at about 20 psi. After inoculation, mist-spray the plants for an hour or so to assure maximum infection (Moore and Munnecke 1954).

Vacuum. At mid-anthesis, put the inoculum in a 1-L glass jar, which is placed in a wire basket that is permanently fixed to the lower end of the inoculating stand. Close the jar with a screw cap into which a short copper pipe has been soldered. Rubber tubing on one end of the pipe leads to the bottom of the jar, and from the other end to a copper pipe in a split rubber stopper (Cherewick and Popp 1950), which is inserted into the bottom of an inoculation cylinder. The inoculation cylinder (5 x 25 cm) is made of plexiglas, with a rubber stopper and copper pipe at its top. Seal the top of the pipe that is in the split rubber stopper and drill two holes in the side of the pipe. The side holes and the top prevent inoculum from squirting to the top of the chamber during evacuation. Insert a plexiglass cylinder (3 x 8 cm) in the vacuum line about 10 cm above the inoculation cylinder to keep inoculum from reaching the valve.

Mount the vacuum pump on a small sled or cart, which can be moved between blocks of hills during inoculation (**Figure 4.10a**). Use a rubber vacuum tube to connect the pump with the valve on the inoculating stand to allow free movement of the stand between rows of hills. Two inoculating stands can be connected to one pump to accommodate two operators. A 1/2 to 4 hp (0.4-3 kW) gasoline engine drives a rotary vacuum pump (Eberbach air-cooled Rotary Air Blast and Suction Pump). The pump maintains a vacuum of about 125 torr in a reservoir of about 6 L. Mount a vacuum gauge on the reservoir.

Figure 4.9. Air blast method of inoculation of wheat plants with teliospores of *U. tritici*.

Figure 4.10a. Vacuum inoculation of wheat with teliospores of *U. tritici*: equipment including the pump mounted on a cart; inoculum container (near operator's head); vacuum cylinder containing spikes (near his left hand), and catchment basin (near his right foot).

To prevent fine dirt from entering the pump (at shutdown), place a ceramic, automotive type filter over the exhaust. Allow droplets of inoculum to settle in a sturdy flask in line with the vacuum reservoir. In the line above the inoculating cylinder, fasten a three-way valve (push-button type, hydraulic, Maxam 703-131-810 A) to the inoculating stand. Select the spikes and insert them into the inoculation cylinder after clamping the split stopper over the stems (**Figures 4.10b-c**). The split stopper must make a good seal with the plexiglass cylinder. Press the valve to create an immediate and high vacuum in the cylinder—this draws inoculum into the cylinder; then pinch shut the flexible inlet hose and maintain the vacuum for 2-3 seconds (**Figure 4.10d**). To close the vacuum line and open the cylinder to the atmosphere, release the valve to the normal position. Repeat the evacuation and release, then unpinch the hose at the bottom of the cylinder, which allows the inoculum to drain back into the storage vessel.

Figure 4.10b. Vacuum inoculation of wheat with teliospores of *U. tritici*: arranging spikes with the split stopper.

With this procedure, two people can inoculate about 200 entries in a day. From each hill, one person selects two or three spikes that are at the appropriate stage and of about equal height and then loosely loops a short piece of brightly insulated copper wire just below the flag leaves to identify spikes to be inoculated and to hold them together. About 1 cm is clipped off the spike tips to mark the inoculated spikes (**Figure 4.10e**) for identification during harvest. The plants can also be marked with bright tags or bright plastic tape. The other person does the inoculating, marks the inoculated entries on a list and removes the wires.

Sow 30 to 40 seeds/entry from inoculated spikes in the greenhouse or sow all the seeds in the field. At heading, establish the percent of infected spikes by counting or estimating. Entries with over 30% infection are classed as susceptible and not tested again. Entries with less than 10% infection are classed as resistant. Since the environment at the time of inoculation and the process of inoculation cannot be standardized, entries identified as resistant in one test should be tested again. In the tests, susceptible cultivars should be inoculated to check the success of the procedure (**Figures 4.11a-b**).

The partial vacuum method should not be used in genetic experiments because there will be some transfer of pollen between lines or cultivars during inoculation that may result in some outcrossing. Depending on the reaction of the "donor" and the mode of inheritance of resistance, it may slightly raise or lower the incidence of infection. This effect can be ignored when the reaction of many diverse genotypes is assessed.

Inoculating differentials. Plant the differentials in hills with a spacing that allows easy access. In a greenhouse or growth cabinet, raise the plants in 15-cm pots with 10 to 12 plants/pot. Cut off all

Figure 4.10c. Vacuum inoculation of wheat with teliospores of *U. tritici*: insertion of spikes into the vacuum cylinder.

Figure 4.10d. Vacuum inoculation of wheat with teliospores of *U. tritici*: inoculation in progress.

Figure 4.10e. Vacuum inoculation of wheat with teliospores of *U. tritici*: spikes that have been tagged and clipped for inoculation.

secondary tillers near the soil level, beginning about 3 weeks after seeding. To have the plants in the green- house or growth cabinet at anthesis at about the same time, plant the differentials in a staggered manner.

In the field, sow inoculated seed in a 1.5- to 2-m row, and evaluate the LS reaction by counting healthy and infected spikes. In the greenhouse, plant 30-40 seeds of each race/differential combination and evaluate the reaction by counting healthy and infected plants. A differential with more than 10% sporulating plants is classed as susceptible; below 10% infection, it is classed as resistant. In practice, the reactions of most differentials either show a high incidence of infected plants, or none at all.

Collecting Field Isolates of Smut

Collect isolates from different cultivars and widely separated parts of a region. Collect single spikes soon after heading, before spores have been exposed to rain. Keep each spike in a paper envelope (not plastic!), and dry them by spreading the envelopes on a table at room temperature for about 14 days. Storing the spores in a desiccator at about 4°C will keep them viable for at least 5 years. If the spores from a single spike are not sufficient to inoculate all differentials, the spores can be increased on susceptible varieties.

Working with Races

Purification. Two compatible monokaryotic haploid lines can be isolated (Nielsen 1972) from one spore of each race, increased separately in liquid shake culture, mixed, and at anthesis injected into florets of a susceptible differential. The methods and media used are described later. Alternatively, spores can be germinated on a dilution-plate, and after 1 week the mycelial colony surrounding a single spore is transferred to an agar medium in a test tube. This mycelium, which is a mixture of mono- and dikaryotic hyphae, is then increased and injected as just described. Use the spores formed in the next generation to inoculate a susceptible differential to increase the race, and to inoculate the set of differentials to confirm the pattern of virulence shown by the original collection.

Preservation. Dry storage of spores was attempted, but early on Stakman (1913) found that spores kept at ambient temperatures lose their viability within a few months. Storage at lower temperatures greatly prolongs viability (Gera and Vashisth 1968). For routine storage, keep dry infected spikes in paper envelopes or glass jars at 2 to 4°C and the spores will be viable for about five years. Spores dehydrated and sealed under vacuum (Vanderwalle 1953) will remain viable much longer if stored in a refrigerator or freezer. For even longer preservation, the races can be kept as dormant mycelia in inoculated seed. Storing infected seed below 0°C had no effect on the level of infection in the crop grown from such seed (Buchheim 1935).

Long-term storage of races of *U. tritici* should be handled as follows (Nielsen 1987a). Inoculate a differential that is susceptible to the fewest races possible (other than the race to be stored). Next dry the infected, mature seed in a vial over silica-gel in a desiccator for one week at room temperature. Then place the vial into another desiccator with silica-gel and store it at about -15°C; regenerate the silica-gel periodically. When a race is needed, remove 20 seeds from storage and plant them to produce spores.

Figure 4.11a. Results of vacuum inoculation of wheat with *U. tritici* showing nearly 100% infection.

Figure 4.11b. Results of vacuum inoculation of wheat with *U. tritici* showing only a few infected spikes.

Determination of races. Various approaches have been used to determine susceptibility:

- Looking for differences in germination of spores in extracts from stigmata (Michalikova 1970), kernel extracts (Gupta et al. 1977, 1979; Nielsen 1981), or from susceptible or resistant cultivars;
- Differentiation of races by immunochemistry (Yamaleev et al. 1975) or by characterization of their polypeptides (Kim et al. 1984);
- Inoculation of very young seedlings (Kavanagh 1961a).

These approaches have limited value in that they are not applicable to all race/ cultivar combinations and inoculation and evaluation of disease is necessary for determining the races. A spore suspension can be injected into the florets using a rubber ball (Poehlmann 1945) or syringe with a hypodermic needle (Figure 4.8; Bever 1947, Rod 1958, do Valle Ribeiro 1963a, Danko and Michalikova 1968). The advantages of such injection are:

- The spores from one spike suffice to inoculate the differential set, which helps ensure purity of races;
- It is easy to change inocula since syringes and needles are readily sterilized in 70% alcohol or boiling water (for routine race-determination, repeated thorough rinsing of the syringes under running water is sufficient);
- No special precautions are required to avoid cross-contamination between spikes.

Crosses between races. In a typical study of the inheritance of virulence, isolate two monokaryotic haploid lines of compatible mating type from one germinating spore of a race avirulent on a certain cultivar; in a similar fashion isolate a pair of lines from a virulent race. Cross these four parental lines in all possible combinations on a common susceptible cultivar to give spores of two selfings and of two F1s. One tetrad, i.e., the four products of meiosis are now isolated from one germinating spore of each F1; cross the eight lines in all possible combinations with each other and with the parental lines. This results in 8 F1-selfings, 8 F1-intercrosses, 16 F1-backcrosses (8 to each parental race), and also 2 parental selfings or intraracial crosses, and two interracial crosses. These are injected into the florets of the cultivars on which virulence/avirulence is to be determined. The presence or absence of sporulation on plants grown from seed inoculated with these 36 crosses reveals whether virulence is recessive or dominant and the number of genes involved.

To do the crosses, transfer the mycelia of monokaryotic haploid cultures from stock cultures on solid medium into 125-ml Erlenmeyer flasks with 35 ml of a liquid medium made up of 2 g asparagine, 2 g yeast extract, 2 g malt extract, and 20 g glucose in 1000 ml water. Hyphal fragments appear in the liquid medium after about 8 days with continuous agitation at 20-22ºC. When a dense suspension has developed, inoculate additional flasks with 5 to 10 ml of this suspension. Use the cultures in these new flasks after 2 to 3 days on the shaker. Mix cultures to be crossed in a 50-ml beaker, using 20 to 25 ml of each culture, but if the concentration of the hyphal fragments is different, adjust the proportion so that cells of each line are present at about equal numbers. Leave the beakers standing for about 30 minutes to allow hyphal fragments to settle loosely at the bottom. When the liquid above the fragments is clear, draw it off with a narrow tube connected to an aspirator, leaving about 12-15 ml. Break up the larger pieces of mycelium in a Sorvall Omni Mixer in a 50-ml vessel. Run the mixer for 4 to 7 seconds at about half speed, depending on the size of the larger pieces of the mycelium, and on the volume. Inject the inoculum into the florets through a 20 gauge needle.

Obtaining Haploid Hyphae

Monokaryotic haploid hyphae can be isolated on special media and by microsurgery (Nielsen 1972). Spread dry teliospores with a fine brush on 1.5% water agar (1.5-2 mm thick) containing aspartic acid (0.147 mg/ml water). Incubate at 20ºC about 30 hours until dikaryons have been formed. Cut 1 x 1-cm squares from the medium and place them on a thick layer of potato dextrose agar at 1/5 the normal nutrient concentration (1/5 PDA) and place in the refrigerator over night. Transfer the squares to another thick layer of 1/5 PDA, pre-warmed to 25ºC, and keep at this temperature four to six hours. Isolate monokaryotic haploids by microsurgery, and transfer them to small slips of water agar 1.5-2 mm thick; place these on a thick layer of 1/5 PDA at 20ºC. Growth will be visible after 3-4 days.

Isolates will stay mycelial and grow on minimal media containing glucose and ammonium or nitrate (Sen and Munjal 1964). They can be maintained on PDA, potato sucrose agar, or potato malt agar. Growth is relatively slow and results in a dense, cream to pinkish-cream surface mycelium. Cultures stored at 3-5ºC may be transferred every 2-3 months. For special studies, all four products of meiosis can be isolated from one germinated teliospore. For intra- and interspecific crosses, the isolates can be increased in a liquid medium on a shaker. The cardinal temperatures for culturing the fungus on both solid and liquid media are 6-8/21-23/31-33ºC.

Most cultures on solid media turn dark gray to brown on aging. If the culture is dikaryotic, the cell walls thicken and the cells may assume a globular shape. These are resting spores or chlamydospores, but not true telio- or ustilospores, which are formed only on a host plant. The fungus cannot complete its life cycle in axenic culture.

CHAPTER 5

Flag Smut

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History

Flag smut (FS) on wheat was first reported in South Australia in 1868 (Anon. 1868). However, it had probably been there earlier because farmers referred to a "black rust" before this date (Miller and Millikan 1934a). In 1848, FS was reported on *Agropyron* sp. in Europe and was attributed to *Uredo agropyri* (Preuss 1848).

The Australian FS pathogen was identified (Wolff 1873) as *Urocystis occulta*, previously described on rye. However, it was later designated as *U. tritici* on the basis of spore morphology (Körnicke 1877).

The history of the disease shows that incidence and severity is dependent on the cultivars grown. For example, in the USA, the replacement of a commonly grown susceptible cultivar by resistant cultivars in Illinois reduced FS incidence to a negligible level (Fischer and Holton 1957). Further, in Australia, serious losses occurred when susceptible and very susceptible cultivars were widely grown at various periods, including 1915 to 1930 and 1949 to 1966. However, the disease declined when susceptible cultivars were replaced by resistant cultivars (Anon. 1970, Brown 1975, Platz and Rees 1980, Ballantyne 1993). Chemical seed treatment has also contributed to FS control.

Distribution and Importance

FS is present on all continents (**Figure 1.5**), although it has not been reported on wheat in the Scandinavian countries, Poland, or England (Anon. 1991). FS has been reported from Australia (Anon. 1868, Cobb 1891), Chile (Anon 1991), China (Miyake 1912), Egypt (El-Helaly 1948), India (Sydow and Butler 1906), Japan (Hori 1907), Pakistan (Anon. 1991), South Africa (Putterill 1920), Spain (Griffiths 1924), and the USA (Humphrey and Johnson 1919).

While FS has been a serious problem at times in all Australian wheat growing areas, it is now difficult to find (Ballantyne 1993; J.S. Brown, R. Loughman, R. Rees, and H. Wallwork, pers. comm.).

When FS was discovered on wheat in Illinois, USA, it was considered a sufficient threat for quarantine action (Coons 1919). Shortly afterwards, it was found in Missouri and Kansas, but has not been reported there since the early 1930s (Boewe 1960). FS was recorded on wheat in Oregon and Washington in 1940 (Holton 1941, Purdy and Holton 1963) and may be found on wheat at a low level in the Pacific Northwest at the present time. Purdy (1965) speculated that wheat FS may have originated in the USA from a grass-attacking form, since many grass species are known to be hosts for FS throughout the USA. It is not known on wheat elsewhere in the USA (R.F. Line, pers. comm.).

A specimen of FS was collected in the USA prior to 1879 on *Agropyron repens* (No. 293 in the Ellis and Everhart set of exsicatti named North American Fungi, quoted as Ell. and Ev. N. Amer. Fungi). It was used to describe *Urocystis occulta* var. *tritici* Ellis.

The origin of the disease in Australia is not known, but there is evidence that Australia exported the pathogen to some other countries. For example, FS spores were found on leaf fragments in Australian wheat received in California (Pollack 1945). Outbreaks occurred in Mexico in 1945 after the importation of wheat from Australia (Borlaug et al. 1946), in South Africa (Verwoerd 1929), and in Egypt (El-Helaly 1948), suggesting that the disease was imported from Australia.

Australian cultivars were extensively grown overseas earlier in the century. For example, the susceptible cultivars Federation and Baart were widely grown from 1910 until the 1930s in the Pacific Northwest of the USA (Callaghan and Millington 1956, Lupton and Derera 1981, Patterson and Allan 1981). Federation was also grown in India (Macindoe and Walkden Brown 1968) and a range of Australian cultivars were widely used in Chile and Peru (Wrigley and Rathjen 1981).

FS caused serious losses, sometimes in isolated areas, in China, India, Italy, Japan, and Pakistan early in the century (Purdy 1965). In Australia, it caused widespread and substantial losses, sometimes even total crop failure, at times when popular cultivars were susceptible (Brittlebank 1920, Carne 1924, Anon. 1960). In Pakistan, FS incidence was as high as 70% during 1975-76 (Khan et al. 1984).

Hosts

Wheat is the most commonly reported host, but certain grasses are also susceptible (**Table 5.1**). Since 1879, FS has been reported on a range of grasses in 13 widely separated states of the USA (Clinton 1904, Griffiths 1907, Garrett 1910, Standley 1916, Zundel 1933, Durán 1968). There were no apparent relationships between genome and FS reaction.

Rees and Platz (1973) reported naturally occurring FS on *Agropyron scabrum* var. *plurinerve* Vickery form A, a common grass in southern Queensland. Wheats inoculated with an *Agropyron* collection were infected, and an *Agropyron* inoculated with a wheat isolate was smutted. Rye grass (*Lolium* spp.) occasionally shows symptoms in southern New South Wales. No inoculation tests have been carried out, and the distribution does not suggest that rye grass is a source of inoculum for wheat (Ballantyne, unpublished observations).

Based on morphological examinations only, *Triticum* hosts include *T. aegilopoides, T. dicoccoides, T. dicoccum, T. orientale, T. palaestinum, T. persicum, T. polonicum*, and *T. timopheevi* (Purdy 1965).

Table 5.1. Susceptible (S) and resistant (R) reactions of grasses inoculated with flag smut from wheat.

Reference/ species	Reaction
McIntosh (1968)	
Diploid *Triticum*	
T. tauschii	S
T. bicorne	S/R
T. comosum (Richter acc.)	S
T. species	R
Tetraploid *Triticum*	
T. turgidum	R/S
Polyploid *Triticum*	
T. ventricosum	S
T. triumcale	S
T. columnare	S
T. ovatum	S
T. crassum	S
T. juvenale	S
T. cylindricum	R
T. kotchyi	R
T. triaristalum	R
Fischer and Holton (1943)	
Agropyron caninum	S
A. dosystachum	S
A. desertorum	S
A. repens	S
A. semicostatum	S
A. spicatum	S
A. trachycaulum	S
Elymus canadensis	S
E. glaucus	S
E. triticoides	S
Hordum jubatum var. *caespitosum*	S
Purdy (1965)	
Aegilops squarrosa	S
Agropyron elongatum	S
Griffiths (1924) and Tisdale et al. (1927)	
Triticum spelta	S/R
T. turgidum	S/R

S/R = some accessions S, others R.

Pathogen

Taxonomy

Fischer (1953) regards the wheat- and grass-attacking forms of the FS pathogen as the same species. If this is valid, the name is *Urocystis agropyri* (Preuss) Schröter. However, Vanky (1985) and other European mycologists consider the FS fungus on wheat to be distinct from the one on grasses. If this is the case, the name is *Urocystis tritici* Körnicke (Körniche 1877). The extended synonymy is in Fischer (1953).

Urocystis occulta, the FS pathogen of rye (*Secale cereale*), has often been confused with *U. agropyri*, although the two species differ in spore ball morphology. In *U. agropyri*, the dark spores are covered by an almost complete layer of hyaline to tinted sterile cells, but this layer of cells is incomplete or absent in *U. occulta* (Mordue and Ainsworth 1984).

Other smuts commonly associated with FS-like symptoms on a wide range of grass hosts are *Ustilago striiformis* and *U. macrospora* (Mordue and Waller 1981b). These two smuts are distinguished from *U. agropyri* by single spores rather than spore balls (Mordue and Waller 1981a).

Teliospores and Spore Balls

Spore balls occur in dark stripes in leaves or occasionally in the inflorescence. They are dusty, dark brown to black. The spore ball consists of up to six (usually three) teliospores (ustilospores) (Hawkesworth et al. 1983) that are hyaline or lightly tinted and surrounded by flattened, sterile peripheral cells (**Figure 5.1**). Teliospores are globose to subglobose, reddish to olivaceous, 8-18 µm in diameter; spore balls are 18-52 µm in diameter (Langdon et al. 1976, Mordue and Waller 1981a).

Longevity. Teliospores survive in soil for 4 to 7 years (McDairmid 1912, Verwoerd 1929, Sattar and Hafiz 1952a) and in the laboratory, when stored at low humidity at 13-31ºC, for at least 10 years (Noble 1934). Spores are viable after passing through the digestive systems of horses and cattle (McAlpine 1910, Clayton 1925, Verwoerd 1929).

Germination. Teliospores of *U. agropyri* germinate capriciously (McAlpine 1910, Griffiths 1924, Verwoerd 1929). Those collected immediately after maturity do not germinate until after 42-82 days (Verwoerd 1929). Noble (1923) broke the dormancy by drying for 48 hours at room temperature over concentrated sulfuric acid.

A wide range of plant extracts, organic chemicals and surface active agents promoted germination especially of aged presoaked teliospores (Noble 1923, 1934; Griffiths 1924;Verwoerd 1929; Allan and Durán 1979; Goel and Jhooty 1987). The delay in germination may have survival value.

The teliospore germinates to produce a bulbous or club-shaped promycelium (basidium) that becomes cylindrical (**Figure 5.2a**). The promycelium is septate or nonseptate, 23-29 x 4.5 µm in size, with 2-5 protuberances forming on its tip and

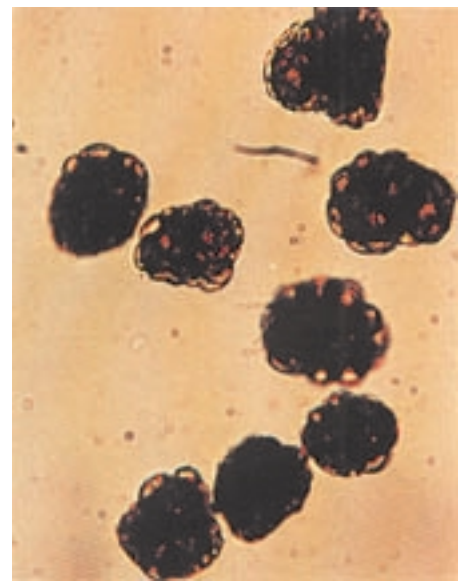

Figure 5.1. Spore balls of *U. agropyri* showing dark pigmented teliospore areas and sterile peripheral cells.

developing into sporidia (basidiospores). The young sporidia are born in clusters, but at maturity they are distinct, fingerlike projections (**Figure 5.2b**), oblong, translucent, and smooth, and 25 x 4.5 µm in size. A septum may develop between the sporidium and the germinal tube (**Figures 5.2d**,**e**; Griffiths 1924, Noble 1924, Verwoerd 1929, El-Khadem et al. 1980, Nelson and Durán 1984, Goel and Jhooty 1986). When mature, sporidia produce a germinal tube or infection thread (**Figure 5.2f**) up to 250 µm long and always septate.

Cytology of germination. Meiosis occurs in the teliospore followed by mitosis. Haploid nuclei migrate to elongated basidiospores, generally one nucleus per spore, but sometimes two. Plasmogamy occurs *in situ* between mononucleate basidiospores and the nucleus of one basidiospore migrates into the protoplast of the other. Usually plasmogamy occurs between two hyphal pegs, one at the base of each basidiospore, although apical fusions have also been seen. Some dikaryons also form fusions between promycelia and basidiospores or by migration of two nuclei into single basidiospores or from the promycelium itself when teliospores germinate directly.

Cultures derived from unbroken smutted tissue are slow growing, but they grow well on different media (Wu 1949). Isolates differ in cultural characteristics that persist after transfer. A typical 60-day-old culture on potato dextrose agar was 26 mm in diameter, white to dark olive or buff in color, raised umbonate in topography, coriaceous and mycelioid in consistency, velvety surface with zonation, lacerate, lobate or entire and mycelioid, appressed on the margin. The tough mycelial growth produced few if any sporidia (Wu 1949).

Mating behavior. Mating is bipolar with multiple alleles controlling heterothallism (El-Khadem et al. 1980). However, secondary homothallism was occasionally suggested when basidiospores received two nuclei (Nelson and Durán 1984) .

Physiologic Specialization

Physiologic specialization has been reported, but does not appear to have been a problem in breeding programs. Some of the tester wheats susceptible to certain Chinese, Pakistani, and Indian collections are resistant to collections from other regions such as Australia and the USA. It is possible that local cultivars have exerted selection pressure over a long period of time in these Asian countries. This raises the possibility that the center of origin of the pathogen lies in Asia. There are suggestions that some subsequent spread occurred in wheat exported from Australia early in the century.

Races. The FS pathogen has shown less physiologic specialization than any of the cereal smuts. The variation that has occurred has not been a problem in breeding, except possibly in China and India.

Yu et al. (1936, 1945) demonstrated physiologic races in collections from China using three infection classes: 0-5% infection = resistance, 5.1-20% infection = intermediate, and 20.1-100% infection = susceptible. The reactions of tester wheats to 12 races of the FS pathogen are shown in **Table 5.2**.

Holton and Johnson (1943) designated a collection from Kansas in central USA as race 1 and another, able to overcome the resistance of Oro/Federation selections 38 and 40, from Washington State in the Pacific Northwest, as race 2.

Further variation was reported with collections from Australia, Chile, China Cyprus, India, Pakistan, South Africa, and the USA (Hafiz 1951, and Johnson 1959). These data have been rearranged and are presented in **Tables 5.3** and **5.4**, respectively.

Host accessions. Of the five differential tester wheats chosen by Yu et al. (1936, 1945) in Table 5.2, TH 19323, Ngochen, Grassland, and TH559 were all commercial cultivars and Nanking 716 had been bred for FS resistance.

The groupings in Table 5.4 indicate similarities of reaction in Johnson's tests. For example, Ghurka 616, Rex, and N.P. 4 were similar in their resistant reactions to most collections. The exceptions were the intermediate responses with some

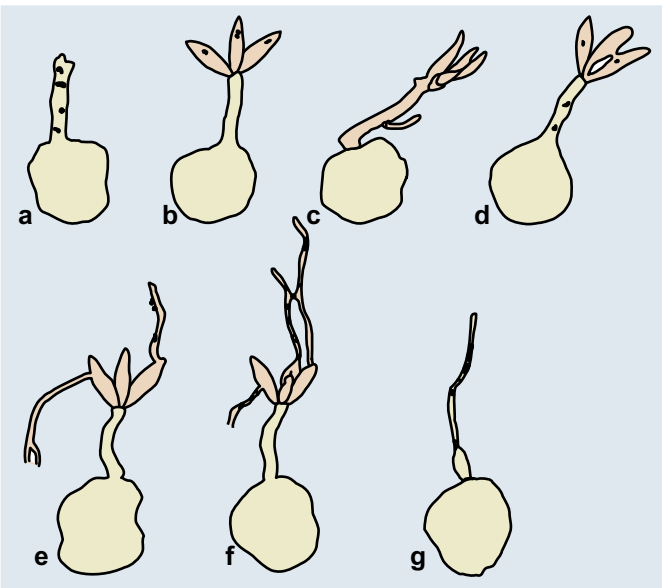

Figure 5.2. Diagrams of germinating teliospores of *U. agropyri* contained in a spore ball showing various stages of germination, sporidia formation, and infection hyphae: a) promycelium and nuclei (x1000); b) sporidia with nuclei on promycelium (x1200); c) lateral sporidium and secondary sporidia (x1000); d) fusion of sporidia with nuclei (x1000); e) infection hyphae from sporidia (x1000); f) fusion of infection hyphae (x1200); g) infection hypha from promycelium (x800) (from Noble 1923 and Verwoerd 1929).

Table 5.2. Reactions of 5 testers to 12 physiologic races of flag smut pathogen in China.

Tester wheats	Physiologic race											
	1	2	3	4	5	6	7	8	9	10	11	12
TH 1932	R	S	R	R	I	R	R	I	R	R	I	R
Nanking 716	R	R	R	I	I	R	R	R	R	R	R	R
Ngochen	I	R	I	I	I	R	S	I	R	I	R	R
Grassland	R	R	I	I	I	R	R	R	I	S	S	R
TH 559*	R	R	R	R	R	R	R	R	R	R	R	I

* Poulard wheat (*T. turgidum*).

Table 5.3. Reactions of seven wheats to 11 collections from 6 countries (Hafiz 1951).

Tester wheats	Collections										
	1	2	3	4	5	6	7	8	9	10	11
	Groups										
	1	2					3			4	
Federation	S	S	S	S	S	I	I	I	I	I	I
Oro/Federation-1	R	S	S	S	S	I	I	I	I	I	I
Oro/Federation-36	-	-	I	-	I	I	I	I	I	I	I
Oro/Federation-38	I	S	S	S	S	I	I	I	I	I	I
Unknown Chinese Wheat	I	S	S	S	S	I	I	I	I	I	I
Punjab T90	I	I	I	I	R	I	I	I	I	S	S
NP.80.5	I	R	I	I	I	I	I	I	I	I	I

collections of group 3. Nanking 716, Grassland, and Ngochen were resistant to most collections, but were susceptible to Chinese isolate C10. The Indian cultivars were tested with a more limited group of collections and reacted differentially. Most were susceptible to the Indian isolates, but gave intermediate or resistant responses in comparison with Chinese wheats and Baart.

Oro/Federation selections 38 and 40 reacted similarly to all pathogen collections with the exception of the Chinese smut collection, C4. Oro/ Federation-38 and 40 are the differentials for distinguishing Johnson's races 1 and 2. Oro/Federation-1 was clearly different from other selections, being susceptible to race 1. Oro, a Turkish selection, was used as a source of bunt resistance in early breeding in Washington State. The

Table 5.4. Reactions of selected tester wheats with flag smut collections from USA, China, Australia, India, Chile, South Africa, and Japan. From Johnson, 1959.

Tester wheats		Groups																	
		i						ii		iii					iv	v	vi	vii	viii
Collections		1'	C1	C2	C8	C11	C12	C4	C9	2	3	7	C7	4-6	8	9	C3	C6	C10
Accessions resistant to most cultures																			
Ghurka 616	U *	R	R-	R-	R-	R-	R-	R	R-	I-	I	R-	R-	I	R-	R-	R	R-	R-
Rex	U	R-	R-	R-	R-	R	R	R-	R-	R-	R-	I	R-	I	-	-	R-	R-	R
N.P. 4	I	R-								R		R		I-	R	R-			
Differentials																			
Nanking 716	C	R-	R-	R	R-	R-	R-	R	R-	I-	I	R	R	I	R-	R	R	R+	S
Grassland	C	R-	R-	R-	R-	R-	R-	I	R	R-	R-	R	R	R	R	I	I-	S	S
N gochen	C	R-	R-	R-	I	R	R	I	I	I	I	I	I	S	I	R	I	I+	S
N.P. 80.5	I	R-								R	-	R		S	R-	R-			
V-184	I	R-								R+	-	R-		R	R-	S			
N.P. 101	I	R-								R-	-	R-		R	R	S+			
Hindi D	I	R								S	-	S		I	R-	S+			
Punjab 8A	I	R-								I	-	R		I	S	S			
N.P. 12	I	R-								I	-	I		S	S-	S-			
Hindi 62	I	R								S+	-	S+		S+	I	S+			
N.P. 165	I	R-								S	-	S		S	S	S			
Baart	U	I+	R	R	I	I	R	S	S	I+	I+	S	S	S	R	I	S	I	I
Oro/Federation - 38	U	R-	R-	R-	R-	R-	R-	R	R-	S	S	S+	S	S+	R-	I	I	I	S
Oro/Federation - 40	U	R-	-	-	R-	-	-	I	R-	S+	S	S+	S+	S+	R-	R-	I-	I	S
Accessions susceptible to most cultures																			
Oro/Federation - 1	U	S	S+	S+	S	I-	S-	S	S+	S	S	S	S	S+	S	R-	S	S+	S
Spring Early	S	I+	-	-	I+	-	-	S	S	S	S	S	S	S	I+	R-	S	S	S
T.H. 3929	C	S	S	S	S+	S	S	S+	S	S	S+	S	S	S+	S	R+	S	S+	S
Kanred/Hard Federation	U	S	-	-	S	-	-	S	S+	S	S	S	S	S+	S	R+	S	S+	S

Most accessions of similar reaction and pathogen collections of similar reaction are grouped together.

* U = USA
I = Indian
C = Chinese
S = From South africa

' 1 & 2 U.S.
3 Chile
4 - 6 Australia
7 South Africa
8 Japan
9 India

C 1-12 China

Ghurka = Gallipoli/Currawa//Indian 4E/Federation
Rex = White Odessa/Hard Federation
Baart = Early Baart
Oro = Selection from Turkey

Chinese wheat TH3929 and Kanred/Hard Federation were almost identical in response, being susceptible to all collections except No. 9 from India.

Seed of the tester wheats (Table 5.4) is in the Australian Winter Cereals Collection, Tamworth, NSW, Australia.

Pathogen collections. The collections of Hafiz (1951) (Table 5.3) fall clearly into four groups, with group 1 from the USA as race 1, group 2 from Australia and Italy the most complex resembling USA race 2. Group 3 from China and Cyprus gave no disease on these testers, even though the spores were viable. Group 4 from Pakistan was distinctly different and simpler than the others.

Johnson's data (Table 5.4) formed eight groups. Group i, similar to US race 1 from USA and China, gave a susceptible reaction only on Oro/Federation 1. Group ii of two Chinese collections was similar to race 1 except for giving susceptible reactions on Baart and Spring Early. Collections in Group iii, made up of three from Australia and one each from China, Chile, South Africa and USA, were all similar to US race 2. Johnson (1959) designated the Australian isolates as race 2A because they gave more disease on Ngochen, NP80.5, N.P. 12, and Baart. Similarly, the South African collection also gave higher scores on certain testers and was designated race 2B. Verwoerd (1929) and Johnson (1959) also reported that Australian and South African collections produced more disease than the US smut. There is a report (Gorlenko 1946 as quoted by Dickson 1956) of an Azerbaijan collection similar to race 2.

Group iv from Japan was distinct in being able to overcome the resistance of some of the Indian testers and was designated as race 3. The one collection from India (Group v), designated as race 4, caused disease on an even wider range of Indian testers. Groups vi, vii, and viii—each with one collection from China—were screened on an incomplete set, but appeared to be different from each other and other groups. Group viii (collection C10) was clearly different and gave susceptible reactions on three Chinese cultivars resistant to most other collections. Johnson did not name these Chinese smuts.

Khan et al. (1984), working with Pakistan collections and using a different and limited range of tester wheats, reported that their race 1 (designations of Yu et al. 1945) was similar to Johnson's race 1, but their race 4 resembled Johnson's race 2A.

A three-year survey showed no variation in Australian collections (Watson 1958). This was despite the fact that FS was reportedly more destructive and widespread over longer periods this century in Australia than in any other country.

R.F. Line (pers. comm.) has not detected any race differences in the USA Pacific Northwest.

The greatest variation has been in the pathogen collected from China and India. Certain Chinese and Indian collections have overcome resistance of cultivars from China and India, but these genotypes were resistant to collections from other geographic regions (Yu et al. 1945). This suggests that FS may have originated in the regions of China and India. However, the sample size for all these studies is relatively small so such comments are speculative.

Studies on variation in this pathogen are resource-intensive because of the need to grow relatively large wheat populations to maturity and to have uniformity of inoculum and favorable environmental conditions at the time of germination and infection.

Collections from grasses. Inoculation tests with *U. occulta* from rye and *U. agropyri* from wheat demonstrated that, while *U. occulta* did not cause symptoms on wheat and *U. agropyri* gave no infection of rye, certain grass accessions supported both *U. occulta* and *U. agropyri* (Fischer and Holton 1943). Their Pacific Northwest wheat FS collection caused disease on *Agropyron caninum* and *A. spicatum*, which were resistant to the collection from the USA Midwest.

Infection and Disease cycle

Infection Process

FS infects seedlings before they emerge from the soil. See life cycle in **Figure 1.9**. Spores carried on the seed or present in soil provide the inoculum. The apical cells of dikaryotic infection hyphae form appressoria with penetration pegs that enter directly through the epidermis (Wolff 1873, Noble 1924, Verwoerd 1929, Emmett and Parbery 1975, Nelson and Duran 1984). Invasion of the host occurs only through the young coleoptile before the first leaf emerges (Jarrett 1932). The FS fungus infects both resistant and susceptible plants, but symptoms develop only in susceptible genotypes.

After infection, the fungus grows both inter- and intra-cellularly until sporulation. The mycelium ramifies through almost all host parts. It is unbranched in the internodal tissues and branched in nodal regions. The host tissues are often so heavily invaded by mycelium that cells are forced apart. Sporulation occurs between the epidermal layers and the vascular tissues of the plant (Noble 1924, Verwoerd 1929).

High levels of inoculum are reported to produce a higher incidence of infection than lower levels (Miller and Millikan 1934a, Millikan and Sims 1937, El-Helaly 1948).

Dissemination

Spore balls produced on diseased leaves dislodge during harvest and fall to the ground. A soot-like dust is often visible behind harvesters in heavily infected wheat. This attaches to seed, becomes incorporated into the soil, and may be blown by wind to adjacent fields (Miller and Millikan 1934a). Spore balls may also be disseminated in animal manure (McAlpine 1910, Clayton 1925), on the hooves of animals (Putterill 1920), in irrigation water (Putterill 1920), and in straw used for packing (Minz 1943).

Figure 5.3. Flag smut of wheat caused by *U. agropyri* showing twisted foliage and dark linear sori.

Figure 5.4. Flag smut of wheat caused by *U. agropyri* showing white striations, upright growth, and dark linear sori.

Symptoms and Other Effects

Infected seedlings are characteristically twisted and bent (**Figure 5.3**; Angell 1934a,b). Raised white areas with a blistered or vesicular appearance ("leprous spots") may develop on coleoptiles of some susceptible cultivars (Churchward 1934, McIntosh 1968).

Miller and Millikan (1934a) noted that, before typical symptoms are seen, infected plants may be distinguished by a large number of thin, stunted, wilted, and yellowish green leaves. In the field, symptoms may develop any time after the third or fourth leaf. However, the disease is more apparent after spikes form and is most obvious at the end of the season on late tillers.

On older leaves, first symptoms appear as white striations, which occasionally extend into the inflorescence (**Figure 5.4**). The striations change from white through shades of gray to black. Infected plants produce increased numbers of stunted, twisted, and distorted tillers and may not develop spikes. Infected plants also produce less developed roots (Angell et al. 1938a,b), fewer fertile spikes and seeds per spike, lighter seeds (Brown 1975, Bhatnagar et al. 1978), and poorer germination of seeds (Pal and Mundkur 1941).

Cultivars vary widely in response to FS. Some genotypes may be severely stunted and have blackened tillers and few if any spikes (**Figure 5.5**), whereas others may show only the occasional sign of infection. Some plants with a degree of resistance may have symptoms only on late tillers. Subterranean sporulation has been observed on *Phleum alpinum* and *Deschampsia caespitosa* (Durán 1968).

Association with Other Diseases

Urocystis agropyri, *Fusarium culmorum*, and other *Fusarium* spp. may simultaneously occur in the same wheat plant infected with FS. Seedling blight caused by the *Fusarium* spp. was more prevalent

Figure 5.5. Flag smut of wheat caused by *U. agropyri* showing stunted plant, tillering, necrotic lower leaves, yellow striations on upper leaves, and dark linear sori.

when FS was present (Geach 1933, Angell 1934a). Other pathogens that have been observed in plants infected with *U. agropyri* include: *Tilletia caries* (McAlpine 1905), *T. controversa* (Holton and Jackson 1951), *Puccinia striiformis* (Purdy and Holton 1963), and *Ustilago tritici* (Aujla and Sharma 1977).

Influence of Environment

Soil type, soil moisture, and temperature affect the incidence of the disease mainly by influencing the seedling germination and emergence rates. The seedling is susceptible only until the first leaf breaks through the coleoptile (Griffiths 1924). Hence any factor that slows emergence, such as deep sowing (Miller and Millikan 1934a, El-Helaly 1948, Sattar and Hafiz 1952a), may increase FS incidence.

As a general rule, FS occurs more frequently in light, relatively dry soils (McAlpine 1910, Purdy 1966) in the 18-24°C temperature range (Purdy 1965, 1966, Purdy et al. 1964, McIntosh 1968, Greenhalgh and Brown 1984).

Because soil temperature changes during the season, time of sowing is an important factor. In Missouri, USA, the disease was more severe in early sowings from mid-October until early November than after mid-November (Griffiths 1924). However, in southern Australia, where the crop cycle is different, the incidence of FS is lower in the early sowings in April-May than in June (Ballantyne 1993).

The effect of fertilizer is controversial (Foster and Vasey 1929; Miller and Millikan 1934b; Millikan 1939a,b; Yasu and Yoshino 1963).

Soil pH from 5.5 to 8.7 favored FS infection (Miller and Millikan 1934b). In the laboratory, Noble (1924) found that the range was pH 4.1-6.4, but the optimum was at pH 5.1-5.7.

Variation in Infection Frequency and Expression

Generally, FS is more severe in glasshouse trials than in field tests, but considerable variation occurs. Cultivars that have little or no infection in the glasshouse may be severely infected in the field and vice versa (R.F. Line, per. comm.; Griffiths 1924; Johnson 1959; Purdy et al. 1964; McIntosh 1968).

Cutting back plants favors the development of FS in the regrown tissues. Often, some resistant plants are rated as susceptible after being cut back (Griffiths 1924, Jarrett 1932, Miller and Millikan 1934a, Purdy et al. 1964). Cutting back at the two-leaf stage had a greater impact than cutting back after spike formation (Griffiths 1924).

Variation in the incidence of infection at different sites and in different seasons or when inoculum is seedborne rather than soilborne is a common feature of FS screening (Pridham and Dwyer 1930, Purdy et al. 1964, McIntosh 1968, Platz and Rees 1980, Ballantyne 1993).

Control Strategies

Physiologic Resistance

Sources. Resistance to FS is quite widespread as reported by many workers (USA: Reed and Dungan 1920; Tisdale et al. 1923, 1927; Griffiths 1924; Melchers 1938; Purdy et al. 1964; Line 1972; Australia: Shelton 1924; Carne and Limbourn 1927; Pridham et al. 1929; Pridham and Dwyer 1930; Limbourn 1931; Miller and Millikan 1934a; Millikan and Sims 1937; Sims et al. 1943; Anon. 1952; Cass Smith 1954; Anon. 1960; Ballantyne 1993; India: Pal and Mundkur 1941; Joshi et al. 1970; Goel and Jhooty 1984; South Africa: Verwoerd 1929; China: Yu and Chen 1931; Yu et al. 1933, 1934; Fischer and Holton 1957; Purdy 1965; Egypt: El-Helaly 1948; Mexico: Borlaug et al. 1946). Some details are in **Table 5.5**.

Breeding for FS resistance does not appear to be a high priority in any breeding program. In Australian breeding programs, advanced material and candidate parents are commonly screened. This enables culling of some susceptible segregates and planning of crosses. Ballantyne (1993) recently reviewed the Australian resistance situation and reported that most

Table 5.5. Selected reported reactions of wheat genotypes to flag smut.

USA Midwest
Reed and Duncan (1920), Griffiths (1924), Tisdale et al. (1923, 1927), Melchers (1938), Churchward (1938). **Resistant:** Bobs, Clarkan, Early May, Fulcaster, Galgalos*, Gypsy, Hope*, Kawvale, Red Man, Red Rock*, Redwave, Reliable, Tenmarq, Turkey.

USA Pacific Northwest
Purdy et al. (1964), Line (1972). **Resistant:** Burt*, Cheyenne, Columbia, Itana, Kendee, Moro, Nugaines, Wanser. **Intermediate:** PI178383*, PI194349*, Dickson 114*, Norin Brevor Sel 14*. **Susceptible:** Brevor, Gaines, Luke, Omar, Paha.

Australia
Shelton (1924), Carne and Limbourn (1927), Pridham et al. (1929), Pridham and Dwyer (1930), Limbourn (1931), Miller and Millikan (1934a), Millikan and Sims (1937), Shen et al. (1938), Sims et al. (1943), Anon. (1952, 1960, 1970), Cass Smith (1954), McIntosh (1968), Ballantyne (1993). **Resistant:** Baringa, Baroota Wonder, Bencubbin, Bunyip, Charter, Condor, Currawa, Dan*, Dundee, Egret, Festival, Fife, Firbank*, Ford, Florence, Geeralying*, Ghurka*, Gullen, Heron, Insignia, Kite, Nabawa*, Pusa 4, Timgalen, Wandilla King, Most WW15 derivatives. **Susceptible:** Baart, Federation, Gabo, Hard Federation, Mendos, Rosella, White Federation, WW33G.

India
Pal and Mundkur (1941), Joshi et al. (1970). Goel and Jhooty (1984). **Resistant:** Bluebird, Ranee, PI13569, PI13751, PI14355, PI14391, Sonalika. **Susceptible**: Most semidwarf wheats, C306, Chhoti Lerma, Kalysansona, WH147, UP301.

China
Yu and Chen (1931), Yu et al. (1933, 1934), Shen et al. 1938). **Resistant:** Nanking 16, Nanking 716, Nanking 4592*, TH1932*.

Egypt
El-Helaly (1948). **Resistant:** Hindi, Mabarouk.

Mexico
Borlaug et al. (1946). **Susceptible:** Most Mexican wheats (not CIMMYT).

* Subjected to genetic analysis of flag smut reaction.

recommended cultivars and locally adapted CIMMYT lines were resistant, but a number of introductions from the USA were susceptible or very susceptible in a series of field trials. Most of the durum wheats screened have been resistant.

Inheritance. Resistance is generally controlled by a number of genes and transgressive segregation is common (Shen 1934, McMillan 1935, Shen et al. 1938, Purdy and Allan 1967, McIntosh 1968, Helm and Allan 1971). There are a few reports of simple inheritance (Churchward 1938, Helm and Allan 1971, Miller and Millikan 1934a). Resistance has generally been recessive, but there are occasional reports of dominance (Shen 1934, Helm and Allan 1971).

Purdy and Allan (1967) obtained heritability estimates of 49-74% from regression analyses. The genetic variance estimated in the F2 generation was mainly additive in the populations studied by McIntosh (1968). Genotype-environment interactions were small in relation to genetic variance. Broad sense heritability estimates determined from F3 data, involving individual crosses, were usually within the 40-70% range .

Churchward (1938) reported loose genetic linkage in Hope wheat between a possible single gene pair determining FS resistance and resistance to bunt. In a comparison of isolines (Allan 1975), wheats with the semidwarf trait had increased susceptibility to FS possibly due to the delayed emergence typical of semidwarf wheats, which probably offered increased opportunity for infection. Allan and Pritchett (1976) reported that club wheats were likely to be susceptible to FS. The *C* allele responsible for the club head type reduces rates of seedling growth and emergence, so again a delay in emergence appears to be involved.

Breeding. McMillan (1935) obtained infection data for a number of crosses in succeeding generations and recommended that selection be based upon progeny tests in later generations, not earlier than the F3 population.

Seed Treatment

Water and anaerobic seed treatments have not been reported for FS.

Control of the common bunt fungi (*Tilletia tritici* and *T. laevis*) was the primary impetus for cereal seed dressing, and this has influenced the chemicals used for control of FS. The literature has been summarized by Holton and Heald (1941), Martin (1959), Callan (1973), and Hoffman (1981). Most of the fungicides used for control of bunt are also effective against FS, but there are some exceptions, notably hexachlorobenzene. Information about such chemical applications, which began in the early 1900s, is given in **Table 5.6**.

The seed dressings currently recommended for control of FS in New South Wales include the triazoles (bitertanol, tecbuconazole, triademefon) and the carbozanilides (carboxin, flutriafol). Carboxin is advised for the control of seedborne inoculum only, but the other systemic fungicides are recommended for both seedborne and soilborne infection. The rates are 70-100 g/110 kg of seed for the powder formulations or 100-250 ml/100 kg of seed for the liquid treatments (Verrell and Komoll 1991, Gammie 1994).

Cultural Practices

Cultural practices that have been advised to control FS include rotations with a

Table 5.6. Fungicides that have been evaluated for the control of wheat flag smut.

Reference/ chemical	Comments
McAlpine (1906)	
Formaldehyde	Effective but inconvenient
Copper sulfate	Effective but inconvenient
Morwood (1930, 1931)	
Copper carbonate	Effective except at high inoculum doses
Cass Smith (1954), Purdy (1957, 1961, 1963, 1965), Line (1972), Holton and Purdy (1954)	
Formalin	Effective only with seedborne inoculum
Copper	Effective only with seedborne inoculum
Mercury*	Effective only with seedborne inoculum
Hexachlorobenzene*	Effective with both seed- and soilborne inoculum
Moore and Kuiper (1974)	
Mancozeb	Effective
Kuiper and Murray (1978), Kuiper (1968), Metcalfe and Brown (1969), Line (1972)	
Benomyl	Less effective
Pentachloronitrobenzene	Less effective
Line and Scott (1987)	
Carboxin	Each compound gives complete
Oxycarboxin	control and is systemic
Triademinol	
Triademefon	
Loughman (1989)	
Triademefon	Effective to complete control
Triademenol	Effective to complete control
Diniconazole	Effective to complete control
Carboxin	Effective
Flutriafol	Effective

* Withdrawn in Australia on toxicological grounds.

nonhost species, stubble burning, variations in planting time, and variations in depth of sowing (Hamblin 1921, Purdy 1965, Brown 1975).

Purdy (1965) and Brown (1975) recommended rotations with a nonhost crop, but the wheat-free period needs to be longer than that used for control of root rots and foliar pathogens.

Stubble burning may reduce the infection. In one experiment in northern New South Wales, FS incidence was 1.25% where stubble was burned, but 9% where stubble was plowed under (Brown 1975). On self-mulching soils, stubble burning may be less effective because flag smut spore balls are likely to be buried in the soil and so escape the high temperatures.

In Australia, early sowing in autumn into warm soils favors infection, whereas later sowing into cool soils during the winter may result in disease-free conditions (Brown 1975). However, in Egypt, early sowing in October in warm soils resulted in almost complete control of FS, whereas late sowing in December in cooler soils resulted in severe FS (El-Helaly 1948).

Quarantine

In the USA, an FS-infested area was quarantined (Stakman 1922) immediately after the disease was discovered (Humphrey and Johnson 1919) and the area affected was extended as the known distribution of the disease increased. The movement of wheat and farm machinery from the infested areas was prohibited. Machinery was to be used only in infested areas and was to be disinfected with formaldehyde before harvesting oats or straw at the end of the season. Resistant cultivars, seed treatment with copper sulfate, and lime and stubble burning after harvest were recommended.

USA Federal Quarantine No. 39, effective August 15, 1919 (Anon. 1919, Weber 1930), prohibited importation of cereals in the raw (seed) or uncleaned state from Australia, Italy, France, Germany, Belgium, Great Britain, Iceland, Brazil, and India except by special permit. Quarantine 39 was superseded by Quarantine No. 59, which prohibited the importation of wheat from many countries where FS occurs (Anon. 1925). Germany was released from restrictions in 1954 since FS of wheat was apparently absent from its territory (Anon. 1954).

USA Federal Quarantine No. 69, which is related to packing material and became effective June 8, 1953, exempts from its provisions wheat straw, chaff, and hulls used for packing material when subject to the FS quarantine (Anon. 1953).

In addition to wheat, Australian oats and barley are prohibited imports to the USA because they are grown in wheat areas and may therefore be contaminated by spores of *U. agropyri* (Ballantyne 1993).

The potential danger of FS is recognized by several regions and countries by quarantine regulations: Transcaucasia (Szembel 1934), Russia (Anon. 1935), Canada (Anon. 1930, 1932, 1955), Mexico (Anon. 1928, 1931) and Iran (Anon. 1946). Careful inspections are required (Minz 1943, Pollack 1945).

Techniques for Study

Inoculation

Inoculate seed by shaking spores onto seeds (Jarrett 1932; Miller and Millikan 1934a; Rewal and Jhooty 1986b), by mixing inoculum with soil before planting, or by spraying inoculum on the open row (Rewal and Jhooty 1986b, Line and Scott 1987). Planting in naturally infested soil has also been done (Miller and Millikan 1934a, Loughman 1989, Ballantyne 1993). All methods give satisfactory results if the environmental conditions are favorable. McIntosh (1968) and Greenhalgh and Brown (1984) developed precise methods for determining cultivar reaction.

Disease Rating Scales

Most workers have used percentage infection to record responses to FS, although Pridham et al. (1929) noted that this did not measure the severity on individual plants.

Yu et al. (1945) established three infection classes on an arbitrary basis for race identification studies:

- 0-5% infection = Resistant (R).
- 5.1-20% infection = Intermediate (I).
- 20.1-100% infection = Susceptible (S).

Goel and Jhooty (1984) distinguished six categories:

- No disease = Immune.
- 0.1-2.0% infection = Highly resistant.
- 2.1-5.0% infection = Moderately resistant.
- 5.1-10.0% infection = Moderately susceptible.
- 10.1-20.0% infection = Susceptible.
- >20 % infection = Highly susceptible.

Ballantyne (1993) used a 0-9 scale 15-20 plants per row:

- 0 = no disease = highly resistant.
- 1-2 = trace to two infected tillers per row = resistant.
- 3-4 = Three or four tillers per row of one plant partially infected = moderately susceptible.
- 5-6 = a few plants partially or severely infected = susceptible.
- 7-9 = Many to all plants severely infected = highly susceptible.

References

Abdel-Hak, T., and E. Ghobrial. 1969a. Studies on the control of bunt of wheat, *Tilletia foetida* Wallr. in U.A.R. Tech. Bull. No. 7, 1968. Plant Protection Dept., Min. of Agric., Cairo. 25 pp.

Abdel-Hak, T., and E. Ghobrial. 1969b. Studies on the control of flag smut of wheat, *Urocystis tritici Koern.* in U.A.R. Tech. Bull. No. 8, 1968. Plant Protection Dept., Min. of Agric., Cairo. 25 pp.

Abdul Sattar, M.H., and M.N. Haithami. 1986. Diseases of major crops in Democratic Yemen and their economic importance. Plant Prot. Bull. 34:73-76.

Aebi, H. 1956. Recherches sur la Germination des Chlamydospores de la Carie Naine du Froment (Investigations on the germination of dwarf bunt chlamydospores from wheat). Stations Fédérales d'Essais Agricoles, Lausanne 509:533-542.

Agarwal, V.K., A. Singh, and H.S. Verma. 1976. Outbreak of Karnal bunt of wheat. Plant Prot. Bull. 24:99-100.

Agarwal, V.K., H.S. Verma, and R.K. Khetarpal. 1977. Occurrence of partial bunt on triticale. Plant Prot. Bull. 25:210-211.

Agarwal, V.K., M. Agarwal, R.K. Gupta, and H.S. Verma. 1981. Studies on loose smut of wheat. I. A simplified procedure for the detection of seedborne infection. Seed Res. 9:49-51.

Agarwal, V.K., D.V. Singh, and S.B. Mathur. 1993a. Karnal bunt (*Tilletia indica*). In pages 31-43, S.B. Mathur and B.M. Cungfer, eds., Seedborne Diseases and Seed Health Testing of Wheat. Institute of Seed Pathology for Developing Countries. Danish Government. ABC Grafik, Fredeirksberg, Denmark.

Agarwal, V.K., S.S. Chahal, and S.B. Mathur. 1993b. Loose smut (*Ustilago tritici*). In pages 59-67, S.B. Mathur and B.M. Cungfer, Seedborne Diseases and Seed Health Testing of Wheat. Institute of Seed Pathology for Developing Countries. Danish Government. ABC Grafik, Fredeirksberg, Denmark.

Ahmad, S., and Attaudin. 1991. Appraisal of Karnal bunt of wheat in the North West Frontier Province of Pakistan. Pakistan J. Phytopath. 3:35-37.

Ahmed, H. 1986. Prevailing wheat diseases in Bangladesh. In pages 124-134, Third National Wheat Training Workshop. August 4-5, 1986. Wheat Research Centre, Bangladesh Agricultural Research Institute, CIMMYT/CIDA Wheat Program, Dhaka, Bangladesh.

Ahmed, S.M., M.A. Razzaque, N.K. Saha, and M.A. Sufian. 1986. Varietal development of wheat for irrigated and water stressed situations and some ideas on future research requirements. In pages 66-73, Third National Wheat Training Workshop. August 4-5, 1986. Wheat Research Centre, Bangladesh Agricultural Research Institute, CIMMYT/CIDA Wheat Program, Dhaka, Bangladesh.

Akbari, A., and M. Zolghadri. 1988. Current status of crop production, research, infrastructure and constraints in Iran. In pages 181-189, J.P. Srivastava, M.C. Saxena, S. Varma, and M. Tahir, eds., Winter Cereals and Food Legumes in Mountainous Areas. ICARDA, Aleppo, Syria.

Allan, R.E. 1975. Relation of flag smut incidence to semidwarf growth habit in wheat isolines. Crop Sci. 15:427-429.

Allan, C.R., and R. Durán. 1979. *Urocystis agropyri*: the influence of aging on teliospore germination. Plant Dis. Rep. 63:841-843.

Allan, R.E., and J.A. Pritchett. 1976. Relation of flag smut incidence to spike type in wheat isolines. Crop Sci. 16:596-597.

Amaya, C.A. 1982. Efecto de diferentes niveles del hongo causante del carbón parcial sobre las características organolépticas del pan. Boletín Técnico. Laboratorio de Calidad Industrial. CIMMYT, Mexico.

Andrews, J.A., and D.J. Ballinger. 1987. Potential for bunt resistant wheats. Austral. Plant Path. 16:52-53.

Angell, H.R. 1934a. A preliminary note on the recognition of flag smut or bunt infection based on the deformation of seedlings. Council for Sci. and Ind. Res. 7:110-114.

Angell, H.R. 1934b. Flag smut of wheat—early symptoms. Council for Sci. and Ind. Res. 7:153-156.

Angell, H.R., F.W. Hely, and F.E. Allen. 1938a. The effect of *Urocystis tritici* Koern. on the extent of development of the roots and aerial parts of the wheat plant I. Council for Sci. and Ind. Res. 10:136-142.

Angell, H.R., F.W. Hely, and F.E. Allen. 1938b. The effect of *Urocystis tritici* Koern. on the extent of development of the roots and aerial parts of the wheat plant II. Council for Sci. and Ind. Res. 10:256-257.

Anon. 1868. Report of the South Australian Commission on Diseases on Cereals, 1968. Quoted by Miller and Millikan (1934b).

Anon. 1919. Federal Horticultural Board, U.S. Dep. Agr. Quarantine on account of flag smut and take-all diseases. Notice of Quarantine No. 39 (with regulations). U.S.D.A. Serv. and Regulatory Announcements 64: 77-79. Quoted by Purdy (1965).

Anon. 1925. Quarantine on account of the flag smut disease. Notice of Quarantine No. 59. U.S. Dep. Agr. Fed. Hort. Board Leaflet. Quoted by Purdy (1965).

Anon. 1928. Decreto por el cual se establece la cuarantena exterior Num. 8 contra las especies y variedades de trigo (*Triticum* spp.). Bol Mens. Ofic. Def. Agr. Estados Unidos Mexicanos 2:633-635. Rev. Appl. Mycol. 8:272 (1929). (Abstr.)

Anon. 1930. Canada Dept. Agr. Destructive Insect and Plant Pest Act Advisory Board. Regulations under the Destructive Insect and Plant Pest Act P.C. 368, Regulation No. 18. Quoted by Purdy (1965).

Anon. 1931. Legislative and administrative measures. Mexico. Internat. Bull. Plant Protect. 5:122-123. Rev. Appl. Mycol. 10:816 (1931). (Abstr.)

Anon. 1932. Canada Dept. Agr. Destructive Insect and Pest Act Advisory Board. Regulation under the Destructive Insect and Pest Act P.C. 643. Regulation 18 (foreign), 1st revision. Quoted by Purdy (1965).

Anon. 1935. List of insect pests and diseases prohibited by external quarantine legislation from importation into the USSR (translated title) USSR Peoples Commissariat Agr. Sect. intern. extern. Pl. Quar. 2, Moscow. Rev. Appl. Mycol. 15:399-400. (Abstr.)

Anon. 1943. Annual Report. Wheat Sub-Station, Gurdaspur, India. pp. 1-15.

Anon. 1946. Plant Quarantine Restrictions, Iran. Service and Regulatory Announcements, Bureau of Entomology and Plant Protection, U.S.D.A. 168:51-54.1947. Quoted by Purdy (1965).

Anon. 1952. Wheat Varieties of Australia. Farmer's Bulletin 173. N.S.W. Dept. of Agric. 132 pp.

Anon. 1953. Plant quarantine announcement. Food and Agriculture Organization. Plant Prot. Bull. 1:337-353.

Anon. 1954. Quarantine and other official announcements. Announcements relating to flag smut quarantine (No. 59) Service and Regulatory Announcement, Bur. Ent. Washington, 183:3-4.

Anon. 1955. Plant quarantine announcements. Plant Prot. Bull. 3:110-111.

Anon. 1960. Flag smut of wheat. Agricultural Gazette of New South Wales 71:140-142.

Anon. 1968. *Tilletia controversa* Kuhn. Map No. 297. Distribution Maps of Plant Diseases. Commonwealth Mycological Institute. Surrey, England.

Anon. 1969. Information Bulletin on the Near East Wheat and Barley Improvement and Production Project. Vol. 6, No. 1. FAO Rome, 21 October 1969, 100 pp.

Anon. 1970. Flag smut of wheat. Agric. Gaz. N.S.W. 81:588-590.

Anon. 1976. Maladies, nematodes, insectes (1.cereales). In pages 19-36, Maladies et Ravageurs des Plantes Cultivees au Maroc. Tome 1. Min. Agric. et de la Reforme Agraire. Direc. Rec. Agronom. Rabat, Morocco.

Anon. 1977. *Tilletia foetida* (Wallr.) Liro. Map No. 295. Distribution Maps of Plant Diseases. International Mycological Institute, Surrey, England.

Anon. 1982. *Ustilago nuda* (Jens.) Rostr. Map No. 368. Disribution Maps of Plant Diseases. Commonwealth Mycological Institute, Surrey, England.

Anon. 1983. U.S. Quarantine of Mexican wheat: dilemma for researchers and exporters. Diversity 5:13-14.

Anon. 1989a. *Tilletia indica* Mitra. Map No. 173. Distribution Maps of Plant Diseases. Edition 4. Commonwealth Mycological Insitute. Surry, England.

Anon. 1989b. Normas de calidad, programa de compras de trigo, cosecha primavera/verano 1989-89. Compañía Nacional de Subsistencias Populares. Mexico.

Anon. 1990. *Tilletia tritici (Bjerk.)* R. Wolff. Map No. 294. Distribution Maps of Plant Diseases, International Mycological Institute, Surrey, England.

Anon. 1991. *Urocystis agropyri* (Preuss) Schroter. Map No. 80. Distribution Maps of Plant Diseases. International Mycological Institute, Kew, Surrey, England.

Antonelli, E.F. 1983. Principal pathogens affecting wheat production in Argentina. In pages 377-387, Cereal Breeding and Production Symposium. Marcos Juarez, Argentina, November 7-12, 1983. Special Report 718, Agric. Expt. Sta. and Crop Sci. Dept., Oregon State University. INTA, OSU and US-AID.

Atac, A. 1988. Investigations on the reactions of some triticale and wheat varieties against wheat bunt disease [*Tilletia foetida* (Wallr.)Liro.] in Cukurova region. J. Turk. Phytopath. 17:75-79.

Atkins, I.N., O.G. Merkle, K.B. Porter, K.A. Lahr, and D.E. Weibel. 1963. The influence of environment on loose smut percentages, reinfection and grain yields of winter wheat at four locations in Texas. Plant Dis. Rep. 47:192-196.

Aujla, S.S., and Y.R. Sharma. 1977. Simultaneous occurrence of *Ustilago nuda tritici* and *Urocystis agropyri*. Indian Phytopath. 30:262.

Aujla, S.S., Y.R. Sharma, K. Chand, and S.A. Sawney. 1977. Influence of weather factors on the incidence and epidemiology of Karnal bunt disease of wheat in the Punjab. Indian J. Ecol. 4:71-74.

Aujla, S.S., A.S. Grewal, K.S. Gill, and I. Sharma. 1980. A screening technique for Karnal Bunt disease of wheat. Crop Imp. 7:145-146.

Aujla, S.S., K.S. Gill, Y.R. Sharma, D. Singh, and G.S. Nanda. 1981. Effect of date of sowing and level of nitrogen on the incidence of Karnal bunt of wheat. Indian J. Ecol. 8:175-179.

Aujla, S.S., A.S. Grewal, K.S. Gill, and I. Sharma. 1982. Artificial creation of Karnal bunt disease of wheat. Cer. Res. Com. 10:171-175.

Aujla, S.S., A.S. Grewal, and I. Sharma. 1983. Relative efficiency of Karnal bunt inoculation techniques. Indian J. Mycol. Plant Path. 13:99-100.

Aujla, S.S., I. Sharma, K.S. Gill, and V. Kour. 1985. *Neovossia indica* on wild species of wheat. Indian Phytopath. 38:191.

Aujla, S.S., I. Sharma, K.S. Gill, and A.S. Grewal. 1986a. Prevalence of Karnal Bunt in Punjab as influenced by varietal susceptibility and meteorological factors. Plant Dis. Res. 1:51-54.

Aujla, S.S., I. Sharma, and B.B. Singh. 1986b. Effect of various fungicides on teliospore germination of *Neovossia indica*. J. Res. Punjab Agric. Univ. 23:442-443.

Aujla, S.S., I. Sharma, and B.B. Singh. 1987. Physiologic specialization of Karnal Bunt of wheat. Indian Phytopath. 40:333-336.

Aujla, S.S., I. Sharma, and B.B. Singh. 1989. Rating scale for identifying wheat varieties resistant to *Neovossia indica*. Indian Phytopath. 42:161-162.

Azmeh, M.F., and T. Kousaji. 1982. Contribution to survey of plant diseases in Syria: unreported fungal diseases. 22nd Science Week, Board of High Education, Ministry of High Education. Nov. 1982. Damascus, Syria.

Baker, E.P. 1967. Inheritance of resistance to bunt in Turkey wheat selections. Proc. Linn. Soc. N.S.W. 91:189-210.

Ballantyne, B. 1993. Reactions of wheat varieties to flag smut in Southern New South Wales. Plant Disease Survey. Austral. Plant Path. 22:100-104.

Ballinger, D.J., and G.J. Gould. 1989. Occurrence of spores of *Tilletia laevis* and *T. tritici* in wheat deliveries in western Victoria. Austral. Plant Path. 18:6-7.

Bamberg, R.H., C.S. Holton, H.A. Rodenhiser, and R.W. Woodward. 1947. Wheat dwarf bunt depressed by common bunt. Phytopathology 37:556-560.

Bamdadian, A. 1993a. Wheat smuts. In pages 18-51, Diseases of Wheat in Iran. In press.

Bamdadian, A. 1993b. A short report on the wheat bunt in Iran. Plant Protection Research Institute. Evin, Iran. 2 pp.

Banowetz, G.M., E.J. Trione, and B.B. Krygier. 1984. Immunological comparisons of teliospores of two wheat bunt fungi, *Tilletia* species, using monoclonal antibodies and antisera. Mycologia 76:51-62.

Bansal, R., D.V. Singh, and L.M. Joshi. 1983. Germination of teliospores of Karnal bunt of wheat. Seed Res. 11:258-261.

Bansal, R., D.V. Singh, and L.M. Joshi. 1984a. Comparative morphological studies in teliospores of *Neovossia indica*. Indian Phytopath. 37:355-357.

Bansal, R., D.V. Singh, and L.M. Joshi. 1984b. Effect of Karnal bunt pathogen [*Neovossia indica* (Mitra) Mundkur] on weight and viability of wheat seed. Indian J. Agric. Sci. 54:663-666.

Baraev, A.I. 1983. Spring Wheat in North Kazakhstan. Translated from Russian. USDA and the National Sci. Foundation, Washington D.C., Amerind Publishing Co. Pvt. Ltd. New Delhi. 233 pp.

Bassiouni, A.A., S. Abou El-Naga, A. Gouda, and W.A. Youssef. 1988. Sources of resistance to loose smut of wheat caused by *Ustilago tritici* (Pers.) Rostrup, 39/41. 7th Europ. Medit. Cereal Rusts and Mildews Conf. Sept. 5-9, 1988, Vienna, Austria.

Batts, C.C.V. 1955a. Loose smut (*Ustilago tritici* (Pers.) Rostr.) of wheat: physiological specialization and reaction of varieties in England. Ann. Appl. Biol. 43:533-537.

Batts, C.C.V. 1955b. Observations on the infection of wheat by loose smut (*Ustilago tritici* (Pers.) Rostr.). Trans. Brit. Mycol. Soc. 38:465-475.

Batts, C.C.V., and A. Jeater. 1958a. The development of loose smut (*Ustilago tritici*) in susceptible varieties of wheat, and some observations on field infection. Trans. Brit. Mycol. Soc. 41:115-125.

Batts, C.C.V., and A. Jeater. 1958b. The reaction of wheat varieties to loose smut as determined by embryo, seedling, and adult plant tests. Ann. Appl. Biol. 46:23-29.

Baylis, R.J. 1958. Studies of *Tilletia controversa*, the cause of dwarf bunt of winter wheat. Can. J. Bot. 36:17-32.

Bedi, S.K.S., M.R. Sikka, and B.B. Mundkur. 1949. Transmission of wheat bunt due to *Neovossia indica* (Mitra) Mundkur. Indian Phytopath. 2:20-26.

Bedi, P.S., M. Meeta, and J.S. Dhiman. 1981. Effect of Karnal bunt of wheat on weight and quality of the grains. Indian Phytopath. 34:330-333.

Begum, S., and S.B. Mathur. 1989. Karnal bunt and loose smut in wheat seed lots of Pakistan. Plant Prot. Bull. 37:165-173.

Bever, W.M. 1947. Physiologic races of *Ustilago tritici* in the eastern soft wheat region of the United States. Phytopathology 37:889-895.

Bever, W.M. 1953. Further studies on physiologic races of *Ustilago tritici*. Phytopathology 43:681-683.

Bever, W.M. 1961. Experiments on the control of *Ustilago nuda* and *Ustilago tritici* by means of thermo-chemical treatments. Lucrari Stiint. 17:1-7.

Bathnagar, G.C., R.B.L. Gupta, and V.L. Mishra. 1978. Effect of flag smut, caused by Urocystis agropyri on yield components of wheat cultivars in Rajastan, India. Plant Dis. Rep. 62:348-350.

Bhatti, M.A.R., and M.B. Ilyas. 1986. Wheat Disease in Pakistan. Pages 20-30, L.M. Joshi, D.V. Singh and K.D. Srivastava, eds., Problems and Progress of Wheat Pathology in South Asia. Malhotra Publishing House, New Delhi.

Bhutta, A.R. and S.I. Ahmed. 1991. A report on the incidence of loose smut of wheat crop and seed in some parts of the Punjab Province during 1990-91. Pakistan J. Phytopath. 3:7-11.

Bicici, M., Y. Dede, A. Erkilic, and S. Toker. 1991. The prevalence and incidence of bunt disease in wheat growing area in GAP region and their effect on the crop yield. J. Turk. Phytopath. 20:82.

Boewe, G.H. 1960. Diseases of wheat, oats, barley and rye. Illinois Nat. Hist. Survey. Circ. 48. 157 pp. Quoted by Purdy 1965.

Bonde, M.R., J.M. Prescott, T.T. Matsumoto, G.L. Peterson. 1987. Possible dissemination of teliospores of *Tilletia indica* by the practice of burning wheat stubble. Phytopathology 77:639 (Abstr.).

Böning, K. 1958. Ergebnisse von Untersuchungen an Herbarmaterial von auf Getreide und Gräsern vorkommenden Steinbrandarten. Pflanzenschutz. 53:222-224.

Borlaug, N.E., J.G. Harrar, and E.C. Stakman. 1946. Flag smut of wheat in Mexico. Phytopathology 36:479.

Brefeld, O. 1883. Botanisch Untersuchungen uber Hefenpilze. 5. De Brandpilze. I. Ustilagineen. Verlag von A. Felix. Leipzig.

Brefeld, O., and R. Falck. 1905. Die Blueteninfection bei den Brandpilzen und die natuerliche Verbreitung der Brandkrankheiten. In: Brefeld, O. Untersuchungen aus dem Gesammtgebiete der Mykologie. Heft 13. Muenster. 74 pp.

Bremer, H., G. Karel, K. Biyikoglu, N. Goskel, and F. Petrak. 1952. Beiträge zur Kenntnis der Parasitischen Pilze der Turkei. Rev. Fac. Sci. Univ. D'Istanbul, Ser. B. 17:161-181.

Brennen, J.P., and G.M. Murray. 1988. Australian wheat diseases: assessing their economic importance. Agric. Sci. 1:26-35.

Brennan, J.P., E.J. Warham, J. Hernández, D. Byerlee, and F. Coronel. 1990. Economic losses from Karnal bunt of wheat in Mexico. CIMMYT Economics Working Paper 90/02.

Briggs, F.N. 1926. Inheritance of resistance to bunt, *Tilletia tritici* (Bjerk.) Winter, in wheat. J. Agric. Res. 32:973-990.

Briggs, F.N. 1930. Inheritance of the second factor for resistance to bunt, *Tilletia tritici*, in Hussar wheat. J. Agric. Res. 40:225-232.

Briggs, F.N. 1933. A third genetic factor for resistance to bunt, *Tilletia tritici*, in wheat hybrids. J. Genet. 27:435-441.

Brittlebank, C.C. 1920. Seedborne diseases, "take-all and flag smut". J. Agric. Victoria 19:447.

Brown, J.F. 1975. Diseases of wheat: their incidence and control. In pages 304-363, A. Lazenby and E.M. Matheson, eds., Australian Field Crops, Volume I, Wheat and Other Temperate Cereals. Angus & Robertson, Sydney.

Buchheim, A.N. 1935. Effect of freezing on the germinability of wheat seeds infected with loose smut, and on the development of plants raised from them. Plant Prot. (Leningrad) 6:134-137.

Callaghan, A.R., and A.J. Millington. 1956. The Wheat Industry in Australia. Angus and Robertson, Sydney. 486 pp.

Callan, I.W. 1975. Achievements and limitations of seed treatments. Outlook on Agriculture 8:271-274.

Campagna, E. 1926. New white smut of wheat, collected at Ste. Anne de la Pocatière. Ann. Rept. Que. Soc. Prot. Plants 18:71-72.

Carne, W.M. 1924. Flag smut of wheat (*Urocystis agropyri*) J. Dep. Agric. Western Australia, Series 2, 1:142-147.

Carne, W.M., and E.J. Limbourn. 1927. Flag smut of wheat. Varietal resistance test. J. Dep. Agric. Western Australia 4:4-7.

Carreon Zuniga, M.A. 1992. Perspectives on the KB problem: Mexico. In pages 2-3, G. Fuentes-Davila and G.P. Hettel, eds., Update on Karnal Bunt Research in Mexico. Wheat Special Report No. 7a. Mexico, D.F.: CIMMYT.

Cashion, N.L., and E.S. Luttrell. 1988. Host-parasite relationships in Karnal bunt of wheat. Phytopathology 78:75-84.

Cass Smith, W.P. 1954. Cereal smut diseases and their control. J. Dep. Agric. Western Australia 3:329-338.

Cherewick, W.J. 1953. Smut diseases of cultivated plants in Canada. Can. Dept. Agric., Ottawa, Publ. 887. 58 pp.

Cherewick, W.J. 1958. Cereal smut races and their variability. Can. J. Plant Sci. 38:481-489.

Cherewick, W.J., and R.H. Cunningham. 1956. Further improvement in the partial vacuum method of inoculation with loose smut fungi. Phytopathology 46:355-358.

Cherewick, W.J., and W. Popp. 1950. A modification of Moore's method of inoculating wheat and barley with loose smut. Phytopathology 40:1054-1056.

Chona, B.L., R.L. Munjal, and K.L. Adlakha. 1961. A method for screening wheat plants for resistance to *Neovossia indica*. Indian Phytopath. 14:99-101.

Churchill, A.C.L., and D. Mills. 1985. Heterokaryon formation in planta by genetically marked strains of Tilletia caries. Can. J. Bot. 63:1924-1927.

Churchward, J.G. 1931. Studies in the inheritance of resistance to bunt in a cross between Florence X Hard Federation wheats. J. Roy. Soc. N.S.W. 64:298-319.

Churchward, J.G. 1934. A note on the occurrence of seedling lesions caused by cereal smuts. Proc. Linn. Soc. N.S.W. 59:197-199.

Churchward, J.G. 1938. Studies on physiological specialization of the organism causing bunt in wheat and the genetics of resistance to this and certain other wheat diseases. Part II. Genetical studies. J. Roy. Soc. N.S.W. 71:547-90.

Churchward, J.G. 1940. The initiation of infection by bunt of wheat *Tilletia caries*. Ann. Appl. Biol. 27:58-65.

Clayton, E.S. 1925. A common source of infection with flag smut. Agric. Gaz. N.S.W. 36:860.

Clinton, G.P. 1904. North American Ustiliginales. Proc.Boston Soc. Nat. Hist. 31:329-529. Quoted by Purdy (1965).

Cobb, N.A. 1891. Smut. Agric. Gaz. N.S.W. 2:672-677.

Conners, I.L. 1954. The organism causing dwarf bunt of wheat. Can. J. Bot. 32:426-431.

Coons, C.R. 1919. St. Louis Conference on Take-all and Flag Smut of Wheat. Phytopathology 9:330-332.

Coons, G.H., and F.A. Spragg. 1918. Resistance and susceptibility of certain wheat varieties to loose smut. Phytopathology 8:69-70.

Cooper, F.E. 1983. Wheat from Mexico. Plant Protection and Quarantine, Animal and Plant Inspection Station, U.S.D.A.

Cramer, H.H. 1967. Plant Protection and World Crop Production. Byer, Pflanzenschutz, Leverkusen, Germany. 524 pp.

Danko, J., and A. Michalikova. 1968. Evaluation of methods of artificial inoculation of wheat with *Ustilago tritici* (Pers.) Jens. Polnohospodarstvo 14:665-675.

Danko, J., and A. Michalikova. 1969. Influence of some external factors on the germination of chlamydospores of *Ustilago tritici* (Pers.) Jens. Polnohospodarstvo 15:124-130.

Dastur, J.F. 1921. Cytology of *Tilletia tritici* (Bjerk.) Wint. Ann. Bot. 35:399-407.

Dean, W.M. 1969. The effect of temperature on loose smut of wheat (*Ustilago nuda*). Ann. Appl. Biol. 64:75-83.

De Bary, A.H. 1853. Untersuchungen uber die Brandpilze und die durch sie verursachten Krankheiten der Pflanzen mit Rucksicht auf das Getreide und andere Nutzflanzen. Muller. Berlin.

Dewey, W.G. 1961. Studies of the effect of straw cover and method of inoculation of dwarf bunt of winter wheat in Utah. Plant Dis. Rep. 45:663-665.

Dewey, W.G. 1963. The use of vermiculite cover as a possible means of increasing dwarf bunt in winter wheat test nurseries. Plant Dis. Rep. 47:657-661.

Dewey, W.G., and R.S. Albrechtsen. 1974. Effect of thiabendazole seed treatment on the incidence of dwarf bunt and on the yield of winter wheat. Plant Dis. Rep. 58:743-745.

Dewey, W.G., and J.A. Hoffmann. 1975. Susceptibility of barley to *Tilletia controversa*. Phytopathology 65:654-657.

Dhaliwal, H.S., A.S. Randhawa, K. Chand, and D. Singh. 1983. Primary infection and further development of Karnal bunt of wheat. Indian J. Agric. Sci. 53:239-244.

Dhaliwal, H.S., R. Navarrete-Maya, and J.C. Valdez. 1989. Scanning electron microscope studies of penetration mechanism of *Tilletia indica* in wheat spikes. Rev. Mexicana Fitopat. 7:150-155.

Dhiman, J.S. 1982. Epidemiology of Karnal bunt of wheat in Punjab. Ph.D. Thesis, Punjab Agricultural University, Ludhiana, India.

Dhitaphichit, P., P. Jones, and E.M. Keane. 1989. Nuclear and cytoplasmic gene control of resistance to loose smut (*Ustilago tritici* (Pers.) Rostr.) in wheat (*Triticum aestivum* L.). Theor. Appl. Genet. 78:897-903.

Dickson, J.G. 1956. Diseases of Field Crops. McGraw-Hill Book Company. London. 517 pp.

Diekmann, M. 1987. Monitoring the presence of Karnal bunt (*Tilletia indica*) in germplasm exchange at ICARDA. Phytopath. Medit. 26:59-60.

do Valle Ribeiro, M.A.M. 1963a. Investigations into the pathogenicity of certain races of *Ustilago nuda*. Trans. Brit. Mycol. Soc. 46:49-62.

do Valle Ribeiro, M.A.M. 1963b. The inheritance of the resistance of wheat to two races of *Ustilago nuda*. Trans. Brit. Mycol. Soc. 46:179-192.

Dupler, M., J.L. Smilanick, and J.A. Hoffmann. 1987. Effect of matric and osmotic potential on teliospore germination of *Tilletia indica*. Phytopathology 77:594-598.

Durán, R. 1968. Subterranean sporulation in two graminicolous smut fungi. Phytopathology 58:390.

Durán, R. 1972. Further aspects of teliospore germination in North American smut fungi. II. Can. J. Bot. 50:2569-2573.

Durán, R., and R. Cromarty. 1977. *Tilletia indica*: a heterothallic wheat bunt fungus with multiple alleles controlling incompatibility. Phytopathology 76:812-815.

Durán, R., and G.W. Fischer. 1956. Further studies on the taxonomy, identity, and host range of the dwarf bunt fungus. Phytopathology 46:11 (Abstr.).

Durán, R., and G.W. Fischer. 1961. The genus *Tilletia*. Wash. St. Univ. Press. 138 pp.

Efthimiadis, P. 1988. Myclobutanil seed treatment—a new approach to control cereal diseases. In pages 887-893, Brighton Crop Prot. Conf.—Pests and Diseases.

El-Helaly, A.F. 1948. The influence of cultural conditions on the flag smut of wheat. Phytopathology 38:688-697.

El-Khadem, M., R.A. Omar, A.H. Kamel, and S.A. Abou-Naga. 1980. Physiologic and pathogenic races of *Urocystis agropyri*. Phytopath. Z. 98:203-209.

El-Meleigi, M.A., A.A. El-Rokiabah, G.H. Ibrahim, S.M. El-Shair, and M.E. Al-Omir. 1993. Survey of wheat diseases in Central Saudi Arabia in 1987, 1988 and 1989. Phytopathology 83:1335 (Abstr.).

El-Zayat, M.M., M. El-Shater, and M.Y. Yousef. 1977. Studies of wheat diseases. In pages 61-63, First Seminar on Wheat Research Production in the Socialist People's Libyan Arab Jamahiriya, 17-19 April 1977. Agricultural Research Centre, Tripoli, Libya.

Emmett, R.W., and D.G. Parbery. 1975. Appressoria. Ann. Rev. Phytopath. 13:147-167.

Ettel, G.E., and W. Halbsguth. 1964. The effect of trimethylamine, calcium nitrate, and light on the germination of smut spores of *Tilletia tritici* (Bjerk.) Winter. Beiträge zur Biologie der Pflanzen. 39:451-488.

Ezzahiri, B., and M. Elyamani. 1992. Survey of fungal diseases of wheat and barley in Morocco in 1991. Report submitted to IAV, Hassan II. Rabat, Morocco. 24 pp.

Fernández, J.A., and R. Durán. 1978. Hypodermic inoculation: A rapid technique for producing dwarf bunt in wheat. Plant Dis. Rep. 62:336-337.

Fernández, J.A., R. Durán, and J.E. Schafer. 1978. Histological aspects of dwarf bunt resistance in wheat. Phytopathology 68:1417-1421.

Figueroa-Lopez, P., and T. Espinoza-Salazar. 1988. Tratamiento quimico con fungicidas a la semilla de trigo para el control del carbon parcial *Tilletia indica* Mitra en laboratorio. Informe tecnico del campo agricola experimental valle del Yaqui. CAEVY-CIANO-INIFAP. pp. 22.

Finci, S. 1981. Studies on the pathogenic races of *Tilletia foetida* and *T. caries* and their pathogenicity on some wheat varieties in Turkey. EPPO Bull. 11:77-82.

Finci, S., Y. Parlak, O. Bilgin, H. Gümüstekim, I. Aktuma, and M. Tunadeunir. 1983. Investigation on the distribution of the pathogenic races of wheat bunt [*Tilletia foetida* (Wallr.) Liro and *T. caries* (DC)Tul.] in Turkey. Bitli Koruma Büll. 23:124-147.

Fischer, G.W. 1952. *Tilletia brevifaciens*, sp. nov., causing dwarf bunt of wheat and certain grasses. Res. Stud. State Coll. Washington 20:11-14.

Fischer, G.W. 1953. Manual of North American Smut Fungi. New York. Ronald Press Co. 343 pp.

Fischer, G.W., and R. Durán. 1956a. Some new distribution and early incidence records of dwarf bunt of wheat and rye. Res. Stud. State Coll. Wash. 24:267-274.

Fischer, G.W., and R. Durán. 1956b. Some new records of dwarf bunt of wheat and rye. Phytopathology 46:12. (Abstr.).

Fischer, G.W., and C.S. Holton. 1943. Studies of the susceptibility of forage grasses to cereal smut fungi. IV. Cross-inoculation experiments with *Urocystis tritici*, *U. occulta*, and *U. agropyri*. Phytopathology 33:910-921.

Fischer, G.W., and C.S. Holton. 1957. Biology and control of the smut fungi. Ronald Press. Co. 662 pp.

Fischer, G.W., and L.J. Tyler. 1952. Is there an earlier record of dwarf bunt than 1931? Plant Dis. Rep. 36:445-447.

Flor, H.H. 1933. Studies on physiologic specialization in *Tilletia tritici* and *T. levis* in the Pacific Northwest. J. Agric. Res. 47:193-213.

Flor, H.H. 1947. Inheritance of reaction to rust in flax. J. Agric. Res. 74:241-262.

Foster, H.C., and A.J. Vasey. 1929. The relation between flag smut infection and manurial treatment. J. Dept. Agric. Victoria 27:321-330.

Freeman, E.M., and E.C. Johnson. 1909. The loose smuts of wheat and barley. USDA Bureau Plant Ind., Bull. 152. 48 pp.

Fuentes, S., W.C. James, E. Torres, C. Garcia, S. Delgado, and A. Elias-Calles. 1983. Karnal bunt of wheat: risk appraisal and evaluation of the efficacy of seed dressings to control seedborne inoculum. CIMMYT Report. 49 pp.

Fuentes, S., E. Torres, and C. Garcia-Dominguez. 1982. Estudios sobre el control del carbon parcial del trigo. Congreso de la Asociacion Colombiana de Fitopatologia (ASCOLFI), Cali, Colombia, July, 1982. pp. 19.

Fuentes-Davila, G. 1984. Biology of *Tilletia indica* Mitra. M.S. Thesis. Department of Plant Pathology, Washington State University. 66 pp.

Fuentes-Davila, G. 1989. Studies on the incompatibility system of *Tilletia indica*. Revista Mexicana Fitopatol. 7:201-207.

Fuentes-Davila, G., and R. Durán. 1986. *Tilletia indica*: cytology and teliospore formation *in vitro* and in immature kernels. Can. J. Bot. 64:1712-1719.

Fuentes-Davila, G., and D.A. Lawn. 1992. Effect of Methyl bromide and Dazomet on germination of *Tilletia indica* teliospores in field soil. Trop. Pest Manag. 38:323-324.

Fuentes-Davila, G., and S. Rajaram. 1994. Sources of resistance to *Tilletia indica* in wheat (*Triticum aestivum*). Crop Prot. 13:20-24.

Fuentes-Davila, G., and R. Rodríguez-Ramos. 1993. Fuentes de resistencia a *Tilletia indica* Mitra en genotipos derivados de *Triticum aestivum* y X *Triticosecale*. Rev. Fitotec. Mexicana 16:172-178.

Fuentes-Davila, G., S. Rajaram, W.H. Pfeiffer, and O. Abdalla. 1992. Results of artificial inoculation of the 4th Karnal Bunt Screening Nursery (KBSN). Ann. Wheat Newsl. 38:157-162.

Fuentes-Davila, G., S. Rajaram, and G. Singh. 1995. Inheritance of resistance to Karnal Bunt (*Tilletia indica* Mitra) in bread wheat (*Triticum aestivum* L.). Plant Breeding 114:250-252.

Gaines, E.F., and W.K. Smith. 1933. Reaction of varieties and hybrids of wheat to physiologic forms of bunt. J. Amer. Soc. Agron. 25:273-284.

Gair, R., J.I.I. Jenkins, and I. Lester. 1972. Cereal Pests and Diseases. Farming Press Limited, Ipswich, Sufflok, United Kingdom. 223 pp.

Gammie, R.L. 1994. Winter cereal variety sowing guide 1994. 44 pp. NSW Agriculture, Orange.

Gardner, J.S., J.V. Allen, W.M. Hess, and R.K. Tripathi. 1983a. Sheath structure of *Tilletia indica* teliospores. Mycologia 75:333-336.

Gardner, J.S., W.M. Hess, and R.K. Tripathi. 1983b. Surface rodlets of *Tilletia indica* teliospores. J. Bacteriol. 154:502-504.

Garrett, A.O. 1910. The smuts and rusts of Utah. Mycologia 2:265-304.

Gaskin, T.A., and J.F. Schafer. 1962. Some histological and genetic relationships of resistance of wheat to loose smut. Phytopathology. 52:602-607.

Gassner, G. 1938. Untersuchungen uber Keimgeschwindigkeit und Infektionsvermögen verschiedener Stamme von *Tilletia foetens* und *Tilletia tritici*. Phytopath. Z. 11:489-516.

Gassner, G., and H. Kichhoff. 1938. Einige abschliessende Versuche ueber die Wirkung der Warmbenetzungsbeize. Phytopath. Z. 11:115-120.

Gassner, G., and R. Niemann. 1954. Investigations of the relationship of temperature and light to spore germination of different *Tilletia*. Phytopath. Z. 21:367-394.

Gattani, M.L. 1964. Report to the Government of Afghanistan on Plant Diseases and Their Control. Report No. 1817. FAO, Rome. 24 pp.

Gaudet, D.A., and B.J. Puchalski. 1990. Influence of planting dates on the aggressiveness of common bunt races (Tilletia tritici and T. laevis) to Canadian spring wheat cultivars. Can. J. Plant Path. 12:204-208.

Gaudet, D.A., B.J. Puchalski, and T. Entz. 1989. Effect of environment on efficacy of seed-treatment fungicides for control of common bunt in spring and winter wheat. Pestic. Sci. 26:241-252.

Gaudet, D.A., B.J. Puchalski, and T. Entz. 1992. Application methods influencing the effectiveness of carboxin for control of common bunt caused by *Tilletia tritici* and *T. laevis* in spring wheat. Plant Dis. 76:64-66.

Gaunt, R.E., and J.G. Manners. 1971a. Host-parasite relations in loose smut of wheat. I. The effect of infection on host growth. Ann. Bot. 35:1131-1140.

Gaunt, R.E., and J.G. Manners. 1971b. III. The utilization of carbon 14 labelled assimilates. Ann. Bot. 35:1151-1161.

Gaunt, R.E., and J.G. Manners. 1973. The production of trehalose and polyols by *Ustilago nuda* in culture and their utilization in healthy and infected wheat plants. New Phytol. 72:321-327.

Gautam, P.L., S. Pal, S.K. Malik, and D.P. Saini. 1977. Note on reaction of promising wheat strains to Karnal bunt. Pantnagar J. Res. 2:228-229.

Geach, W.L. 1933. Foot and root rots of wheat in Australia. The influence of the combined action of *Fusarium culmorum* (W.G. Smith) Sacc. and *Urocystis tritici* Koern. on the occurrence of seedling blight. J. Commonwealth Sci. Ind. Res. 6:269:278.

Gera, S.D., and K.S. Vashisth. 1968. Studies on Indian cereal smuts. IX. Longevity of the spores of *Ustilago nuda tritici* Schaffn. Indian Phytopath. 21:447-449.

Ghafoor, A., and S.A.J. Khan. 1976. List of Diseases of Economic Plants in Pakistan. Dept. Plant Protection, Ministry of Food, Agriculture and Under Developed Areas, Pakistan. 85 pp.

Ghosheh, Z. 1975. Wheat improvement in Jordan. In pages 532-535, Proc. 2nd International Winter Wheat Conference, Zagreb, Yugoslavia, June 9-19, 1975. USDA, USAID, and Univ. of Nebrasaka, Lincoln, NE.

Gill, K.S. 1990. Genetic evaluation and utilization of wheat germplasm for breeding varieties resistant to Karnal bunt, Third Annual Research Report. Department of Plant Breeding, Punjab Agricultural University, Ludhiana, Punjab, India.

Gill, K.S., and S.S. Aujla. 1987. Breeding for Karnal Bunt resistance in wheat. Crop Imp. 14:109-118.

Gill, K.S., I. Sharma, and S.S. Aujla. 1993. Karnal bunt and wheat production. Punjab Agricultural University. 153 pp.

Goates, B.J. 1988. Histology of infection of wheat by *Tilletia indica*, the Karnal bunt pathogen. Phytopathology 78:1434-1441.

Goates, B.J. 1992. Control of dwarf bunt by seed treatment with Dividend 3FS, 1991. Fungi. and Nemati.Tests 47:264.

Goates, B.J., and J.A. Hoffmann. 1979. Somatic nuclear division in *Tilletia* species pathogenic on wheat. Phytopathology 69:592-98.

Goates, B.J., and J.A. Hoffmann. 1986. Formation and discharge of secondary sporidia of the bunt fungus, *Tilletia foetida*. Mycologia 78:371-379.

Goates, B.J., and J.A. Hoffmann. 1987. Nuclear behavior during teliospore germination and sporidial development in *Tilletia caries*, *T. foetida*, and *T. controversa*. Can. J. Bot. 65:512-517.

Goel, R.K., and J.S. Jhooty. 1984. Screening of wheat germplasm for resistance to flag smut. Indian J. Agric. Sci. 54:739-41.

Goel, R.K., and J.S. Jhooty. 1986. Germination behaviour of some teliospore collections of *Urocystis agropyri*. Indian J. Mycol. Plant Pathol. 16:146-150.

Goel, R.K. and J.S. Jhooty. 1987. Stimulation of germination in teliospores of *Urocystis agropyri* by volatiles from plant tissues. Ann. Appl. Biol. 111:295-300.

Goel, L.B., D.V. Sing, K.D. Srivastava, L.M. Joshi, and S. Nagarajan. 1977. Smuts and Bunts of Wheat in India. Bull. Division of Mycology and Plant Pathology, IARI, New Delhi, India. 38 pp.

Gopal, S., and K.S. Sekhon. 1988. Effect of Karnal bunt disease on the milling, rheological, and nutritional properties of wheat: effect on the quality and rheological properties of wheat. J. Food Sci. 53:1558-1559.

Gorlenko, M.V. 1946. Twenty-five years of cereal diseases in the United Socialistic Soviet Republics. Jour. Bot. USSU 31:3-17. Quoted by Dickson (1956).

Gorter, G.J.A.M. 1964a. Loose smut of wheat in South Africa: pathogenic races and reaction of locally grown cultivars. S. African J. Agric. Sci. 7:57-64.

Gorter, G.J.A.M. 1964b. Seedling crown node test as a means to rapid identification of pathogenic races of loose smut of wheat. S. African J. Agric. Sci. 7:397-404.

Gothwal, B.D. 1972a. Proper stage for the inoculation of wheat plants with loose smut pathogen. Indian J. Mycol. Plant Pathol. 2:89.

Gothwal, B.D. 1972b. Effect of loose smut on the growth and morphology of spring wheats. Indian J. Mycol. Plant Pathol. 2:171.

Graham, S.O. 1959. The effects of various reagents, mounting media, and dyes on the teliospores walls of *Tilletia contraversa* Kühn. Mycologia 51:477-491.

Grasso, V. 1968. Gli Ustilaginali dei cereali in Italia (1938-1968). Ann. D. Facolta Agraria D. Universita di Bari 22:239-308. Bari, Italy.

Greenhalgh, F.C., and J.S. Brown. 1984. A method for determining the reactions of wheat breeding lines to flag smut. Austral. Plant Path. 13:36-38.

Grevel, F.K. 1930. Untersuchungen ueber das Vorhandensein biologischer Rassen des Flugbrandes des Weizens (*Ustilago tritici*). Phytopathol. Z. 2:209-234.

Grey, W.E., D.E. Mathre, J.A. Hoffmann, R.L. Powelson, and J.A. Fernández. 1986. Importance of seedborne *Tilletia controversa* for infection of winter wheat and its relationship to international commerce. Plant Dis. 70:122-125.

Griffith, R.B., F.P. Zscheile, and J.W. Oswald. 1955. The influence of certain environmental factors on expression of resistance to bunt in wheat. Phytopathology 45:428-434.

Griffiths, D. 1907. Concerning some west American fungi. Bull. Torrey Bot. Club 34:207-211.

Griffiths, M. 1924. Experiments with flag smut of wheat and the casual fungus, *Urocystis tritici* Kcke. J. Agric. Res. 27:425-449.

Gupta, A.K., R.K. Grewal, and R.K. Goel. 1977. A quick screening method for identifying wheat varieties resistant to loose smut (*Ustilago nuda tritici*). Cereal Res. Commun. 5:261-264.

Gupta, A.K., K.L. Bajaj, and R.K. Grewal. 1979. *In vitro* detection of resistance to loose smut and its relationship to polyphenols. In pages 1098-1101, Proc. V. Intern. Wheat Genet. Symp.

Hafiz, A. 1951. Physiologic specialization in *Urocystis tritici* Koern. Phytopathology 41:809-812.

Hafiz, A. 1986. Plant Diseases. Pakistan Agricultural Research Council, Islamabad. 552 pp.

Hall, W.E. 1975. Trip Report. Development Assistance Corp. 18-22 July 1975, Vaduz, Liechtenstein. 2 pp.

Hamblin, C.O. 1921. Flag smut and its control. Agric. Gaz. N.S.W. 32:23.

Hanna, W.F. 1938. The association of bunt with loose smut and ergot. Phytopathology 28:142-146.

Hansen, F. 1958. Anatomical investigations on the penetration and extension of *Tilletia* species in cereals in relation to the stage of development of the host plant. Phytopath. Z. 34:169-208.

Hansing, E.D. 1967. Systemic oxathiin fungicides for control of loose smut (*Ustilago tritici*) of winter wheat. Phytopathology 57:814 (Abstr.).

Hardison, J.R., J.P. Meiners, J.A. Hoffmann, and J.T. Waldher. 1959. Susceptibility of gramineae to *Tilletia contraversa*. Mycologia 51:656-664.

Harwood, P.M., and P.G. Holden. 1892. Smut in oats and wheat: Jensen or hot water treatment. Michigan Agric. Expt. Sta. Bull. 87.

Hashmi, N.I., and Ch. Ghulam Ahmed. 1988. Status of wheat production research, infrastructure, and constraints in Pakistan. In pages 220-227, J.P. Srivastava et al., eds., Proc. Winter Cereals and Food Legumes in Mountainous Areas. ICARDA 136 En. Aleppo, Syria.

Hassan, S.F. 1968. Situation of wheat diseases in Pakistan during 1966-67. FAO Information Bulletin—Near East Wheat and Barley Project. 5:17-18.

Hassan, S.F. 1970. Cereal disease situation in Pakistan. In pages 255-258, Proceedings of Third FAO/Rockefeller Foundations Wheat Seminar, Ankara, Turkey, 29 Apirl-13 May 1970. FAO, AGP:FC/20, Rome.

Hassan, S.F. 1972. Wheat disease situation in Pakistan during 1970 and 1971. FAO Information Bulletin—Near East Wheat and Barley Project. 9:9-11. FAO, Rome.

Hassan, S.F. 1974. Wheat diseases and their relevance to improvement and production in Pakistan. In pages 284-286, Proceedings of Fourth FAO/Rockefeller Foundation Wheat Seminar, Tehran, Iran, 21 May-2 June 1973. FAO, AGP:FC21, Rome.

Hassan, S.F., M. Hussain, and S.A. Rizvi. 1979. Wheat disease situation in Pakistan. In pages 231-234, Proc. Wheat Research and Production in Pakistan, August 6-9, 1979. Pakistan Agric. Res. Council, Islamabad.

Hawkesworth, D.L., B.C. Sutton, and G.C. Ainsworth. 1983. Ainsworth & Bisby's Dictionary of the Fungi. Commonwealth Mycological Institute, Kew Surrey. Seventh Edition. 445 pp.

He, Z., and T. Chen. 1991. Wheat and Wheat Breeding in China. Wheat Special Report No. 2. Mexico, D.F.: CIMMYT. 14 pp.

Heinrich, J. 1970. Studies of genetic resistance of wheat to loose smut, *Ustilago tritici* (Pers.) Rostr. Hod. Rosl. Aklim. Nasienn. 14:393-404.

Hellman, R., and B.J. Christ. 1991. Isozyme variation in *Ustilago hordei*. Phytopathology. 81:1207. (Abstr.).

Helm, J.H., and R.E. Allan. 1971. Inheritance of reaction to flag smut in wheat (*Triticum aestivum*) Crop Sci. 11:177-180.

Hendry, G.W., and H.N. Hansen. 1934. The antiquity of *Puccinia graminis* and *Ustilago tritici* in California. Phytopathology 24:1313-1314.

Hermis, Y.A., and S.A.A. Hussain 1979. Constraints to cereal production and possible solutions in Iraq. In pages 69-78, The Gap between: Present Farm Yield and the Potential. Proc., Fifth Cereals Workshop, Vol. I, Algiers, Algeria, May 5-9, 1979.

Hess, W.M., and J.S. Gardner. 1983. Development and nature of the partition layer in *Tilletia caries* teliospore walls. J. Bacteriol. 154:499-501.

Hess, W.M., and E.J. Trione. 1986. Use of electron microscopy to characterize teliospores of *Tilletia caries* and *T. controversa*. Plant Dis. 70:458-460.

Heyne, E.G., and E.D. Hansing. 1955. Inheritance of resistance to loose smut of wheat in the crosses of Kawvale x Clarkan. Phytopathology 45:8-10.

Hindorf, H., H.C. Weltzien, and F. Klingauf. 1978. Phytomedizinische Erhebungen in der Arabischen Republik Jemen. Bamberger Geogr. Schriften 1:139-147.

Hoffmann, J.A. 1971. Control of common and dwarf bunt of wheat with systemic fungicides. Plant Dis. Rep. 55:1132-1135.

Hoffmann, J.A. 1981. Chemical seed treatments for controlling seedborne and soilborne common bunt of wheat. Plant Dis. 65:256-259.

Hoffmann, J.A. 1982. Bunt of wheat. Plant Dis. 66:979-987.

Hoffmann, J.A., and B.J. Goates. 1981. Spring dormancy of *Tilletia controversa* teliospores. Phytopathology 71:881. (Abstr.).

Hoffmann, J.A., and R.J. Metzger. 1976. Current status of virulence genes and pathogenic races of the wheat bunt fungi in the northwestern USA. Phytopathology 66:657-660.

Hoffmann, J.A., and L.H. Purdy. 1967. Effect of stage of development of winter wheat on infection by *Tilletia controversa*. Phytopathology 57:410-413.

Hoffmann, J.A., and D.V. Sisson. 1987. Evaluation of bitertanol and thiabendazole seed treatment and PCNB soil treatment for control of dwarf bunt of wheat. Plant Dis. 71:839-841.

Hoffmann, J.A., and J.T. Waldher. 1964. Additions to the host range of dwarf bunt. Plant Dis. Rep. 48:575-577.

Hoffmann, J.A., and J.T. Waldher. 1981. Chemical seed treatments for controlling seedborne and soilborne common bunt of wheat. Plant Dis. 65:256-259.

Hoffmann, J.A., W.G. Dewey, J.E. Call, and S.M. Rine. 1983. Systemic fungicides for control of dwarf bunt of wheat. I. Seed treatment. Plant Dis. 67:294-297.

Holton, C.S. 1941. Flag smut of wheat in Washington. Plant Dis. Rep. 25:335-336.

Holton, C.S. 1942. Inheritance of chlamydospore and sorus characters in species and race hybrids of *Tilletia caries* and *T. foetida*. Phytopathology 34:586-592.

Holton, C.S. 1943. Chlamydospore germination in the fungus causing dwarf bunt of wheat. Phytopathology 33:732-735.

Holton, C.S. 1949. Observations on *Neovossia indica*. Indian Phytopath. 2:1-5.

Holton, C.S. 1954a. Genetic phenomena in the smut fungi as related to the dynamics of the species. Phytopathology 44:352-355.

Holton, C.S. 1954b. Natural hybridization between common and dwarf bunt as related to the problem of delimitation of species of *Tilletia* occurring on wheat. Phytopathology 44:493. (Abstr.).

Holton, C.S. 1967. Smuts. In pages 337-353, K.S. Quisenberry and L.P. Reitz, eds., Wheat and Wheat Improvement. Agronomy Monograph 13. Amer. Soc. of Agron., Madison, WI, USA.

Holton, C.S., and F.D. Heald. 1941. Bunt or Stinking Smut of Wheat (A World Problem). Burgess, Minneapolis, MN. 211 pp.

Holton, C.S., and T.L. Jackson. 1951. Varietal reaction to dwarf bunt and flag smut, and the occurrence of both in the same wheat plant. Phytopathology 41:1035-1036.

Holton, C.S., and A.G. Johnson. 1943. Physiologic races of *Urocystis tritici*. Phytopathology 33:169-171.

Holton, C.S., and E.L. Kendrick. 1956. Problems on the delimitation of species of Tilletia occurring on wheat. Res. Stud. State Coll. Washinton 24:318-325.

Holton, C.S., and L.H. Purdy. 1954. Control of soilborne common bunt of winter wheat in the Pacific Northwest by seed treatment. Plant Dis. Rep. 38:753-754.

Holton, C.S., and D.A. Vogel. 1952. Evidence of pathogenic specialization in dwarf bunt of winter wheat in the Pacific Northwest. Plant Dis. Rep. 36:434.

Holton, C.S., R.H. Bamberg, and R.W. Woodward. 1949a. Progress in the study of dwarf bunt of winter wheat in the Pacific Northwest. Phytopathology 39:986-1000.

Holton, C.S., R.J. Bamberg, and R.W. Woodward. 1949b. Tester races for dwarf bunt resistance factors in winter wheat varieties. Agron. J. 41:250-252.

Holton, C.S., J.A. Hoffmann, and R. Durán. 1968. Variation in the smut fungi. Ann. Rev. Phytopath. 6:213-242.

Hori, S. 1907. Seed infection by smut fungi of cereals. Bull. Imp. Cent. Agr. Expt. Sta. I. 2:163. Quoted by Miller and Millikan (1934a).

Hulluka, M., G. Woldeab, Y. Andnew, R. Desta, and A. Badebo. 1991. Wheat pathology research in Ethiopia. In pages 173-217, H. Gebre-Mariam, D.G. Tanner, and M. Hulluka. Wheat Research in Ethiopia: An Historical Perspective IAR/CIMMYT, Addis Ababa, Ethiopia.

Humphrey, H.B., and A.G. Johnson. 1919. Take-all and flag smut, two wheat diseases new to the United States. U.S.D.A. Farmers' Bull. 1063 :1-8.

Humphrey, H.B., and V.F. Tapke. 1925. The loose smut of rye (*Ustilago tritici*). Phytopathology 15:598-606.

Hussain, M., M. Sharif, M. Ullah, and M. Sarwar. 1988. Studies on the feasible use of Karnal bunt infected wheat for bread and "chapati" making. Proc. 1st Nat. Food Proc. Workshop. Lahore, India, June 1988.

Ibrahim, I.F. 1988. A new record of a hybrid between *Tilletia caries* and *T. foetida* in Iraq. Indian Phytopath. 41:155-156.

Ibrahim, I.F., K.M. Tamimi, H.A. Hadwan, A.H. Mahmood, and H.O. Haidar. 1985. Response of cultivated wheat to local collections of common bunt fungus in Iraq. Indian Phytopath. 38:569-571.

Iren. 1981. Wheat Diseases in Turkey. EPPO Bull. 11:47-52.

Jamil Khan, S.A., Khanzada, A.K. and M. Aslam. 1992. Effect of load of inoculum on the development of complete bunt disease on wheat under field conditons. Pakistan J. Agric. Res. 13:98-100.

Jarrett, P.H. 1932. Investigations on flag smut on wheat. J. Council Sci. and Ind. Res. 5:1-6.

Jensen, J.L. 1888. Om Kornsorterners Brand. Copenhagen.

Johnson, A.G. 1959. Further studies of physiologic races in *Urocystis tritici*. Phytopathology 49:299-302.

Johnson, V.A., and H.L. Beemer Jr., eds. 1977. Wheat in the People's Republic of China. A Trip Report of the American Wheat Studies Delegation. CSCPRC Report No. 6. Nati. Acad. Sci. Washington D.C. 190 pp.

Johnsson, L. 1991a. Survey of common bunt (*Tilletia caries* (DC) Tul.) in winter wheat during the period 1967-1987 in Sweden. Zeit. Pflanzenkrank. Pflanzensch. 98:67-72.

Johnsson, L. 1991b. The nomenclature concerning *Tilletia contraversa* Kühn. Zeit. Pflanzenkrank. Pflanzensch. 98:627-629.

Jones, G.D., and B.C. Clifford. 1978. Cereal Diseases: Their Pathology and Control. BASF United Kingdom Limited, Agrochemical Division, Ipswich, Suffolk. 279 pp.

Joshi, L.M., B.L. Renfro, E.E. Saari, R.D. Wilcoxson, and S.P. Raychaudhuri. 1970. Rust and smut diseases of wheat in India. Plant Dis. Rep. 54:391-394.

Joshi, L.M., K.D. Srivastava, D.V. Singh, L.B. Goel, and S. Nagarajan. 1978. Annotated Compendium of Wheat Diseases in India. Indian Council of Agricultural Research, New Delhi. 332 pp.

Joshi, L.M., D.V. Singh, K.D. Srivastava, and R.D. Wilcoxson. 1983. Karnal bunt: A minor disease that is now a threat to wheat. Bot. Rev. 49:309-330.

Joshi, L.M., D.V. Singh, and K.D. Srivastava. 1986. Wheat and Wheat Disease in India. In pages 1-19, L.M. Joshi, D.V. Singh, and K.D. Srivastava, eds., Problems and Progress of Wheat Pathology in South Asia. Malhotra Publishing House, New Delhi.

Kamal, M., and A.A. Al Agbari. 1985. Manual of Plant Diseases in Yeman Arab Republic. Agric. Res. Authority, Min. Agric. and Fischeries, YAR. Precision Press, London. 75 pp.

Kamel, A.H., M. Harrabi, M. Deghaies, H. Halila, and M. Ben Salah. 1987. Wheat and barley diseases in Tunisia. Rachis 6:24-29.

Kanehira, T., and M. Shinohara. 1986. Comparison of electrophoretic patterns of enzymes from *Ustilago* spp. parasitic on barley, wheat and oats. Ann. Phytopath. Soc. Japan 52:660-668.

Kassam, A.H. 1988. Some agroclimatic characteristics of high-elevation areas in North Africa, West Asia and Southeast Asia. In pages 1-32, J.P. Srivastava, M.C. Saxena, S. Varma, and M. Tahir, eds., Winter Cereals and Food Legumes in Mountainous Areas. ICARDA, Aleppo, Syria.

Kavanagh, T. 1961a. Inoculating barley seedlings with *Ustilago nuda*, and wheat seedlings with *U. tritici*. Phytopathology 51:175-177.

Kavanagh, T. 1961b. Temperature in relation to loose smut in barley and wheat. Phytopathology 51:189-193.

Kawchuk, L.M., and J. Nielsen. 1987. Improved bunt inoculation of wheat utilizing adventitious shoots. Can. J. Bot. 65:1284-1285.

Kawchuk, L.M., W.K. Kim, and J. Nielsen. 1988. A comparison of polypeptides from the wheat bunt fungi *Tilletia laevis*, *T. tritici*, and *T. controversa*. Can. J. Bot. 66:2367-2376.

Kellerman, W.A., and W.T. Swingle. 1889. Report on the loose smuts of cereals. Kansas Agric. Exp. Sta. Manhattan Ann. Rpt., pp. 213-288.

Kendrick, E.L., and C.S. Holton. 1961. Racial population dynamics in *Tilletia caries* and *T. foetida* as influenced by wheat varietal populations in the Pacific Northwest. Plant Dis. Rep. 45:5-9.

Kendrick, E.L., R.G. Metzger, and C.R. Rhode. 1964. Overwintering of race T-18 of *Tilletia caries*. Plant Dis. Rep. 48:379-380.

Khan, M.A., M.S. Mirza, S.J. Hamid, and C.K. Khokhar. 1984. Physiologic races in flag smut of wheats. Pakistan J. Agric. Res. 5:172-174.

Khanna, A., and M.M. Payak. 1968. Teliospore morphology of some smut fungi. II. Light microscopy. Mycologia 60(3):655-662.

Khanna, A., M.M. Payak, and S.C. Mehta. 1966. Teliospore morphology of some smut fungi. I. Electron microscopy. Mycologia 58(4):562-569.

Khanzada, A.K., and M. Aslam. 1982. Reaction of wheat varieties to loose smut. National Seminar on Wheat Research and Production. Pakistan Council of Agricuture Reseach, Islamabad.

Khanzada, A.K., W.J. Rennie, S.B. Mathur, and P. Neergaard. 1980. Evaluation of two routine embryo test procedures for assessing the incidence of loose smut infection in seed samples of wheat (*Triticum aestivum*). Seed Sci. Technol. 8:363-370.

Khatib, H., P. Davet, and G. Sardy. 1970. Liste des maladies des plantes au Liban. Inst. Rec. Agron. Lab. de Phytopathologie, Fanar. Lebanon. 41 pp.

Khazra, H., and A. Bamdadian. 1974. The wheat disease situation in Iran. In pages 292-299, Proc. Fourth FAO/Rockefeller Foundation Wheat Seminar, Tehran, Iran, 21 May-2 June 1973. FAO, Rome.

Khetarpal, R.K., V.K. Agarwal, and K.P.S. Chauhun. 1980. Studies on the influence of weather conditions on the incidence of black-point and karnal bunt of triticale. Seed Res. 8:108-110.

Kim, W.K., N.K. Howes, and R. Rohringer. 1982. Detergent-soluble polypeptides in germinated uredospores and differentiated uredosporelings of wheat stem rust. Can. J. Plant Path. 4:328-333.

Kim, W.K., R. Rohringer, and J. Nielsen. 1984. Comparison of polypeptides in *Ustilago* spp. pathogenic on wheat, barley and oats: a chemotaxonomic study. Can. J. Bot. 62:1431-1437.

Kiryukhina, R.I., and L.I. Shcherbakova. 1976. On the viability of the pathogen of Indian smut of wheat. Zashchita Rastenii 7:50. (Abstr.).

Klushnikova, E.S. 1928. Distribution of the mycelium of *Ustilago tritici* in the tissues of the host plant, and anatomical changes caused by it. Bol. Rast. 17:1-25.

Knox, R.E., and N.K. Howes. 1994. A monoclonal antibody chromosome marker anaysis used to locate a loose smut resistance gene in wheat chromosome 6A. Theor. Appl. Genet. 89:787-793.

Kollmorgen, J.F., and D.J. Ballinger. 1987. Detection and morphology of hyphae of common bunt fungi *Tilletia laevis* and *T. tritici* in wheat seedlings. Trans. Brit. Mycol. Soc. 88:555-559.

Kollmorgen, J.R., W.M. Hess, and E.J. Trione. 1979. Ultrastructure of primary sporidia of a wheat bunt fungus *Tilletia caries* during ontogeny and mating. Protoplasma 99:189-202.

Kollmorgen, J.F., J.V. Allen, W.M. Hess, and E.J. Trione. 1978. Morphology of primary sporidial development in *Tilletia caries*. Trans. Br. Mycol. Soc. 71:223-229.

Körnicke, F. 1877. Mykologische Beitrage. Hedwigia 16:33-40. Quoted by Purdy (1965).

Krishna, A., and R.A. Singh. 1981. Aberrations in the teliospore germination of *Neovossia indica*. Indian Phytopath. 34:260-262.

Krishna, A., and R.A. Singh. 1982a. Effect of physical factors and chemicals on the teliospore germination of *Neovossia indica*. Indian Phytopath. 35:448-455.

Krishna, A., and R.A. Singh. 1982b. Evaluation of fungicides for the control of Karnal bunt of wheat. Pesticides 16:7.

Krishna, A., and R.A. Singh. 1982c. Investigation on the disease cycle of Karnal bunt of wheat. Indian J. Mycol. Plant Path. 12:124.

Krishna, A., and R.A. Singh. 1983a. Longevity of teliospores of *Neovossia indica* causing Karnal bunt of wheat. Indian J. Mycol. Plant Path. 13:97-98.

Krishna, A., and R.A. Singh. 1983b. Effect of some organic compounds on teliospore germination and screening of fungicides against *Neovossia indica*. Indian Phytopath. 36:233-236.

Krishna, A., and R.A. Singh. 1983c. Method of artificial inoculation and reaction of wheat cultivars to Karnal Bunt. Indian. J. Mycol. Plant Path. 13:124-125.

Krivchenko, V.I. 1984. Resistance of the small grains to smut diseases. Kolos Publ., Moscow. 304 pp.

Kühn, J. 1874. *Tilletia contraversa*. In Rabenhorst, Fungi Euroaei. Hedeigia 13:188-189.

Kuiper, J. 1968. Experiments on the control of cereal smuts with a derivative of 1,4-oxathiin. Austral. J. Exper. Agric. and Anim. Husb. 8:232-237.

Kuiper, J., and G.M. Murray. 1978. Spoilage of grain by fungi. Agric. Gaz. N.S.W. 89:39-40.

Lambat, A.K., R. Nath, M.A. Majumdar, I. Rani, P. Kaur, J.L. Varshney, P.C. Agrawal, R.K. Khetarpal, and U. Dev. 1983. International spread of Karnal bunt of wheat. Phytopath. Medit. 22:213-214.

Langdon, R.F.N., J.F. Kollmorgen, and J. Walker. 1976. The nomenclature of the smuts of wheat, barley, oats, rye, and certain grasses. Austral. Plant Path. Soc. Newsl. 5:52-54.

Larose, E., Jaques, A., and J. Lounsky. 1946. The control of cereal loose smut (*Ustilago nuda* Jensen and *U. nuda* tritici Schaff.) by seed disinfection with hot water. Bull. Inst. Agron. Gembloux 15:3-18.

Leppik, E.E. 1970. Gene centers of plants as sources of disease resistance. Ann. Rev. Phytopath. 8:323-244.

Limbourn, E.J. 1931. Flag smut of wheat. Variety resistance tests 1926 to 1930. J. Dept. Agric., Western Australia Ser. 2, 5:453-457.

Lindquist, J.C., A.A. Sarasola, R. Saravi Cisneros, and J.M. Carranza. 1954. La Caries enana del trigo *Tilletia controversa* en la Argentina y el Uruguay. Rev. Fac. Agron. Eva Peron. 30:231-243.

Line, R.F. 1972. Chemical control of flag smut of wheat. Plant Dis. Rep. 56:636-640.

Line, R.F. 1993a. Dwarf bunt (*Tilletia controversa*). In pages 23-29, S.B. Mathur and B.M. Cunfer, eds., Seedborne Diseases and Seed Health Testing of Wheat. Institute of Seed Pathology for Developing Countries. Danish Government. ABC Grafik, Fredeirksberg, Denmark.

Line, R.F. 1993b. Common bunt (*Tilletia tritici* and *Tilletia laevis*). In pages 45-52, S.B. Mathur and B.M. Cunfer, eds., Seedborne Diseases and Seed Health Testing of Wheat. Institute of Seed Pathology for Developing Countries. Danish Government. ABC Grafik, Fredeirksberg, Denmark.

Line, R.F. 1993c. Flag Smut (*Urocystis agropyri*). In pages 53-57, S.B. Mathur and B.M. Cunfer, eds., Seedborne Diseases and Seed Health Testing of Wheat. Institute of Seed Pathology for Developing Countries. Danish Government. ABC Grafik, Fredeirksberg, Denmark.

Line, R.F., and R.B. Scott. 1987. Wheat (*Triticum aestivum* 'Paha') Flag smut; *Urocystis agropyri*. Fungic. Nematic. Tests 42:146-147.

Lira Ibarra, M. 1992. Karnal bunt (*Tilletia indica*) of wheat in the State of Sonora, Mexico. In pages 6-9, G. Fuentes-Davila and G.P. Hettel, eds., Update on Karnal Bunt Research in Mexico. Wheat Special Report No. 7a. CIMMYT, Mexico D.F.

Locke, C.M., and A.J. Watson. 1955. Foreign plant diseases intercepted in quarantine inspections. Plant Dis. Rep. 39:518.

Lopez-Lugo, F. 1986. Efecto de los factores climaticos en la incidencia de carbon parcial del trigo en cuatro fechas de siembra en el Valle del Yaqui. Ciclo otono-invierno 1985-86. Reporte interno CAEVY-CIANO, Sonora, Mexico.

Loria, R., Wiese, M., and A.L. Jones. 1982. Effects of free moisture, head development and embryo accessibility on infection of wheat by *Ustilago tritici*. Phytopathology 72:1270-1272.

Loughman, R. 1989. Chemical control in Western Australia of soilborne flag smut caused by *Urocystis agropyri*. Austral. Plant Path. 18:94-95.

Lowther, C.V. 1950. Chlamydospore germination in physiologic races of *Tilletia caries* and *T. foetida*. Phytopathology 40:590-603.

Lupton, F.G.H., and N.F. Derera. 1981. Soft wheat breeding in Europe and Australia. In pages 99-127, W.T. Yamazaki and C.T. Greenwood, eds., Soft Wheat: Production, Breeding, Milling and Uses. Amer. Assoc. of Cereal Chemists Inc., St. Paul, MN.

Luthra, J.C. 1953. Solar energy treatment of wheat loose smut (*Ustilago tritici* (Pers.) Rostr.). Indian Phytopath. 6:49-56.

Lyamani, A. 1990. Fungal foliar diseases of wheat and barley in Morocco. In pages 281-283, Proc. 8th Cong. Medit. Phytopath. Union, October 28-November 3, 1990. Agadir, Morocco.

Maddox, F. 1896. Smut and bunt. Agric. Gaz. Tasmania 4:92-95.

Mamluk, O.F. 1992. Seedborne diseases of wheat and barley. In pages 40-47, Proc. Workshop on Quarantine for Seed in the Near East Region. FAO/ICARDA/DGISP, 2-9 November 1991. Aleppo, Syria.

Mamluk, O.F., and A. Zahour. 1993. Differential distribution and prevalence of *Tilletia foetida* (Wallr.) Liro and *T. caries* (DC) Tul. on bread wheat and durum wheat. Phytopath. Medit. 32:25-32.

Mamluk, O.F., W.I. Abu Gharbieh, C.G. Shaw, A. Al-Musa, and L. Al-Banna. 1984. A Check List of Plant Diseases in Jordan. University of Jordan. Amman, Jordan. 107 pp.

Mamluk, O.F., M.P. Haware, K.M. Makkouk, and S.B. Hanounik. 1989. Occurrence, losses, and control of important cereal and food legume diseases in West Asia and North Africa. In pages 131-140, Proc. 22nd Inter. Symp. on Tropical Agric. Res., 25-27 August 1988. Kyoto, Japan.

Mamluk, O.F., M. Al Ahmed, and M.A. Makki. 1990. Current status of wheat diseases in Syria. Phytopath. Medit. 29:143-150.

Mamluk, O.F., O. Tahhan, R.H. Miller, B. Bayaa, K.M. Makkouk, and S.B. Hannounik. 1992. A checklist of cereal, food legume, and pasture and forage crop diseases and insects in Syria. Arab J. Plant Prot. 10:166-225.

Mantle, P.G. 1961a. Further observations on the reaction of wheat varieties to physiologic races of loose smut. Ann. Appl. Biol. 49:510-514.

Mantle, P.G. 1961b. The effect of vernalization on embryos of field-resistant wheat varieties infected by loose smut (*Ustilago tritici*). Ann. Appl. Biol. 49:692-703.

Mantle, P.G. 1961c. Further observations on an abnormal reaction of wheat to loose smut. Trans. Brit. Mycol. Soc. 44:529-545.

Mantle, P.G. 1961d. Persistence of loose smut infection within the growing point region of susceptible wheat varieties. Trans. Brit. Mycol. Soc. 44:546-548.

Mantle, P.G. 1962. Competitive association of loose smut and bunt in wheat. Trans. Brit. Mycol. Soc. 45:75-80.

Mardoukhi, W. 1989. Laboratory and farm methods of distinguishing *Tilletia controversa*, cause of dwarf bunt and its control. In pages 64-67, Abstracts, Cereal Research Seminar. Seed and Plant Improvement Institute, 14-19 January 1989. Iran Agric. College Tehran Univ., Plant Pests and Dis. Res. Inst. and Water Soil Res. Inst. Karaj, Iran (English and Farsi).

Martens, J., W.W.L. Seaman, and T.H. Atkinson. 1984. Diseases of Field Crops in Canada. Canadian Phytopath. Soc., Ottawa, Canada.

Martin, H.E. 1959. The scientific principles of crop protection. Edward Arnold, London. 359 pp.

Martin, H.E. 1970. Plant Diseases and Fungi Recorded in Saudi Arabia 1966-1970. Ministry of Agriculture & Water Research and Development Department Plant Protection Division. Riad, Saudi Arabia. 10 pp.

Mather, S.C., and E.D. Hansing. 1960. Root and shoot development of wheat infected with loose smut, *Ustilago tritici*. Phytopathology 50:645 (Abstr.).

Mathre, D.E., and R.H. Johnston. 1976. Presence of dwarf and common smut in Montana wheat. Plant Dis. Rep. 60:580-583.

Mathur, R.S. 1968. The fungi and plant diseases of Iraq. Published by the author. Page 34.

Mathur, S.B., and B.M. Cunfer, eds., 1993. Seedborne Diseases and Seed Health Testing of Wheat. Institute of Seed Pathology for Developing Countries. Danish Government. ABC Grafik, Fredeirksberg, Denmark. 168 pp.

Mathur, S.C., and S. Ram. 1963. Longevity of chlamydospores of *Neovossia indica* (Mitra) Mundkur. Sci. and Culture 29:411-412.

Mazahreh E. 1981. Covered Smut of Wheat in Jordan, Causal Agents and Losses. Fac. Agric., University of Jordan. Amman, Jordan. 14 pp.

McAlpine, D. 1905. Flag smut of wheat (*U. occulta*). J. Dept. Agric., Victoria 3:168-169.

McAlpine, D. 1906. Effect of formalin and bluestone on the germination of seed wheat. Agric. Gaz. N.S.W. 17:423-439.

McAlpine, D. 1910. The smuts of Australia, their structure, life history, treatment, and classification. Dept. Agric. Victoria, Melbourne. 288 pp.

McDairmid, R.W. 1912. Field experiments with wheat, 1911. Agric. Gaz. N.S.W. 23:650-652.

Macindoe, S.L., and C. Walkden Brown. 1968. Wheat breeding and varieties in Australia. Sci. Bull. 76, N.S.W. Dept. Agric., Third edition. 255 pp.

McIntosh, R.A. 1968. Genetic and cytogenetic studies on resistance to flag smut in wheat. Ph.D. Thesis. The University of Sydney. 184 pp.

McIntosh, R.A. 1983. A catalogue of gene symbols for wheat. In page 1226, Proc. 6th Intern. Wheat Genet. Symp., Kyoto, Japan.

McMillan, J.R.A. 1935. The unreliability of selection in the F2 for breeding wheat resistant to flag smut. J. Council Sci. Ind. Res. 8:214-22.

Medina, C. L. 1985. Efecto de diferentes niveles de infección con carbón parcial en la calidad de trigo y las características organolépticas del pan. Tesis de Licenciatura. Departamento de Química e Ingeniería Química, Instituto Tecnológico de Sonora, Ciudad Obregón, Sonora, México. pp. 63.

Meeta, M., J.S. Dhiman, P.S. Bedi, and M.S. Kang. 1980. Incidence and pattern of "Karnal" Bunt symptoms on some triticale varieties under adaptive research trial in the Punjab. Indian J. Mycol. Plant Path. 10:Lxxxiv.

Mehdi, V., L.M. Joshi, and Y.P. Abrol. 1973. Studies on chapati quality VI. Effect of wheat grains with bunts on the quality of chapaties. Bull. Grain Tech. 11:195-197.

Mehta, Y.R. 1993. Manejo Integrado de Enfermedades del Trigo. CIAT, Santa Cruz de la Sierra, Boliva. 314 pgs.

Meiners, J.P. 1959. Methods of infecting wheat with the dwarf bunt fungus. Phytopathology 49:4-8.

Meiners, J.P., and J.T. Waldher. 1959. Factors affecting spore germination of twelve species of *Tilletia* from cereals and grasses. Phytopathology 49:724-728.

Meiners, J.P., E.L. Kendrick, and C.S. Holton. 1956. Depth of seeding as a factor in the incidence of dwarf bunt and its possible relationship to spore germination on or near the soil surface. Plant Dis. Rep. 40:242-243.

Melchers, L.E. 1938. Smuts of cereal and forage crops in Kansas and their control. Kansas Agric. Exp. Stat. Bull. 279:37.

Metcalfe, P.B., and J.F. Brown. 1969. Evaluation of nine fungicides in controlling flag smut of wheat. Plant Dis. Rep. 53:631-633.

Metzger, R.J. 1970. Wheat genetics. Wheat Newsl. 17:122-125.

Metzger, R.J., and J.A. Hoffmann. 1978. New races of common bunt useful to determine resistance of wheat cultivars to dwarf bunt. Crop Sci. 18:49-51.

Metzger, R.J., and B.A. Silbaugh. 1971. A new factor for resistance to common bunt in hexaploid wheats. Crop Sci. 11:66-69.

Metzger, R.J., C.R. Rhode, and J.A. Hoffmann. 1977. Inheritance of resistance to common bunt in *Triticum aestivum* L. 'PI 178383'. Agron. Abstr., p. 64.

Metzger, R.J., C.R. Rhode, and E.J. Trione. 1963. Inheritance of genetic factors which condition resistance of the wheat variety Columbia to selected races of smut, *Tilletia caries*, and their association with red glumes. Agron. Abstr., p. 85.

Metzger, R.J., C.W. Schaller, and C.R. Rhode. 1979. Inheritance of resistance to common bunt in wheat, CI 7090. Crop Sci. 19:309-312.

Michalikova, A. 1970. The influence of stigma extracts of individual varieties of wheat on the germination of chlamydospores of *Ustilago tritici* (Pers.) Jens. Polnohospodarstvo 16:19-23.

Miller, W.B., and C.R. Millikan. 1934a. Investigations on flag smut of wheat caused by *Urocystis tritici* Koern. Part I. J. Dept. Agric., Victoria 32:365-380.

Miller, W.B., and C.R. Millikan. 1934b. Investigations on flag smut of wheat caused by *Urocystic tritici* Koern. Part II. J. Dept. Agric., Victoria 32:418-432.

Millikan, C.R. 1939a. The influence of nutrition on the reaction of wheat to *Urocystis tritici* Koern. Part II. J. Dept. Agric., Victoria 37:349-356.

Millikan, C.R. 1939b. The influence of nutrition on the reaction of wheat to *Urocystis tritici* Koern. Part III. J. Dept. Agric., Victoria 37:587-596.

Millikan, C.R, and H.J. Sims. 1937. The reaction of wheat varieties to flag smut. J. Dept. Agric., Victoria 35:514-520.

Mills, D., and A.C.L. Churchill. 1988. *Tilletia* spp., bunt fungi of the Gramineae. In pages 401-414, G.S. Sidhue, ed., Advances in Plant Pathology. Vol. 6, Academic Press, London.

Minz, G. 1943. Parasitic fungi on imported straw. Hassadeh 24:36. (Hebrew) Rev. Appl. Mycol. 23:336. (Abstr.).

Mirza, M.S., and M.A. Khan. 1983. A new race L-2, of *Tilletia foetida* from Pakistan. Pakistan J.Agric. Res. 4:37-39.

Mishra, R.P., and A.C. Jain. 1968. Evaluation of different techniques of inoculation of loose smut of wheat, *Ustilago nuda* f. *tritici*. J.N.K.V.V. Res. J. 2:17-19.

Mitra, M. 1931. A new bunt of wheat in India. Ann. Appl. Biol. 18:178-179.

Mitra, M. 1935. Stinking smut (bunt) of wheat with a special reference to *Tilletia indica* Mitra. Indian J. Agric. Sci. 5:1-24.

Mitra, M. 1937. Studies on the stinking smut or bunt of wheat in India. Indian J. Agric. Sci. 7:459-478.

Miyake, I. 1912. Studies on Chinese fungi. Bot. Mag. Tokyo, 26:51-66. Quoted by Purdy (1965).

Moore, M.B. 1936. A method for inoculating wheat and barley ears with smuts. Phytopathology 26:397-400.

Moore, K.J., and J. Kuiper. 1974. New treatments for bunt of wheat. Agric. Gaz. N.S.W. 85:16-17.

Moore, M., and D.E. Munnecke. 1954. An air blast method for rapid inoculation of wheat and barley with loose smut. Phytopathology 44:499 (Abstr.). Also Rept. 7th Hard Red Winter Wheat Imp. Conf. Manhattan, Kansas, 19-21 January 1955. U.S.D.A Agronomy Dept. Agric. Exp. Stat., Lincoln, Nebraska.

Mordue, J.E.M., and G.C. Ainsworth. 1984. Ustilaginales of the British Isles. Mycol. Papers 33:381-388.

Mordue, J.E.M., and J.M. Waller. 1981a. *Urocystis agropyri*. CMI descriptions of pathogenic fungi and bacteria, No. 716. CMI, Kew Surrey, England.

Mordue, J.E.M., and J.M. Waller. 1981b. *Urocystis striiformis*. CMI descriptions of pathogenic fungi and bacteria, No. 717. CMI, Kew Surrey, England.

Morwood, R.B. 1930. Flag smut in wheat experiments, 1929. Queensland Agric. J. 33:381-388.

Morwood, R.B. 1931. Flag smut in wheat experiments, 1930. Queensland Agric. J. 35:363-367.

Mozgovoi, A.F., I.Y. Saakyan, and M.V. Novikova. 1987. Gene pool of sources and donors for breeding winter bread wheat for immunity to bunt. Vavilova. 176:11-15.

Mulder, D. 1958. Plant diseases of economic importance in northern region, United Arab Republic. Plant Prot. Bull. 7:1-5.

Mundkur, B.B. 1940. A second contribution towards a knowledge of Indian Ustilaginales. Trans. Brit. Mycol. Soc. 24:312-336.

Mundkur, B.B. 1943a. Karnal bunt, an airborne disease. Curr. Sci. 12:230-231.

Mundkur, B.B. 1943b. Studies in cereal smuts. V. Mode of transmission of the Karnal bunt of wheat. Indian J. Agric. Sci. 13:54-58.

Munjal, R.L. 1975. Status of Karnal bunt (*Neovossia indica*) of wheat in Northern India during 1968-69 and 1969-70. Indian J. Mycol. Plant Path. 5:185-187.

Murray, G.M., and J.F. Brown. 1987. The incidence and relative importance of wheat diseases in Australia. Austral. Plant Path. 16:34-37.

Mwitirwa, K. 1988. Status of wheat production research in Zaire. In pages 173-181, M. van Ginkel and D.G. Tanner, eds. The Fifth Regional Wheat Workshop for Eastern, Central and Southern Africa and the Indian Ocean, Antsirabe, Madagascar, October 5-10, 1987. CIDA and CIMMYT, Mexico.

Nagarajan, S. 1989. Epidemiology and loss of rice, wheat, and pearl millet crops due to diseases in India. In pages 21-30, Proc. 22nd International Symposium on Tropical Agriculture Research, 25-27 August 1988. Kyoto, Japan.

Nagarajan, S. 1991. Epidemiology of Karnal bunt of wheat incited by *Neovossia indica* and an attempt to develop a disease prediction system. Wheat Special Report No. 4. Mexico D.F.: CIMMYT. 69 pp.

Nath, R., A.K. Lambat, P.M. Mukewar, and L. Rani. 1981. Interceptions of pathogenic fungi on imported seed and planting material. Indian Phytopath. 34:282-286.

Nelson, B.D., and R. Durán. 1984. Cytology and morphological development of basidia, dikaryons, and infective structures of *Urocystis agropyri* from wheat. Phytopathology 74:299-304.

Nielsen, J. 1972. Isolation and culture of monokaryotic haplonts of *Ustilago tritici*, observations on their physiology, and the taxonomic relationship between *U. tritici* and *U. nuda*. Can. J. Bot. 50:1775-1781.

Nielsen, J. 1973. Reaction of Triticale and spring rye to loose smut of wheat. Can. J. Plant Sci. 53:749-753.

Nielsen, J. 1977. Inheritance of virulence of loose smut of wheat, *Ustilago tritici*, on the differential cultivars Renfrew, Florence x Aurore, Kota, and Little Club. Can. J. Bot. 55:260-263.

Nielsen, J. 1978. Host range of the smut species *Ustilago nuda* and *Ustilago tritici* in the tribe Triticeae. Can. J. Bot. 56:901-915.

Nielsen, J. 1981. Is there a quick screening method for identifying wheat varieties resistant to loose smut? Cereal Res. Commun. 9:249-252.

Nielsen, J. 1982. Inheritance of virulence of *Ustilago tritici* on the differential cultivars Carma, Red Bobs, and a derivative of the cross Thatcher x Regent. Can. J. Bot. 60:1191-1193.

Nielsen, J. 1983. Spring wheats immune or highly resistant to *Ustilago tritici*. Plant Dis. 67:860-863.

Nielsen, J. 1985. *Ustilago* spp. pathogenic on *Aegilops*. II. *Ustilago tritici*. Can. J. Bot. 63:765-771.

Nielsen, J. 1987a. Races of *Ustilago tritici* and techniques for their study. Can. J. Plant Path. 9:91-105.

Nielsen, J. 1987b. Reaction of *Hordeum* species to the smut fungi *Ustilago nuda* and *U. tritici*. Can. J. Bot. 65:2024-2027.

Nielsen, J., and P.L. Dyck. 1988. Three improved differential hosts to identify races of *Ustilago tritici*. Can. J. Plant Path. 10:327-331.

Nielsen, J., and V. Tikhomirov. 1993. Races of *Ustilago tritici* identified in field collections from eastern Siberia using Canadian and Soviet differentials. Can. J. Plant Path. 15:193-200.

Niemann, E. 1955. Ueber die Wirkung verschiedener Saatgutaufbereitungs-Verfahren auf den Gersten-und Weizenflugbrandbefall (*Ustilago nuda* and *U. tritici*). Z. Pfl.-bau Pfl.-schutz, pp. 1-7.

Niemann, E. 1956. Taxonomy and germination of physiology of the *Tilletia*-species of cereals and wild grasses). Phytopath. Z. 28:113-166.

Niemann, E. 1962. Beurteilung der thermischen und anaeroben Verfahren zur Flugbrandbekaempfung bei Gerste und Weizen. Angew. Botanik 36:1-15.

Niemann, E., Lemma Kifle, and Awgechew Kidane. 1980. Occurrence and importance of bunt (*Tilletia* spp.) on wheat in Ethiopia. Ethiop. J. Agr. Sci. 2:1-11.

Noble, R.J. 1923. Studies on *Urocystis tritici* Koern., the organism causing flag smut of wheat. Phytopathology 13:127-139.

Noble, R.J. 1924. Studies on the parasitism of *Urocystis tritici* Koern., the organism causing flag smut of wheat. J. Agric. Res. 27:451-490.

Noble, R.J. 1934. Notes on the longevity of spores of the fungus *Urocystis tritici* Koern. J. Proc. Roy Soci. N.S.W. 67:403-410.

Oort, A.J.P. 1939. Inoculation experiments with loose smuts of wheat and barley (*Ustilago tritici* and *U. nuda*). Phytopathology 29:717-728.

Oort, A.J.P. 1944. Studies on loose smut. II. Hypersensitivity of wheat to loose smut. Tijdschr. Plantenz. 50:73-106.

Oort, A.J.P. 1947. Specialization of loose smut of wheat—a problem for the breeder. Tijdschr. Plantenz. 53:25-43.

Oort, A.J.P. 1963. A gene-for-gene relationship in the *Triticum-Ustilago* system, and some remarks on host-pathogen combinations in general. Tijdschr. Plantenz. 69:104-109.

Ortiz-Monasterio, I., K.D. Sayre, G. Fuentes-Davila, M. Camacho C., and O. Moreno. 1993. Algunas prácticas agronómicas relacionadas con la incidencia de carbón parcial. En paginas 47-58, Taller sobre las estrategias para el control del carbón parcial del trigo (*Neovossia indica*) en el estado de Sonora. Publicación Especial No. 6, Campo Experimental "Valle del Yaqui"-CIRNO.

Osmanzai, M., and A. Quayoum. 1972. Afghanistan Uniform Wheat Variety Screening Trial for Bunt, 1970-1971. In pages 1-5, Regional Wheat Workshop, Volume 1—Diseases. The Ford Foundation, Beirut, Lebanon, February 14-17, 1972.

Özkan, M. 1971. Investigations on the distribution of *Tilletia controversa* Kühn on wheat and grasses and *Tilletia secalis* (Cda.) Koern. on rye in Turkey. Bitki Koruma Bulteni 11:101-132.

Özkan, M., and E. Damgaci. 1986. Distribution and control methods of dwarf bunt (*Tilletia controversa* Kühn) in Turkey. In pages 150-154, Proc., 6th Congress of the Medit. Phytopath. Union, 1-6 October, 1986. Cairo, Egypt.

Padwick, G.W. 1939. Report of the Imperial Mycologist. Sci. Rep. Agric. Res. Inst., New Delhi, 1937-38, pp. 105-112 (Abstr.).

Pal, B.P., and B.B. Mundkur. 1941. Studies on Indian cereal smuts. III. Varietal resistance of Indian and other wheats to flag smut. Indian J. Agric. Sci. 11:687-694.

Pandey, D.K., and P.L. Gautam. 1988. Morphology of spike in relation to resistance to loose smut (*Ustilago tritici*) in wheat (*Triticum aestivum*). Indian J. Agric. Sci. 58:713-714.

Parii, I.F. 1973. Infection of wheat and barley with loose smut depending on the type of inflorescence. Sel. Semenovodstvo 38:32-33.

Parlak, Y. 1981. Seedborne pathogens on wheat (particularly smuts). EPPO Bull. 11:83-86.

Patterson, F.L., and R.E. Allan. 1981. Soft wheat breeding in the United States. In pages 33-98, W.T. Yamazaki and C.T. Greenwood, eds., Soft Wheat: Production, Breeding, Milling and Uses. American Association of Cereal Chemists, Inc., St. Paul, Minnesota.

Paulus, A., and G.H. Starr. 1951. Control of loose smut with antibiotics. Agron. J. 43:617.

Peterson, F., L. Reitz, V.R. McMinimy, A.L. Aspelin, W.A. Carlson, C.S. Adams, and R. Pagan. 1967. Agricultural Development in Afghanistan with Special Emphasis on Wheat. Report United States Agric. Review Team. Kabul, Afghanistan. 106 pp.

Peterson, G L., M.R. Bonde, W.M. Dowler., and M.H. Royer. 1984. Morphological comparisons of *Tilletia indica* (Mitra) from India and Mexico. Phytopathology 74:757 (Abstr.).

Pichler, F. 1953. Zur Frage der Keimung von Roggen—und Zwergsteinbrandsporen. (Concerning germination of rye—and dwarf bunt spores). Pflanzenschutz Berichte 11:12-17.

Piekenbrock, P. 1927. Untersuchungen ueber das Verhalten des *Ustilago tritici* an Sorten und Kreuzungen. Kuehn Archiv. 15:411-456.

Platz, G.J., and R.G. Rees. 1980. Flag smut resistance in selected wheat cultivars and lines. Queensland J. Agric. Animal Sci. 37:141-143.

Poehlmann, J.M. 1945. A simple method for inoculating barley with loose smut. Phytopathology 35:640-644.

Pollack, F.G. 1945. Fungi found on imported Australian wheat. Plant Dis. Rep. 29:213-214.

Popp, W. 1958. An improved method of detecting loose smut mycelium in whole embryos of wheat and barley. Phytopathology 48:641-643.

Popp, W. 1959. A new approach to the embryo test for predicting loose smut of wheat in adult plants. Phytopathology 49:75-77.

Preuss, C.G.T. 1848. In Sturm J. Deutschlands Flora 6(25-26):1, Table 1 (1848); Linnaea 24:99-153 1851. Quoted by Purdy (1965).

Prévost, I.B. 1807. Memoir on the immediate cause of bunt or smut of wheat, and of several other diseases of plants, and on preventives of bunt. Paris. 80 pp. English translation by G.W. Keitt. 1939. Phytopath. Classics 6:1-95.

Pridham, J.T., and R.E. Dwyer. 1930. Reaction of wheat varieties to flag smut. Agric. Gaz. N.S.W. 41:148-415.

Pridham, J.T., R.E. Dwyer, and R. Hurst. 1929. Control of flag smut by resistant varieties. Agric. Gaz. N.S.W. 40:520-522.

Purdy, L.H. 1957. Toxicity of Hexachlorobenzene to *Urocystis agropyri*. Plant Dis. Rep. 41:588-559.

Purdy, L.H. 1961. Control of common bunt by seed treatment in the Pacific Northwest. U.S.D.A. Agric. Res. Serv., Crops Res. Div. ARS 21:1-8.

Purdy, L.H. 1963. Comparative effectiveness of seed-treatment chemicals against flag smut of wheat. Plant Dis. Rep. 47:793-796.

Purdy, L.H. 1965. Flag smut of wheat. Bot. Rev. 31:565-606.

Purdy, L.H. 1966. Soil moisture and soil temperature, their influence on infection by the wheat flag smut fungus and control of the disease by three seed treatment fungicides. Phytopathology 56:98-101.

Purdy, L.H., and R.E. Allan. 1967. Heritability of flag smut resistance in three wheat crosses. Phytopathology 57:324-325.

Purdy, L.H., and C.S. Holton. 1963. Flag smut of wheat, its distribution and co-existence with stripe rust in the Pacific Northwest. Plant Dis. Rep. 47:516-518.

Purdy, L.H., and E.L. Kendrick. 1963. Influence of environmental factors on the development of wheat bunt in the Pacific Northwest. IV. Effect of soil temperature and moisture on infection by soil spores. Phytopathology 53:416-418.

Purdy, L.H., R.E. Allan, and C.S. Holton. 1964. Reactions of wheats to flag smut in the Pacific northwest. Plant Dis. Rep. 48:895-899.

Purdy, L.H., E.L. Kendrick, J.A. Hoffmann, and C.S. Holton. 1963. Dwarf bunt of wheat. Ann. Rev. Microbiol. 17:199-222.

Putterill, V.A. 1920. Flag smut of wheat. J. Dept. Agric., South Africa 1:252-257.

Quiñones-Leyva, M.A. 1984. Efecto de los fungicidas Bayleton 25 WP, Baycor 300 CC, Baytan 250 EC, y Tilt 20 en aspersiones al follaje para el control del carbón parcial en trigo (*Tilletia indica* Mitra). Reporte Técnico, CIANO-CAEVY, Cd. Obregón, Sonora, México.

Rai, R.C., and A. Singh, A. 1978. A note on the viability of wheat seeds infected with Karnal bunt. Seed Res. 6:188-190.

Rai, R. C., and A. Singh. 1979. Effect of chemical seed treatment on seedborne teliospores of Karnal bunt of wheat. Seed Res. 7:186-189.

Ram, B., and P.C. Arora. 1986. Smuts of wheat in India. In pages 92-123, L.M. Joshi, D.V. Singh, and K.D. Srivastava, eds. Problems and Progress of Wheat Pathology in South Asia. Malhotra Publishing House, New Delhi.

Reed, G.M., and G.H. Dungan. 1920. Flag smut and take-all. University of Illinois Agric. Exp. Stat. Circ. 242. 4 pp.

Rees, R.G., and G.J. Platz. 1973. Grass host for wheat flag smut. Austral. Plant Path. Soc. Newsl. 2:28.

Rewal, H.S., and J.S. Jhooty. 1986a. Physiologic specialization of loose smut of wheat in the Punjab State of India. Plant Dis. 70:228-230.

Rewal, H.S., and J.S. Jhooty. 1986b. Seed and soil inoculation in relation to the incidence of flag smut in wheat. Indian Phytopath. 39:599-601.

Roberson, R.W., and E.S. Luttrell. 1987. Ultrastructure of teliospore ontogeny in *Tilletia indica*. Mycologia 79:753-763.

Rod, J. 1958. A genetic-physiological study of the resistance of wheat to loose smut. I. Artificial inoculations, their technique and reliability. Rostl. vyroba 4:493-508.

Rodenhiser, H.A., and C.A. Holton. 1942. Variability in reaction of wheat differential varieties to physiologic races of *Tilletia levis* and *T. tritici*. Phytopathology 32:158-165.

Rodenhiser, H.A., and C.A. Holton. 1953. Differential survival and natural hybridization in mixed populations of *Tilletia caries* and *T. faetida*. Phytopathology 43:558-560.

Rodenhiser, H.A., and E.C. Stakman. 1925. The control of loose smut of wheat and barley, and barley stripe by Uspulun, Semesan, and Germisan. Phytopathology 15:51 (Abstr.).

Roemer, T., and F. Kamlah. 1932. Gibt es eine selektive Wirkung der Wirtspflanze (Weizen) auf den Pliz (*Ustilago*)? Phytopathol. Z. 5:41-53.

Rolli, K. 1977. Maladies transmises par les semences des cereals. Bull. Protection des Cultures 2:3-10.

Royer, M.H., and J. Rytter. 1985. Artificial inoculation of wheat with *Tilletia indica* from Mexico and India. Plant Dis. 69:317-319.

Royer, M.H., and J. Rytter. 1988. Comparison of host ranges of *Tilletia indica* and *T. barclayana*. Plant Dis. 72:133-136.

Russell, B.W., and D. Mills. 1993. Electrophoretic karyotypes of *Tilletia caries*, *T.controversa*, and their F1 progeny: Further evidence for conspecific status. Molec. Plant-Microbe Interact. 6:66-74.

Ryzhei, I.P. 1960. Breeding for resistance to loose smut of wheat. Agrobiologiya 2:232-236.

Saad, A.T., and F. Nienhaus. 1969. Plant diseases in Lebanon. Z. Pfl. Krankkh. Pfl. Sch. 76:537-551.

Salazar-Huerta, F.J., and J.M. Prescott. 1986. Proyecto de validación de tecnología. Control químico del carbón parcial en campos comerciales de trigo en los Valles del Yaqui y Mayo, Sonora, Ciclo otoño-invierno 1985-86. CIANO-CAEVY, Cd. Obregón, Sonora, México.

Salazar-Huerta, F.J., and J.M. Prescott. 1987. Validación de tecnología. Contro químico del carbón parcial *Neovossia indica* en campos comerciales de trigo en los Valles del Yaqui y Mayo, Sonora. Ciclo otoño-invierno 1986-87. CIANO-CAEVY, Cd. Obregón, Sonora, México.

Salazar-Huerta, F.J., J. Smilanick, J.M. Prescott, F. Zillinsky, and R. Metzger. 1986a. Evaluación de 11 fungicidas aplicados al follaje en tres fechas de siembra para el control de carbón parcial *Neovossia indica* (Mitra) Mundkur en trigo en el Valle del Yaqui, Sonora, Ciclo otoño-invierno 1985-86, CIANO-CAEVY, Cd. Obregón, Sonora, México.

Salazar-Huerta, F.J., J.M. Prescott, A.R. Navarro-Soto, and T. Espinoza-Salazar. 1986b. Tratamiento químico a la semilla de trigo para el control del carbón parcial *Neovossia indica* (Mitra) Mundkur en laboratorio. Valle del Yaqui, Sonora. Ciclo otoño-invierno 1985-86. CAEVY-CIFAP, Cd. Obregon, Sonora, Mexico.

Salazar-Huerta, F.J., K.S. Osada, S.L. Gilchrist, and G. Fuentes-Davila. 1990. Evaluación de la resistencia de seis genotipos de trigo (*Triticum vulgare* L.) al carbón parcial causado por el hongo *Tilletia indica* Mitra en invernadero. Rev. Mexicana Fitopatol. 8:145-152.

SARH. 1987. Cuarentena interior número 16 contra el Carbón Parcial del trigo. Diario Oficial 33-42. Secretaría de Agricultura y Recursos Hidráulicos, Mexico.

Sattar, A., and A. Hafiz. 1952a. Germination, viability of spores of *Urocystis tritici* Koern., the causal fungus of flag smut of wheat. Pakistan J. Sci. Res. 4:12-16.

Sattar, A., and A. Hafiz. 1952b. Some factors influencing the incidence of flag smut of wheat. Pakistan J. Sci. 4:25-30.

Sattar, A., and A. Hafiz. 1952c. Research on plant diseases of the Punjab. Monogr. Pakistan. Assoc. Adv. Sci. 1. 158 pp.

Savile, D.B.O. 1962. *Tilletia controversa*. Mycologia 54:109-110.

Savulescu, T., A. Hulea, and A. Stanescu. 1942. Das Vorkommin und die Verbreitung der in Rumanien den Weizenstinkbrand hervorbringenden *Tilletia*-Arten. Phytopath. Z. 14:148-187.

Schaller, C.W., C.S. Holton, and E.L. Kendrick. 1960. Inheritance of the second factor for resistance to bunt, *Tilletia caries* and *T. foetida*, in the wheat variety Martin. Agron. J. 52:280-282.

Schmidt, J.W., R. Morris, and V.A. Johnson. 1969. Monosomic analysis for bunt resistance in derivatives of Turkey and Oro wheats. Crop Sci. 9:286-288.

Schuhmann, G. 1960. Kunstliche Infektion von Roggensorten und Grasern mit Zwergsteinbrand (Artificial infection of rye varieties and grasses with dwarf bunt). Bayerische Landwirt Jahr. 38:997-999.

Scott, D.B. 1990. Wheat Diseases in South Africa. Tech. Com. No. 220. Grain Crops Research Institute, Bethlehem, South Africa. 62 pp.

Scott, D.B., and J. Le Roux. 1992. Wheat. In pages 457-476, T.N. Trench, D.J. Wilkinson, and S.P. Esterhuysen, eds., South African Plant Disease Handbook. Kendall and Strachen, South Africa.

Sears, E.R., C.W. Schaller, and F.N. Briggs. 1960. Identification of the chromosome carrying the Martin gene for resistance of wheat to bunt. Can. J. Genet. Cytol. 2:262-267.

Sekhon, K.S., A.K. Saxena, S.K. Randhawa, and K.S. Gill. 1980. Effect of Karnal bunt disease on quality characteristics of wheat. Bull. Grain Tech. 18:208-212.

Sekhon, K.S., S.K. Randhawa, A.K. Saxena, and K.S. Gill. 1981. Effect of washing/steeping on the acceptability of Karnal bunt infected wheat for bread, cookie and chapati making. J. Food Sci. Tech. 18:1-2.

Sen, B., and R.L. Munjal. 1964. Nutritional requirements of *Ustilago nuda tritici* Schaff.—The causal agent of loose smut of wheat. Sci. Cult. 31:196.

Shafik, I., Y.H. El-Daoudi, E. Ghobrial, A.A. Bassiouni, S.A. Abu El-Naga, and S. Sherif. 1990. Wheat loose smut survey and chemical control in Egypt. In pages 497-506, Proc. 6th Egypt. Phytopath. Cong., 5-7 March 1990, Cairo, Egypt.

Shah Samin, S.M. 1969. Plant Diseases Found in Afghanistan. Tech. Bull. No. 12. Kabul, University, Kabul, Afghanistan. 112 pp.

Shariff, G., and A. Bamdadian. 1974. Importance and situation of wheat and barley diseases in Iran. In pages 300-305, Proc. Fourth FAO/ Rockefeller Foundation Wheat Seminar, Tehran, Iran, 21 May-2 June 1973. FAO, Rome.

Sharifnabi, B., and G.H.A. Hedjaroude. 1992. Occurrence and geographical distribution of *Tilletia* species attacking winter wheat in west and north-west of Iran. Iran. J. Plant Path. 28:31-33.

Shelton, J.P. 1924. Breeding wheats resistant to flag smut. Agric. Gaz. N.S.W. 35:336-338.

Shen, T.H. 1934. The inheritance of resistance to flag smut in ten wheat crosses. Bulletin Nanking College of Agriculture and Forestry 17. Rev. Appl. Mycol. 11:295 (Abstr.).

Shen, T.H., S.E. Tai, and S.C. Chang. 1938. The transgressive inheritance of reaction to flag smut, earliness of heading, partial sterility, and stiffness of glumes in a varietal cross of wheat. J. Amer. Soc. Agron. 30:68-79.

Sherif, S., E.H. Ghanem, I. Shafik, E.E. Mostafa, and M.M. Abdel-Aleem. 1991. Integrated control of wheat loose smut in Egypt. Assiut J. Agri. Sci. 22:153-163.

Shinohara, M. 1976. Behaviour of *Ustilago nuda* (Jens.) Rostr. and *U. tritici* (Pers.) Rostr. in their host tissues. Rev. Plant Prot. Res. 9:124-142.

Simmonds, P.M. 1946. Detection of the loose smut fungi in embryos of barley and wheat. Sci. Agric. 26:51-58.

Sims, H.J., C.G. Webb, G. Blackburn. 1943. Mallee wheat experiments. J. Dep. Agric. Victoria 41:169-172.

Singh, A., and R. Prasad. 1978. Date of sowing and meteorological factors in relation to occurrence of Karnal bunt of wheat in U.P. Tarai. Indian J. Mycol. Plant Path. 8:2 (Abstr.).

Singh, A., and R. Prasad. 1980. Control of Karnal bunt of wheat by a spray of fungicides. Indian J. Mycol. Plant Path. 10:1 (Abstr.).

Singh, A., A.N. Tewari, and R.C. Rai. 1985a. Control of Karnal bunt of wheat by a spray of fungicides. Indian Phytopath. 38:104:108.

Singh, A., K.P. Singh, and A.N. Tewari. 1988. Salient findings of Karnal bunt research at Pantnagar. Department of Plant Pathology, G.B. Pant University of Agriculture and Technology, Pantnagar, India.

Singh, B., and S.C. Mathur. 1953. Bunts of wheat. Agric. Animal Husb. in India 3:10-12.

Singh, D.V. 1980. A note on the effect of Karnal bunt infection on the vigour of wheat seed. Seed Res. 8:81-82.

Singh, D.V. 1986. Bunts of wheat in India. In pages 124-161, L.M. Joshi, D.V. Singh, and K.D. Srivastava, eds. Problems and Progress of Wheat Pathology in South Asia. Malhotra Publishing House, New Delhi, India.

Singh, D.V., and K.D. Srivastava. 1990. Investigation on Karnal bunt (*Neovossia indica*) of wheat. Annual Research Progress Report, Division of Mycology and Plant Pathology, Indian Agricultural Research Institute, New Delhi, India.

Singh, D.V., K.D. Srivastava, and L.M. Joshi. 1979. Prevent losses from Karnal bunt. Indian Farming, February 1979, pp. 7-8.

Singh, D.V., L.M. Joshi, and K.D. Srivastava. 1983. Karnal bunt—A new threat of wheat in India. In pages 121-135, H.K. Sakesna Festschrift, A. Hassain, Kishan Singh, B.P. Singh, and V.P. Agnihotri, eds., Recent Advances in Plant Pathology. Print House (India), Luknow.

Singh, D.V., K.D. Srivastava, L.M. Joshi, and B.R. Verma. 1985b. Evaluation of some fungicides for the control of Karnal bunt of wheat. Indian Phytopath. 38:571-573.

Singh, D.V., L.M. Joshi, and K.D. Srivastava. 1986. Varietal susceptibility and spread of Karnal Bunt of wheat in India. Rachis 5:40-42.

Singh, D.V., R. Agarwal, J.K. Shrestha, B.R. Thapa, and H.J. Dubin. 1989. First report of *Tilletia indica* on wheat in Nepal. Plant Dis. 73:273.

Singh, R.A., and A. Krishna. 1982. Susceptible stage for inoculation and effect of Karnal bunt on viability of wheat seed. Indian Phytopath. 35:54-56.

Singh, S.L., and P.P. Singh. 1985. Effect of some fungicide applications against Karnal bunt (*Neovossia indica*, Mitra) Mundkur of wheat. Indian Phytopath. 38:593 (Abstr.).

Singh, S.R., and V.L. Chopra. 1986. Inheritance of resistance to bunt, *Tilletia foetida*, in wheat. Genet. Agr. 40:369-374.

Singh, S.R., and V.L. Chopra. 1987. Identification and characterization of gene(s) for resistance to bunt in Kirac 66 wheat. Proc. Indian Natn. Sci. Acad. B53 No. 2 pp. 149-151.

Singovets, M.E. 1974. Inheritance of resistance to bunt in wheat Agropyron hybrids. Genetika (USSR) 10:25-31.

Sitton, J.W., R.F. Line, J.T. Waldher, and B.J. Goates. 1993. Difenoconazole seed treatment for control of dwarf bunt of winter wheat. Plant Dis. 77:1148-1151.

Skvortzov, S. 1937. A simple method for detecting hyphae of loose smut in wheat grains. Zashch. Rast. (Leningrad) 15:90-91.

Slinkard, A.E., and F.C. Elliott. 1954. The effect of bunt incidence on the yield of wheat in eastern Washington. Agron. J. 46:439-441.

Smeltzer, D.G. 1952. Inheritance of resistance to race T-1 of *Tilletia caries* in Minturke and Cooperatorka wheats. Agron. J. 44:529-533.

Smilanick, J.L., and J.M. Prescott. 1986. Effect of soil fumigation with methyl bromide, metham, and formaldehyde on germination of teliospores of *Tilletia indica*. Phytopathology 76:1060.

Smilanick, J.L., M. Dupler, J.A. Hoffmann, and K. Wiese. 1985a. Evaluation of seed treatments to prevent germination of *Tilletia indica* teliospores. Phytopathology 75:1331 (Abstr.).

Smilanick, J.L., J.A. Hoffmann, and M.H. Royer. 1985b. Effect of temperature, pH, light, and desiccation on teliospore germination of *Tilletia indica*. Phytopathology 75:1428-1431.

Smilanick, J.L., M. Dupler, B.J. Goates, J.A. Hoffmann, D. Clark, and D. Dobson. 1986. Germination of teliospores of Karnal, dwarf, and common bunt fungi after ingestion by animals. Plant Dis. 70:242-244.

Smilanick, J.L., J.A. Hoffmann, N.L. Cashion, and J.M. Prescott. 1987. Evaluation of seed and foliar fungicides for control of Karnal bunt of wheat. Plant Dis. 71:94-96.

Smith, W.K. 1932. The effect of different temperatures on the reaction of Hope wheat to stinking smut. Phytopathology 22:615-627.

Smith, W.K. 1933. Inheritance of reaction of wheat to physiologic forms of *Tilletia levis* and *T. tritici*. J. Agric. Res. 47:89-105.

Sood, A.K. 1988. Note on status of loose smut on wheat in Kullu Valley of Himachal Pradesh. Wheat, Barley and Triticale Abstr. 1992. 009-05542.

Stakman, E.C. 1913. Spore germination of cereal smuts. Minnesota Agric. Exp. Sta. Tech. Bull. 133. 52 pp.

Stakman, E.C. 1922. Diseases of cereal and forage crops in the United States in 1921. Plant Dis. Bull. Suppl. 21:139-254.

Stakman, E.C., and J.G. Harrar. 1957. Principles of Plant Pathology. The Ronald Press Co. New York. 581 pp.

Standley, P.C. 1916. Fungi of New Mexico. Mycologia 8:142-177.

Stanescu, N. 1961. Physiologic changes caused by *Ustilago tritici* (Pers.) Jensen in the wheat plant. Stud. Cerc. Biol., Ser. Biol. Veget. 13:129-140.

Stanford, E.H. 1941. A new factor for resistance to bunt, *Tilletia tritici*, linked with the Martin and Turkey factors. J. Amer. Soc. Agron. 33:559-568

Stockwell, V.O., and E.J. Trione. 1986. Distinguishing teliospores of *Tilletia controversa* from those of *T. caries* by fluorescence microscopy. Plant Dis. 70:924-926.

Stringfield, G.H. 1929. Inoculating wheat with loose smut. J. Amer. Soc. Agron. 21:937-938.

Swinburne, T.R. 1963. Infection of wheat by *Tilletia caries*, the causal organism of bunt. Trans. Br. Mycol. Soc. 46:145-156.

Swingle, W.T. 1892. Treatment of smuts of oats and wheat. USDA Farmers Bull. No. 5.

Sydow, H.P., and E.J. Butler. 1906. Fungi *Indiae orientalis*. Ann. Mycol. 4:427. Quoted by Purdy (1965).

Szemble, S.J. 1934. Threat to wheat in Transcaucasia. Crop Protection, Moscow 8:22-23 (in Russian, translated title). Rev. Applied Mycol. 23 (1935) (Abstr.).

Szepessy, I. 1969. Experiments with high-frequency seed-dressing apparatus. Acta Phytopath. Acad. Sci. Hungary 4:139-151.

Tahir, M., and P.M. Hayes. 1988. A synthesis of winter cereal research and production status in high-elevation areas of West Asia and North Africa. In pages 228-232, Srivastava et al., eds., Winter Cereals and Food Legumes in Mountainous Areas. ICARDA, Aleppo, Syria.

Tamimi, K.M. 1986. Species composition of wheat bunt fungi from different locations in Iraq. In pages 1525-1530, Proc. Fourth Sci. Conf. Agric. Res., Vol. 1, Part 3. October 23-28, 1986. Sci. Res. Council, Baghdad, Iraq.

Tapke, V.F. 1926. Single-bath hot-water and steam treatments of seed wheat for the control of loose smut. USDA Bull. No. 1383.

Tapke, V.F.. 1929. Influence of varietal resistance, sap acidity, and certain environmental factors on the occurrence of loose smut in wheat. J. Agric. Res. 39:313-339.

Tapke, V.F.. 1931. Influence of humidity on floral infection of wheat and barley by loose smut. J. Agric. Res. 43: 503-516.

Tavcar, A. 1934. Lodicules et culture de froment resistant au charbon. 16. Cong. Intern. Agric., Budapest, 1934. Min. Agri. Sect. 4, Theme 2; pp. 816-824.

Taylor, D.K. 1958. The nature and relationship of bunt resistance in a hard red winter wheat selection and certain tester varieties. Diss. Abstr. 18:19.

Taylor, J.W. 1927. Effect of continuous selection of small and large wheat seed on yield, bushel weight, varietal purity and loose smut infection. J. Amer. Soc. Agron. 20:856-867.

Tiemann, A. 1925. Untersuchungen ueber die Empfaenglichkeit des Sommerweizens fuer *Ustilago tritici* und den Einfluss der aeusseren Bedingungen dieser Krankheit. Kuehn Archiv 9:405-467.

Tikhomirov, V.T. 1983. Genetics of resistance of wheat to loose smut. I. Analysis of the wheat/loose smut interaction on the basis of Flor's gene-for-gene hypothesis. Genetika 19:295-303.

Tillet, M. 1755. Dissertation on the cause of the corruption and smutting of the kernels of wheat in the head, and on the means of preventing these untoward circumstances. Bordeaux. 150 pp. English translation by H.B. Humphrey. 1937. Phytopath. Classics 5:1-191.

Tingey, D.C., and B. Tolman. 1934. Inheritance of resistance to loose smut in certain wheat crosses. J. Agric. Res. 48:631-655.

Tisdale, W.H., G.H. Duncan, and C.E. Leighty. 1923. Flag smut of wheat, with special reference to varietal resistance. University of Illinois Agric. Exp. Stat. Bull. 242. 538 pp.

Tisdale, W.H., C.E. Leighty, and B. Koehler. 1927. Further studies on flag smut of wheat. U.S.D.A. Circular 424. 12 pp.

Todorova, V. 1958. When did *T. brevifaciens* first occur in Bulgaria? Bull. Inst. Bot. Sofia. 6:351-361.

Tragner-Born, J., and H. Kaspers. 1981. Control of dwarf bunt on winter wheat with Sibutol. Pflanz Nachr. 34:1-7.

Trail, F., and D. Mills. 1990. Growth of haploid *Tilletia* strains in planta and genetic analysis of a cross of *Tilletia caries* x *T. controversa*. Phytopathology 80:367-370.

Trione, E.J. 1964. Isolation and *in vitro* culture of the wheat bunt fungi *Tilletia caries* and *T. controversa*. Phytopathology 54:592-96.

Trione, E.J. 1974. Free amino-acid changes during germination of teliospores of *Tilletia caries*. Proc. Am. Phytopath. Soc. 1:109 (Abstr.).

Trione, E.J. 1977. Endogenous germination inhibitors in teliospores of the wheat bunt fungi. Phytopathology 67:1245-1249.

Trione, E.J. 1982. Dwarf bunt of wheat and its importance in international wheat trade. Plant Dis. 66:1083-1088.

Trione, E.J., and L.A. Sayavedra-Soto. 1988. Wheat development enhanced by hormone syndrome. Bot. Gaz. 149:317-324.

Trione, E.J., W.M. Hess, and V.O. Stockwell. 1989. Growth and sporulation of the dikaryons of the dwarf bunt fungus in wheat plants and in culture. Can. J. Bot. 67:1671-1680.

Tulasne, L.R., and C. Tulasne. 1847. Mémoire sur les Ustilaginées comparées aux Urédinées. Ann. Sci. Nat. Bot., Ser. 3, 7:12-127.

Tulasne, L.R., and C. Tulasne. 1854. Second Mémoire sur les Uredinées et les Ustilaginees. Ann. Sci. Bot., Ser. 4, 2:77-96.

Tyler, L.J. 1965. Failure of loose smut to build up in winter wheats exposed to abundant inoculum naturally disseminated. Plant Dis. Rep. 49:239-241.

Tyler, L.J., and N.F. Jensen. 1953. Protective cover—a factor influencing the development of dwarf bunt in winter wheat. Plant Dis. Rep. 37:465-466.

Tyler, L.J., and N.F. Jensen. 1958. Some factors that influence development of dwarf bunt in winter wheat. Phytopathology 48:565-571.

Tyner, L.E. 1953. The control of loose smut of barley and wheat by Spergon and by soaking in water at room temperature. Phytopathology 43:313-316.

Valenzuela-Rodriguez, S. 1985. Evaluacion de 4 fungicidas para el control del carbon karnal del trigo. Ciclo otono-invierno 1984-85. CIANO-CAEVY, Cd. Obregon, Sonora, Mexico.

Valenzuela-Rodriguez, S., and A.R. Navarro-Soto. 1985. Evaluacion de la dosis optima de 6 fungicidas en tratamiento a la semilla para el control en laboratorio del carbon karnal del trigo. Ciclo otono-invierno 1984-85. CIANO-CAEVY, Cd. Obregon, Sonora, Mexico.

Vanderwalle. 1953. Essai de conservation des spores de charbon nu du froment et de l'orge (*Ustilago nuda* et *U. nuda tritici*). Parasitica 9:139-144.

Vánky, K. 1985. Carpathian Ustilaginales. Symbolae Botanicae Upsalienses 24(2). Tegelbruksvägen, Gagnef, Sweden. 309 pp.

Vánky, K. 1994. European smut fungi. Gustav Fischer Verlag, New York, 570 pp.

Varenitsa, E.T., L. Yu Saakyan, A.F. Mozgovoi, G.V. Kochetygov, and S.M. Gradskov. 1987. Using derivatives of the variety Zarya as donors of resistance to bunt. Lenina 4:3-5.

Verma, H.S., A. Singh, and V.K. Agarwal. 1984. Application of seedling crown test for screening of systemic fungicides against loose smut of wheat. Seed Res. 12:56-60.

Verrell, A.G., and R.F. Komoll. 1991. Winter Cereal Manag. Guide. N.S.W. Agric. Fish.. 84 pp.

Verwoerd, L. 1929. The biology, parasitism and control of *Urocystis tritici*, Koern., the causal organism of flag smut in wheat (*Triticum* spp.) and recording the occurrence of *Urocystis occulta* (Wallr.) Rab., in South Africa as the cause of "Stem Smut" in rye. Union South Africa Sci. Bull. 76. 59 pp.

von Schmeling, B., and M. Kulka. 1966. Systemic fungicidal activity of 1,4-oxathiin derivatives. Science 152:659-660.

Wagner, F. 1948. On the occurrence of dwarf bunt in Bavaria. Pflanzenschutz 1:1-2.

Wagner, F. 1949. Observations on dwarf bunt. Nachricht. Biolo. Zentral. Braun. 1:39-40.

Wagner, F. 1950. Occurrence, spore germination, and infection of dwarf bunt on wheat. Z. Pflan. Pflanzen. 1:1-13.

Wainwright, A., and D.B. Morris. 1989. Evaluation of triadimenol seed treatments against soilborne bunt of wheat. Ann. Appl. Biol. 114 (Suppl.) 68-69.

Waller, J.M., and J. Bridge. 1978. Plant diseases and nematodes in Sultanate of Oman. PANS 24:313-326.

Waller, J.M., and J.E.M. Mordue. 1983. *Tilletia indica*.. Descriptions of pathogenic fungi and bacteria. No. 748. Commonwealth Mycological Institute Agricultural Bureaux. Surrey, England.

Warham, E.J. 1986. Karnal bunt disease of wheat: a literature review. Tropical Pest Manag. 32:229-242.

Warham, E.J. 1988. Teliospore germination patterns in *Tilletia indica*. Trans. Brit. Mycol. Soc. 90:318-321.

Warham, E.J. 1990. A comparison of inoculation techniques for assessment of germplasm susceptibility to Karnal bunt (*Tilletia indica*) disease of wheat. Ann. Appl. Biol. 116:43-60.

Warham, E.J., and D. Flores. 1988. Farmer surveys on the relation of agronomic practices to Karnal bunt disease of wheat in the Yaqui Valley, Mexico. Tropical Pest Manag. 34:373-381.

Warham, E.J., and J.M. Prescott. 1989. Effectiveness of chemical seed treatments in controlling Karnal bunt disease of wheat. Plant Dis. 73:585-588.

Warham, E.J., A. Mujeeb-Kazi, and V. Rosas. 1986. Karnal Bunt (*Tilletia indica*) resistance screening of *Aegilops* species and their practical utilization for *Triticum aestivum* improvement. Can. J. Plant Path. 8:65-70.

Warmbrunn, K. 1952. Research on dwarf bunt. Phytopath. Z. 19:441-482.

Watson, I.A. 1958. The present status of breeding disease resistant wheats in Australia. Agric. Gaz. N. S. W. 69:630-660.

Waud, J.L., Metzger, R.J. 1970. Inheritance of a new factor (*Bt-8*) for resistance to common bunt in wheat. Crop Sci. 10:703-704.

Weber, G.A. 1930. The plant quarantine and control administration. Its history, activities and organization. Service Monograph, U.S. Government No. 59. Quoted by Purdy (1965).

Weber, G., and K. Schauz. 1985. Characterization of protein patterns in *Tilletia controversa* and *T. caries* with gel electrophoretic methods. Z. Pflan. Pflanzen. 92:600-605.

Weibel, D.E. 1958. Studies on the control of loose smut of wheat. Plant Dis. Rep. 42:737-743.

Weltzien, H. 1963. Plant diseases in the Middle East. Observation in the Republic of Lebanon, 1962. Phytopath. Medit. 2:260-264.

Wiese, M. 1987. Compendium of Wheat Diseases. APS Press, St. Paul, MN. USA. 112 pp.

Williams, E., Jr. 1988. Fungicide seed treatment effectiveness for *Tilletia tritici* control. Phytopathology 78:1525 (Abstr.).

Williams, E., Jr. 1990. Evaluation of fungicide seed treatments for control of seedborne and soilborne common bunt, 1989. Fungic. Nematic. Tests 45:255.

Williams, E., Jr. 1991. Evaluation of fungicide seed treatments for control of seedborne and soilborne common bunt, 1990. Fungic. Nematic. Tests 46:291.

Williams, E., Jr., and F.J. Gough. 1984. Bunt of wheat *Triticum aestivum* caused by *Tilletia caries* widespread in southwestern Oklahoma USA in 1983. Plant Dis. 68:537.

Williams, P.C. 1983. Incidence of stinking smut (*Tilletia* spp.) on commercial wheat samples in northern Syria. Rachis 2:21.

Woestmann, E. 1942. Der fluoreszenzoptische Nachweis von *Ustilago tritici* im Korn. Kuehn-Archiv. 56:247-253.

Wolff, R. 1873. Beitrag zur Kenntnis der Ustilagineen. Bot. Zeit. 31:657-661, 673-677, 689-94. Quoted by Purdy (1965).

Woodward, R.W., and C.S. Holton. 1949. Inheritance of reaction to dwarf bunt, race T-16, and a hybrid in winter wheat crosses. Agron. J. 41:253-254.

Woolman, H.M., and H.B. Humphrey. 1924. Summary of literature on bunt, or stinking smut, of wheat. U.S.D.A. Bull. No. 1210. pp. 1-44.

Wrigley C.W., and A. Rathjen. 1981. Wheat breeding in Australia. In pages 96-135, D.J. Carr, ed., Plants and Man in Australia. Academic Press, Sydney. 313 pp.

Wu, Y. 1949. Temperature and cultural studies on *Urocystis tritici* Koern. Can. J. Res. C27:66-72.

Yablokova, V.A. 1939. Response of the mycelium of *Ustilago tritici* in wheat grain to ultra-violet rays as dependent upon its condition. Comp. Rend. Acad. Sci. USSR, N.S. 23:392-394.

Yamaleev, A.M., V.I. Krivchenko, and I.P. Gavrilyuk. 1975. Differentiation of races of *Ustilago tritici* by proteins in the spores. Selskokh. Biol. 10: 370-374.

Yarham, D. 1993. Soilborne spores as a source of inoculum for wheat bunt. Plant Pathol. 42:654-656.

Yasu, M., and M. Yoshino. 1963. Studies on the ecology of the flag smut of wheat and its control. Saitama Agric. Exp. Stat. Japan Res. Bull. 20, 34 pp. English summary as quoted by Purdy (1965).

Yirgou, D. 1967. Plant Diseases of Economic Importance in Ethiopia. Expt. Stat. Bull. 50. Haile Selassie I University, College of Agriculture, Debre Zeit, Ethiopia. 30 pp.

Young, P.A. 1935. A new variety of *Tilletia tritici* in Montana. Phytopathology 25:40 (Abstr.).

Yu, T.F., and H.K. Chen. 1931. A Chinese wheat resistant to flag smut. Phytopathology 21:1202-1203.

Yu, T.F., H.K. Chen, and L. Hwang. 1933. Varietal resistance and susceptibility to flag smut (*Urocystis tritici* Koern.). Nanking Jour. 3:217-234. Rev. Appl. Mycol. 13:19 (1934). (Abstr.).

Yu, T.F., H.K. Chen, and L. Hwang. 1934. Varietal resistance and susceptibility of wheats to flag smut (*Urocystis tritici* Koern.) II. Agr. Sinica 1:79-81. Rev. Appl. Mycol. 13:752 (1934). (Abstr.).

Yu, T.F., L. Hwang, and C.T. Tsiang. 1936. Varietal resistance and susceptiblity of wheats to flag smut (*Urocystis tritici* Koern.). III. Physiologic specialization in *Urocystis tritici* Koern. Bull. Chinese Bot. Soc. 2:111-113. Rev. Appl. Mycol. 16:305 (1937). (Abstr.).

Yu, T.F., H.R. Wang, and C.F. Fang. 1945. Varietal resistance and susceptibility of wheat to flag smut (*Urocystis tritici* Koern.). IV. Further studies on physiologic specialization in *Urocystis tritici* Koern. Phytopathology 35:332-338.

Yüksel, H., A. Güncan, and M.T. Döken. 1980. The distribution and damage of bunts (*Tilletia* spp.) and wheat gall nematode [*Anguina tritici* (Steinbuch) Chitwood] on wheat in the eastern part of Anatolia. J. Turk. Phytopath. 9:77-88.

Zad, J. 1972. Wheat covered smut in Iran. Proceed. In pages 36, III Congress Medit. Phytopath. Union. Oeiras, Portugal 22-23 October 1972.

Zadoks, J.C., and R.D. Schein. 1979. Epidemiology and Plant Disease Management. Oxford University Press, New York. 427 pp.

Zadoks, J.C., T.T. Chang, and C.F. Konzak. 1974. A decimal code for the growth stages of cereals. Weed Res. 14:415-421.

Zalesskij, V. 1935. Anaerobic method for the control of loose smut of wheat. Zashch. Rast. (Leningrad) 1:135-138.

Zemanek, J., and P. Bartos. 1964. Anaerobic treatment of wheat against loose smut (*Ustilago tritici* (Pers.) Jens.). Rostl.Vyroba 37:371-382.

Zhang, Z., L. Lange, and S.B. Mathur. 1984. Teliospore survival and plant quarantine significance of *Tilletia indica* (causal agent of Karnal bunt) particularly in relation to China. Europ. Plant Prot. Bull. 14:119-128.

Zhivotokov, L.A., S.V. Biryukov, and A.Y. Stepanenko 1989. Wheat. Urozhai, Kiev, Ukraine. 320 pp. (in Russian).

Zillinsky, F.J. 1983. Common Diseases of Small Grain Cereals: a Guide to Identification. CIMMYT, Mexico. 141 pp.

Zogg, H. 1959. Studies on the survival of spores of dwarf bunt in soil (*Tilletia controversa* Kühn). Phytopath. Z. 35:1-22.

Zohary, D., and M. Hopf. 1988. Domestication of Plants in the Old World. Clarendon Press, Oxford University Press, New York. 249 pp.

Zscheile, F.P. 1955. Some physiological aspects of bunt resistance in wheat. Plant Phys. 30:432-437.

Zscheile, F.P. 1966. Photoperiod-temperature-light relationships in development of wheat and bunt. Phytopath. Z. 57:329-374.

Zundel, G.L. 1933. The Ustilaginales of Pennsylvania. Proc. Pennsylvania Acad. Sci. 7:1-8.

Additional Reading

Ainsworth, G.C. 1971. Ainsworth and Bisby's Dictionary of the Fungi. 6th edition. Kew. Commonwealth Mycological Institute.

Holiday, P. 1992. A Dictionary of Plant Pathology. Cambridge University Press. Cambridge.

Johnston, A., and C. Booth, eds. 1983. Plant Pathologist's Pocketbook. Commonwealth Mycological Institute. The Cambrian News Ltd., Aberystwyth, Wales.

Wiese, M.V. 1977. Compendium of Wheat Diseases. The American Phytopathological Society. St. Paul, MN 55108.

Glossary

Aggressiveness—Ability to maintain self in nature; disease-causing capacity in nature; a component (with virulence) in parasitic fitness; a concept similar to parasitic fitness. See Shaner et al. (1992), Annual Review Phytopathology 30:47-66 for further discussion of this concept.

Allantoid—Slightly curved spores, sausage-like in form.

Anastomosis—Fusion of hyphae and exchange of their contents.

Anthesis—Flowering, evident in cereals and grasses by extrusion of the anthers.

Apical meristem—Cells at the tip of a shoot that function in cell division and differentiation.

Apiculus—A short projection on the end of a spore by which it was fixed to the sterigma.

Appressorium—A swelling on a germ tube or hypha for attachment in early stages of host penetration.

Areolate—To divide into small areas by margins that differ in color, texture, or structure.

Avirulent—Inability of a pathogen to overcome host resistance.

Axenic culture—A growing system for one microorganism free of others.

Basidiospore—A propagative cell produced on a basidium after meiosis. It contains one or more haploid nuclei.

Bipolar—A form of heterothallism involving only one pair of alleles at a single locus that controls compatibility .

Biotype—A subdivision of physiologic race the individuals of which have similar genetic makeup; of bacteria, a subdivision of species distinguished by some physiological characteristic.

Bunt—A smut fungus that sporulates only in the ovaries. Also, the disease caused by these fungi.

Bunt ball—Sorus of smut fungi that replaces the kernel.

Caryopsis—A seed of a grass or cereal.

Centipoise—A standard of viscosity equal to 0.01 poise, the c.g.s. unit of viscosity. Water at 20ºC has a viscosity of 1.002 centipoise or 0.002 poise.

Coleoptile—A protective sheath surrounding the shoot in cereals and grasses.

Common bunt—The smut disease caused by *Tilletia tritici* or *T. laevis* that infects seedlings.

Control of disease—To check or reduce the presence of or the effects of plant disease.

Cotyledon—The first seedling leaf or leaves.

Cultivar—A cultivated variety as opposed to a botanical (taxonomic) variety.

Differential host—A line or cultivar that is susceptible to some pathogen isolates, but resistant to other isolates.

Differential set—A group of genotypes used for distinguishing physiologic races that usually include a universal susceptible and a universal resistant genotype among the genotypes that differ in reactions to the pathogen collections.

Dikaryon—A fungal cell containing two genetically distinct and sexually compatible haploid nuclei.

Dikaryophase—One of three phases in the life cycle characterized by cells with two haploid nuclei of opposite mating type.

Diploid—A nucleus carrying two sets of chromosomes.

Diplophase—One of three phases in the life cycle characterized by diploid nuclei.

Disease—An abnormal physiological reaction of a plant to a pathogen, often accompanied by symptoms and altered morphology and/or anatomy.

Disease cycle—The chain of events involved in disease development, including the stages of pathogen development and effects of the disease on the host.

Disomic—Having two sets of homologous chromosomes or genes.

Dissemination—Release and spread of fungus spores from the site of production.

Dominant—An allele that masks the phenotypic effect of another allele of the same gene. The phenotypic effect is the same in the heterozygous as in the homozygous condition.

Dormant—Resting due to reduced physiological activity.

Dwarf bunt—A smut disease caused by *Tilletia controversa* that infects seedlings and severely stunts tillers.

Endosperm—The tissue surrounding the embryo in the seed that provides nutrition to the embryo.

Environment—The physical and biological surroundings of a species or group of species.

Exospore—The outer covering of a spore.

Filiforme—Thread-like structure.

Flag leaf—The uppermost leaf on the stem.

Flag smut—A smut disease that affects primarily leaves and culms, as in *Urocystis agropyri*.

Floret—The flower in the spikelet of a cereal plant.

Formae speciales—A subdivision of a pathogen species based primarily on the host genera that are attacked.

Fungicide—A chemical that is toxic to fungi.

Fungistatic—A chemical that inhibits the growth of fungi without killing.

Fungus—An undifferentiated plant lacking chlorophyll and conductive tissues.

Funiculus—The slender stalk of an ovule or seed.

Fusiforme—Spindle-like in shape.

Gene—A hereditary unit on a chromosome which determines or conditions one or more characteristics.

Gene-for-gene relationship—The specific interaction of a host and a pathogen that is conditioned by complimentary genes of host and pathogen.

Germicide—A chemical that kills micro-organisms, especially pathogens.

Germination—The initiation of growth from a resting propagule.

Glume—A bract at the base of a spikelet of a cereal or grass.

Haploid—Carrying a single set of chromosomes.

Haplophase—One of three phases in the life cycle characterized by nuclei that are haploid.

Haplont—An organism having haploid somatic nuclei.

H-body—A structure formed by the fusion of sporidia.

Heterobasidiomycete—Fungi that have basidia of different form and that are usually divided by septa.

Heterothallic—Two different genotypes or mating types are required for sexual reproduction.

Homothallic—A haploid nucleus can give rise to a mycelium that is capable of sexual reproduction.

Host range—The genera and species that are susceptible to a pathogen; resistant or immune genera or species are not usually included among hosts.

Hypersensitive reaction—Host response to infection characterized by rapid death of cells and tissues that prevents further growth of the pathogen.

Hypocotyl—The part of the stem below the cotyledons in the embryo.

Immune—Free from infection by a pathogen.

Incompatible reaction—Host/pathogen response to infection that results in immune or resistant reactions.

Infect—To establish a pathogenic relationship with a host plant.

Inoculate—To introduce pathogen propagules onto or into a host or culture for the purpose of producing infection or for growing the pathogen in culture.

Inoculum—Propagules of microorganisms used for inoculation.

Isolate—A spore or culture of a microorganism, and derived cultures, that are separated from others.

Karnal bunt—A bunt disease that infects immature seeds, caused by *Tilletia indica;* named for the town of Karnal, India.

Karyogamy—The union and interchange of nuclei after cytoplasmic fusion, the fusion of two compatible haploid nuclei into one diploid nucleus.

Latent period—The time between inoculation and appearance of symptoms and/or spore production.

Lemma—The lower enclosing bract of the flower in cereals and grasses.

Linkage—The association of genes located on the same chromosome.

Lodicules—Two small scale-like structures below the ovary in cereals and grasses that swell up at flowering and open the bracts.

Loose smut—A smut disease caused by *Ustilago tritici* that infects ovaries only.

Mating type—A subdivision of a microorganism on the basis of mating behavior.

Matrix potential—The absorption and capillary forces that bind water to plant and soil constituents.

Meiosis—The reduction division of chromosomes in a diploid nucleus that results in the formation of four haploid gametes.

Micron (μm)—A unit of length equal to 0.001 mm.

Mitosis—The division of a nucleus in which chromosomes are duplicated, giving rise to two daughter nuclei with the original chromosome complement.

Monogenic—Having one type of gene.

Monokaryon—An individual having one nucleus per cell.

Monokaryotic—Mycelia consisting of cells with single nuclei.

Monosomic—A plant with one missing chromosome.

Morphological resistance—Prevention of plant disease by means of structural and anatomical features of plants.

Mycelium—A mass of hyphae or the thallus of a fungus.

***n* —**A single set of chromosomes per nucleus; 2*n*—A double set of chromosomes per nucleus; (*n* + *n*)—Two haploid nuclei of opposite mating type within a cell.

Nucellus—The central portion of an ovule containing the embryo sac.

Ovary—The female reproductive structure that contains the egg.

Palea—The upper enclosing bract of the flower in cereals and grasses.

Partial resistance—Resistance expressed by reduced rate of infection, though symptom expression and macroscopic development of the pathogen are similar to those on a susceptible genotype; in smuts, infection occurs followed by some sporulation.

Pathogen—An organism capable of causing disease.

Pathogenicity—Able to cause disease.

Pathotype—A subdivision of a species with characteristic pathogenicity, especially host range, synonymous with *formae speciales.*

Pericarp—The wall of a ripened ovary.

Physiological resistance—Prevention or reduction of plant disease development or severity due to physiological responses of the plant to the pathogen.

Physiologic race—A population of individuals that are alike in morphology, but differ in their virulences on a set of differential cultivars or that differ in other cultural or physiological characteristics.

Physiologic specialization—The process whereby isolates or races of microorganisms become differentiated in function, as in pathogenicity or ability to grow on specific media.

Plasmogamy—The process of anastomosis of two cells with intermingling of their protoplasts and bringing haploid nuclei of opposite mating type into one cell.

Plumular bud—The apical meristem.

Primary homothallism—The nuclei are of the same genotype and sexual reproduction occurs without the intervention of another spore or thallas.

Promycelium—The germ tube (basidium) of a ustilispore (teliospore) of a smut fungus.

Rachis—The stem of the spike in cereals and grasses.

Recessive—An allele that does not produce a phenotypic effect when heterozygous with the dominate allele, masking the effect of an allele by another allele of the same gene.

Resistance—The genetic characteristic of a plant that prevents or reduces pathogen or disease development toward disease.

Reticulation—The net-like appearance of the teliospore wall of smut fungi like *Tilletia tritici.*

Rogue—To remove unwanted plants from a plot or field.

Scutellum—A flat portion of the embryo of a cereal or grass, probably the cotyledon pressed against the endosperm.

Secondary homothallism—Inclusion of nuclei of compatible mating types within a single spore and sexual reproduction occurs in cultures derived from the single spore.

Septum—Cross wall of a hypha or other fungal structure.

Set of testers—Same as differential set.

Smut—A disease characterized by masses of dark spores, caused by members of the Ustilaginales.

Solopathogen—An isolate of a smut fungus with diploid sporidia due to failure of meiosis. The cultures are detected by infection caused by a single sporidium.

Sorus—A compact spore bearing structure characteristic of smut fungi or other fungi.

Spike—The inflorescence on an axis, or the head of cereals or grasses.

Spikelet—The appendage of a spike that consists of florets and glumes.

Spore ball—A structure made up of sori and peripheral cells, characteristic of flag smut fungi.

Sporidium—Any smut spore other than a ustilospore (teliospore).

Spring wheat—Wheat that does not require vernalization to grow and produce a crop; usually sown and harvested without interruption in the growth of the crop.

Stomata—The openings in the epidermis of a plant that are surrounded by guard cells.

Subcrown internode—The internode between the crown and the germinated seed.

Symptom—The indication of disease in a plant.

Stubble—Standing crop residue.

Substitution lines—Genetic stocks in which an entire chromosome has been replaced by an equivalent chromosome from another cultivar or species.

Teliospore—Analogous to ustilospore, a dikaryotic black resting spore, with two haploid nuclei (*n* + *n*), which become diploid (2*n*) before germinating.

Teliosporogenesis—The formation of teliospores.

Testa—The hard outer covering of a seed.

Tetraploid—A cell with four sets of chromosomes in the nucleus.

Tetrapolar—A form of heterothallism that involves two loci each with a pair of alleles that control compatibility.

Tiller—A shoot or culm of a cereal or grass plant arising from a crown bud at the ground level.

Tilletiaceae—A family of Ustilaginales the teliospores of which germinate by a promycelium (basidium) that bears terminal sporidia (basidiospores).

Transgressive segregation—A pattern of inheritance in which some progeny have superior resistance to either parent and others have inferior resistance.

Trimethylamine—A volatile chemical that causes the foul odor characteristic of bunt diseases.

Ustilaginaceae—A family of Ustilaginales the teliospores of which germinate by a promycelium (basidium) that bears lateral sporidia (basidiospores) or branches.

Ustilaginales—Order of fungi, the smut fungi, consisting of the families Tilletiaceae and the Ustilaginaceae.

Ustilospore—The resting spore of smut fungi that gives rise to the basidiospore. Analogous to teliospore.

Vacuum inoculation—Inoculation of seeds or spikes in a vacuum that is suddenly released to bring inoculum and seed surfaces into close contact.

Vernalization—The act of stimulating reproductive growth in a plant by subjecting seedlings to low temperatures away from light.

Virulence—The specific ability of a pathogen to overcome host resistance.

Winter wheat—Wheat that requires vernalization, usually sown late in the fall and harvested the next growing season.

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