

# Bunt of Wheat

The bunt fungi (*Tilletia* spp.) include some of the most devastating pathogens of mankind's most important cereal crops. The common bunt fungi, *T. caries* (DC.) Tul. and *T. foetida* (Wallr.) Liro, have caused losses to wheat production throughout recorded history wherever wheat is grown. Their early importance is reflected in the classical studies of Tillet, Prevost, and de Bary, which are among the foundations of the science and philosophy of plant pathology.

Although common bunt is no longer a major production problem, another bunt disease, called dwarf bunt, has tenaciously resisted efforts toward control. This disease, caused by *T. controversa* Kühn, while more limited in distribution and sporadic in occurrence, continues to cause occasionally severe losses to winter wheat in the northwestern United States and certain other countries. Recently, this disease achieved additional importance because it disrupted U.S. wheat exports to the People's Republic of China. Dwarf bunt has not been reported in China, and strict quarantine procedures have been implemented to prevent its introduction.



Fig. 1. Reduced height and excessive tillering of dwarf-bunt-infected plants (right) compared with uninfected plants (left) of the winter wheat cultivar Wanser.



Fig. 2. Dwarf-bunt-infected spike (left), with broadened spikelets and bunt sori showing between the glumes, compared with uninfected spike (right).

## Common Bunt

The nearly complete control of common bunt throughout much of the agriculturally developed world must be considered a major accomplishment of the science of plant pathology. This has been achieved primarily through the development and widespread use of chemical seed treatment, progressing during the last century from formaldehyde, copper carbonate, organic

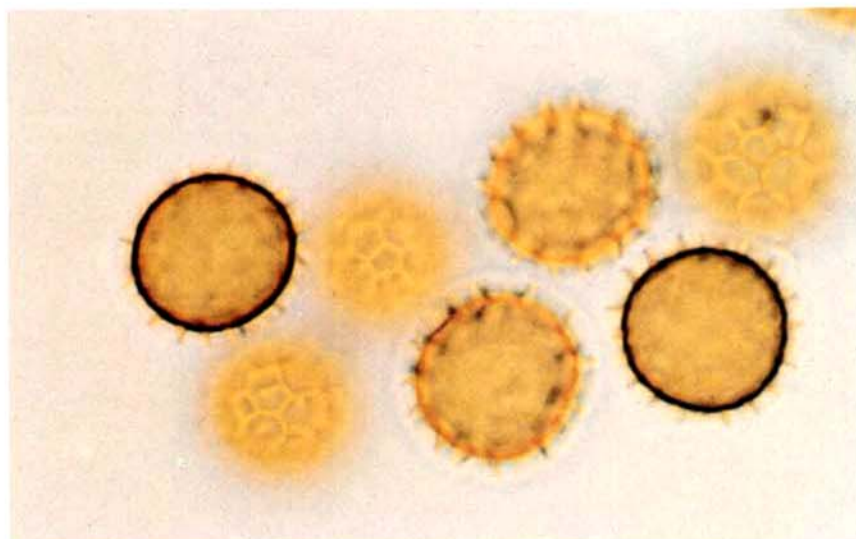


Fig. 3. Teliospores of the dwarf bunt fungus, *Tilletia controversa*. Teliospores of this species are distinguished from those of *T. caries* by the wider and deeper meshes (areolae) of the reticulate exospore and a gelatinoid sheath that typically extends beyond the exospore. These teliospores are from Kühn's type collection of *T. controversa* kindly provided by R. Durán.

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mercuries, and polychlorobenzenes to, finally, systemic fungicides such as carboxin.

The common bunt problem persisted longer in the Pacific Northwest than in other areas of the United States because infection occurred not only from seedborne spores but also from spores in the soil. Soil infestation occurs as a result of windborne spores released at harvest being deposited on fallow land and remaining through the dry summer to germinate and infect wheat sown in the fall. Soilborne inoculum was not controlled by chemical seed treatments until the advent of the polychlorobenzenes, particularly hexachlorobenzene (HCB), which are effective against both seedborne and soilborne inoculum. The preferential use of these materials as seed treatments in the Pacific Northwest since 1956 has relegated common bunt to a minor, if not a rare, problem even in this area. Nevertheless, a recent study of bunt contamination of grain in Montana (14) indicated that sufficient common bunt infection occurs to maintain a threatening level of inoculum.

Continued control of common bunt depends on continued availability and diligent use of effective seed-treatment chemicals. Whereas the number of seed-treatment chemicals available for control of seedborne bunt appears to be adequate, only four registered materials have the potential for controlling soilborne inoculum. Two of these materials, HCB and pentachloronitrobenzene (PCNB), may be withdrawn from use because of adverse impacts on the environment. Most carboxin formulations used as cereal seed treatments contain insufficient carboxin to provide adequate control of soilborne bunt, and thiazobenzazole, which is registered for use on wheat in only five western states, may be too costly to receive wide grower acceptance. Up to the present time, the strains of common bunt fungi tolerant to the polychlorobenzenes and carboxin reported elsewhere have not been detected in the United States. Nevertheless, the continued availability and effectiveness of registered seed-treatment chemicals that control both seedborne and soilborne common bunt is of concern. Fortunately, several of the newer systemic fungicides are highly effective against both seedborne and soilborne common bunt (12). Among these are triadimefon, triadimenol, CGA-64251, fenapanil, and nuarimol. None, however, is registered yet for use on wheat.

It may be of interest to compare the merits of resistant cultivars vs. chemical seed treatment for control of common bunt in the Pacific Northwest. Because of soil infestation, which until the mid-1950s was not amenable to control by seed treatment, the major emphasis of the wheat breeding programs of the Pacific

Northwest was on developing bunt-resistant cultivars. Upward of a dozen bunt-resistant cultivars were developed and each more or less rapidly succumbed to new or previously undetected races of the pathogens. In the late 1950s, breeding efforts culminated in the release of several winter wheat cultivars that combined the Martin (*Bt1*) and Turkey (*Bt4*) bunt resistance and were resistant to all known bunt races. Soon after the release of these cultivars, races of *T. caries* (T-18) and *T. foetida* (L-16) having virulence against the combined Martin and Turkey resistance were detected. Presumably, the spread of these races was held in check by HCB seed treatment, which came into wide use at about the same time. This was most fortunate, inasmuch as breeding efforts could be shifted to control of stripe rust, which became epidemic in the Northwest in 1961.

Many of the wheat cultivars developed in the Pacific Northwest continue to utilize the combined Martin and Turkey bunt resistance. Inasmuch as races virulent against this resistance have been detected in the field and are readily produced artificially, it would seem obvious that the current insignificance of common bunt is a result of effective seed treatments rather than host resistance. At the very least, seed treatment has helped to conserve the supply of bunt resistance genes and permitted breeders to extend their efforts to other pressing disease and agronomic problems. Admittedly, the cost of seed treatment has risen sharply in the last decade owing to the higher cost of development and registration of more sophisticated chemicals. Nevertheless, it should be borne in mind that the continual development of new resistant cultivars also bears a significant cost, albeit not a direct cost to growers, as is seed treatment.

Although common bunt has been relegated to an insignificant problem in much of the world, serious losses continue to occur in many agriculturally undeveloped countries. At a meeting sponsored by the International Center for Agricultural Research in the Dry Areas (ICARDA) in 1979, cereal disease specialists concluded that common bunt was second only to the rusts in importance, accounting for a 5-7% annual loss to wheat production in countries within the ICARDA region (North Africa and central Asia). In this area, probably less than 40% of the seed is treated because of cost, problems of seed and chemical distribution, and potential hazards to human health. Breeding programs leading to incorporation of bunt resistance in suitable wheat cultivars have been slow to develop. Moreover, much of the parent material used in the breeding programs consists of modern, high-yielding types that may have less bunt resistance than the "land varieties" used previously. The advance of modern

agriculture will also bring changes in farming practices, such as seeding with grain drills, mechanical harvesting and threshing, and summer-fallow rotation. Without effective seed treatments or resistant cultivars, such changes are likely to increase the incidence of seedborne bunt and, in some areas with climatic conditions similar to those in the Pacific Northwest, may promote the establishment of soilborne inoculum.

## Dwarf Bunt

Dwarf bunt, caused by *T. controversa*, is also a long-standing disease problem of winter wheat in localized areas of the western United States. A review of dwarf bunt in 1963 (20) presented much of the knowledge concerning the disease acquired to that time. It is not my intention to review the same or subsequent literature here, but rather to bring both old and new information into perspective and present a broad overview of the disease and causal organism.

### Distribution and economic importance.

Dwarf bunt has been recognized as a disease distinct from common bunt only since 1935. Symptom descriptions and herbarium records, however, indicate that the disease occurred in the United States as early as the 19th century and that it probably originated in Asia in ancient times. In addition to the United States, dwarf bunt is known to occur in Canada of North America, Argentina and Uruguay of South America, and several countries of Europe and central Asia, including Austria, Bavaria, Switzerland, France, Italy, Sweden, Poland, Czechoslovakia, Hungary, Romania, Bulgaria, Albania, Yugoslavia, Russia, Turkey, Iran, and Iraq. In general, dwarf bunt is likely to occur in any wheat-growing area where a persistent snow cover occurs regularly.

In the United States, dwarf bunt occurs in localized areas of Colorado, Idaho, Montana, Oregon, Utah, Washington, and Wyoming. The known areas of infestation are estimated to comprise about 260,000 ha. The disease has remained relatively static in distribution; a few new occurrences have been noted in Montana in recent years, whereas the disease has essentially disappeared from other areas of the Pacific Northwest and from New York and Michigan where it was reported earlier. Even in the western United States, the disease usually is not a major production problem except in certain counties of southeastern Idaho and northern Utah. During the late 1960s and early 1970s, dwarf bunt incidences were so high as to nearly eliminate winter wheat production in these areas.

Dwarf bunt occurrence is sporadic. Usually, the incidence is high only after winters with an early and persistent snow cover. In most areas of the Northwest, such conditions do not occur every year. Consequently, dwarf bunt in susceptible



cultivars may vary from trace amounts in most years to more than 60% in years when conditions are conducive to disease development. As with common bunt, yield losses from dwarf bunt are approximately equivalent to disease incidence. Even when yield losses are minor, however, bunt contamination of the grain is often sufficient to cause marketing problems and dockage penalties.

In addition to winter wheat, dwarf bunt occurs on rye, winter barley, and a diversity of wild grasses. Natural occurrence on hosts other than wheat is uncommon, however, and it is not likely that these hosts contribute significantly to the spread and perpetuation of inoculum.

**Symptoms and identification.** As with common bunt, the ultimate expression of dwarf bunt infection is the replacement of the kernel with a sorus (bunt ball) containing a brown to black, fetid mass of teliospores. Other symptoms of dwarf bunt are a height reduction of infected culms and an increase in number of tillers on infected plants (Fig. 1). The expression of both symptoms is highly variable, however, depending on the host-parasite genotypes as well as on the time and degree of infection. Wheat heads infected with dwarf bunt often are broader and have a more "ragged" appearance (Fig. 2) than uninfected heads or those infected with common bunt. This is because even those florets that are normally sterile often contain a bunt sorus. Also, the sori of dwarf bunt tend to be more spherical than the kernel-shaped sori of common bunt.

Morphologic characteristics of the teliospores are the primary basis for species delimitation in *Tilletia*. The criteria most frequently used to distinguish *T. controversa* from other reticulate-spored species are the relatively wide and deep polygonal areolae of the exospore and the presence of a hyaline sheath or capsule extending beyond the exospore (Fig. 3). However, the extreme variability of teliospore characteristics in *T. controversa* as well as in other similar-appearing species often makes positive identification difficult, if not impossible, when spore morphology is used alone. Species identification in *Tilletia* has received considerable attention recently owing to the need for positive identification of *T. controversa* in assessing contamination in grain exported to China. Several approaches have been used in attempts to provide reliable methods for definitive identification (6,22). But, at present there appears to be no way to determine with certainty whether a single teliospore is that of *T. controversa* or that of a number of other morphologically similar species.

In an attempt to assess the amount of morphologic variability and species intergradation, we examined and quan-

tified several teliospore characteristics in numerous collections of *T. controversa* and *T. caries* on wheat and in several collections of the morphologically similar species *T. fusca* occurring commonly on wild grasses. In general, morphologic characteristics showed greater variability in *T. controversa* than in the other species. Also, each of the morphologic features examined showed a considerable degree of overlap among the species. The statistical procedure of discriminant analysis was used to combine the values of several morphologic variables into a single score value. This provided a greater degree of species separation than could be obtained with any single variable. Models using combinations of two to six morphologic criteria were developed that reduced the probability of species misclassifications to about 10%.

Although a conspicuous sheath is not reliably diagnostic of *T. controversa*, its presence on most teliospores provides a sometimes useful indication of species identity in the field. The sheath is extremely hygroexpansive, and when an intact bunt ball of *T. controversa* is placed in water, the expansion of the sheaths ruptures the sorus and the spores are extruded into the water. Mature, intact sori of *T. caries* and *T. foetida*, on the other hand, show this phenomenon to a much lesser degree or not at all.

**Teliospore germination.** Dwarf bunt distribution, disease cycle, epidemiology, and problems of control are all predicated on factors associated with teliospore germination. The requirements for teliospore germination are also probably the best single criterion for dwarf bunt identification.

The dwarf bunt fungus is a cool-temperature organism. The cardinal temperatures for teliospore germination are redefined here as: minimum  $-2$  C, optimum  $3-8$  C, and maximum  $< 15$  C. Exposure to low-intensity light, at least during a portion of the incubation period, stimulates germination of all teliospore collections and is a requirement for maximum germination of most collections. Of course, adequate moisture and oxygen are also required.

Teliospore germination among and within *T. controversa* collections is extremely variable. Even with optimum conditions, the time required for maximum germination varies among collections from about 3 to 10 weeks or even longer. Typically, a few spores germinate in about 3 weeks, with the maximum percentage of germination occurring in about 6-8 weeks. Generally, teliospore collections with a short incubation period have less restrictive requirements for temperature and light than do those with a long incubation period. Occasionally, and for unexplained reasons, wide differences in amount or rate of germination occur with teliospores from the same collection from one year to another or even from one time of year to another. On the other hand, teliospores remain viable for many years over a wide range of storage conditions. In producing inocula for pathogenic race studies with *T. controversa* over the last 10-15 years, our experience has been that a medium of soil-extract agar (SEA), a temperature of  $3-5$  C, and continuous low-intensity fluorescent light or indirect daylight will produce high percentages ( $> 75\%$ ) of teliospore germination with minimum incubation of nearly all collections from wheat.

Numerous factors other than temperature and light are reported to influence teliospore germination in *T. controversa* (20). Among these are substrate, moisture, oxygen, pH, various chemical and physical treatments, and other microorganisms. In spite of many attempts, however, no treatment to our knowledge has reduced the germination period of *T. controversa* teliospores to that of *T. caries* or *T. foetida*. Nor have we found any collections of the dwarf bunt fungus in which the teliospores will germinate in appreciable amounts at  $15$  C in less than 2 weeks. Teliospores of the common bunt fungi, on the other hand, have already germinated and their germination products have largely disintegrated within this period.

With near optimal conditions of temperature, moisture, oxygen, and light, on agar or soil, the teliospores germinate

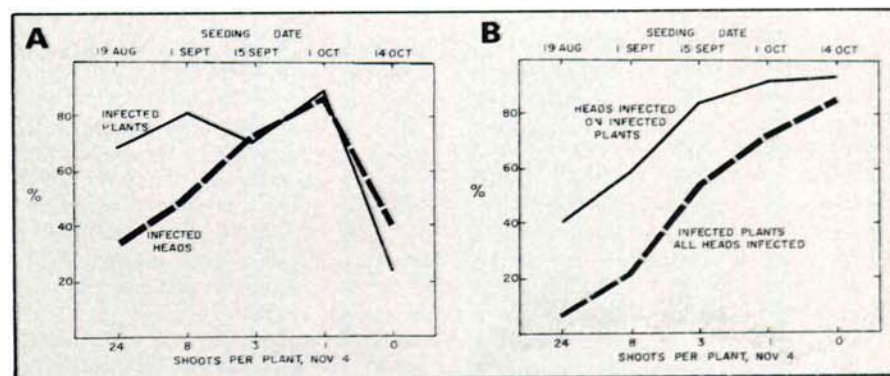


Fig. 4. Relation of wheat growth stage to dwarf bunt infection: Effect of seeding date (tiller development) on (A) percentage of infected plants and infected heads and (B) percentage of heads infected on infected plants.



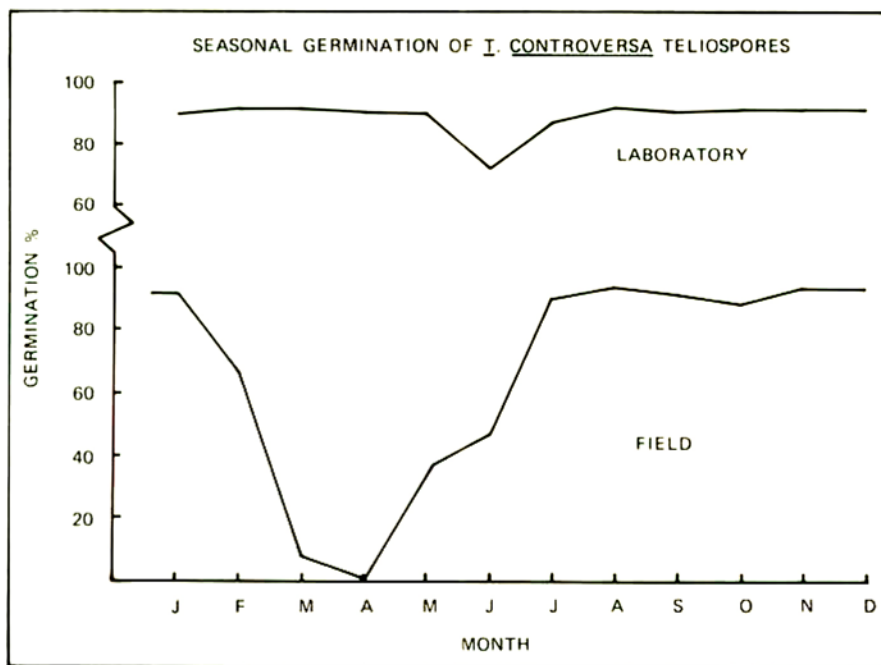


Fig. 5. Percentage germination of *Tilletia controversa* teliospores recovered at monthly intervals from the soil surface in a field at Logan, Utah.



Fig. 6. Yellowing and flecking symptoms of dwarf bunt infection in leaves of immature winter wheat.

with a very short promycelium and immediately produce a terminal whorl of primary sporidia. On soil, both in the laboratory and in nature, the promycelium is oriented upward and the whorl of sporidia is borne aerially. Under suboptimal germination conditions, the promycelia are often long and branched and sporidial production is delayed. Generally, the numbers of primary sporidia produced by teliospores of *T. controversa* are larger and more variable (usually 14-30) than those produced by *T. caries* or *T. foetida* (usually 8-12). Also, monosporidial cultures of *T. controversa* tend to be mycelial with few

secondary sporidia, whereas those of the common bunt species tend to be sporidial. In both common and dwarf bunt fungi, secondary sporidia may occur as either or both of two morphologic types: a filiform type similar in size and shape to primary sporidia and a reniform type of two sizes, the smaller usually uninucleate and the larger, binucleate. The filiform type predominates in liquid culture, whereas both types are variably produced on solid media.

**Disease cycle and epidemiology.** Because of the long incubation period and cool temperatures required for teliospore germination, infection of the germinating seed from spores on the seed or in the soil is largely precluded or at least rare under normal field conditions. All evidence indicates that dwarf bunt infection originates from teliospores germinating at or near the soil surface. Teliospore germination on the soil surface occurs as early as mid-October and may extend over a period of 3-4 months. Periods of unfavorable temperature, including freezing temperatures, suspend or delay germination but have little effect on the viability of teliospores or their germination products. Observations of teliospores incubated on moist soil in the laboratory and subjected to different temperature regimes indicate the primary sporidia remain viable for long periods (8-12 weeks) at temperatures of -5 to 5 C. The factors most likely to reduce the longevity and infective potential of the germination products are exposure to direct sunlight or a dry atmosphere that would cause desiccation and lysis of the primary and secondary sporidia and infection hyphae. Presumably, a persistent snow cover, which is consistently correlated with high disease

incidence, not only provides a continuous temperature favorable for spore germination but also protects the infective germination products from desiccation. At the soil surface beneath snow, within a temperature range of -2 to 2 C, teliospore germination can occur continuously and in high amounts. A saturated atmosphere and, at least occasionally, free water are also present. Moreover, penetration of light is not completely prevented by even a considerable depth of snow.

The site of penetration of the host plant is presumed to be the tiller initials. Studies on time of infection (19) and those relating disease incidence to stage of host development (11) indicate a close correlation between infection and tiller development, but direct evidence from histological studies of naturally infected host plants has not been obtained. Studies on the pathologic histology of both *T. caries* and *T. controversa* describe penetration and infection after artificial inoculation of the wheat coleoptile (3,5). These results, however, may bear little relationship to events occurring with *T. controversa* in nature.

In what we believe to be a definitive study (19), dwarf bunt infection was shown to occur after seedling emergence from December through early April, with most infection occurring during January and February. Date of seeding, or more precisely stage of host development during the infection period, is closely related to dwarf bunt incidence. Plants entering the infection period with only a few tillers generally have the highest incidence, whereas plants with many tillers or those that have not yet emerged at the onset of infection have a low incidence or escape infection entirely. Also, the number of plants completely bunted and the number of bunted heads per plant increase with lateness of seeding and, conversely, decrease with advancing plant development (Fig. 4). This is because in older plants, which have a larger and more differentiated crown, some tillers are beyond the point of susceptibility or are out of reach of the invading mycelium. In young seedlings, however, the entire crown area may be invaded by mycelium before many tillers are differentiated.

Conditions of temperature and moisture that induce dwarf bunt spore germination and infection in late fall and winter occur to some extent also in spring. Thus, spring-sown wheat also could be expected to become infected occasionally with dwarf bunt. To our knowledge, however, dwarf bunt infection in spring-sown wheat has not been observed. Recent studies at our laboratory suggest that teliospore dormancy in spring rather than environmental conditions unsuitable for spore germination and infection may be the reason, at least partially, for the lack of infection in spring-sown wheat. In

experiments repeated over several years, teliospores recovered from the soil surface and plated during summer through late winter germinated in maximal amounts (75–95%), whereas those recovered in late spring (April or May) germinated in only trace amounts (< 1%) (Fig. 5). Preliminary results suggest that teliospore dormancy is induced by protracted cool, moist conditions and is broken by a period of warm, dry conditions.

Another aspect of dwarf bunt epidemiology differing from that of common bunt is the longevity of *T. controversa* teliospores in soil and the perennial nature of soil infestation. Whereas teliospores of the common bunt fungi in or on soil lose viability within 2 years, those of *T. controversa* remain viable for at least 3 years as free spores and up to 10 years in intact sori. Because of the dwarfing effect on the host induced by dwarf bunt infection, bunted spikes are often passed over during harvest and remain in the wheat stubble. During plowing or discing, the bunted spikes are distributed in and on the soil more or less intact. During subsequent cultivation or by weathering, the bunted spikes are broken up, releasing spores over a period of several years. Consequently, in contrast to common bunt, annual reinfestation of the soil with the dwarf bunt fungus is not necessary to maintain a high level of infective soil inoculum. This accounts for a high incidence of dwarf bunt occurring after several years of low incidence, after several years of fallow, or after removal of a perennial crop such as grass or alfalfa.

**Methods of inoculation.** Inasmuch as dwarf bunt infection rarely occurs from seedborne spores and is highly dependent on irregularly occurring environmental conditions, much attention has been given to the development of reliable methods of artificial inoculation. In field experiments involving many rows or plots and in which the same teliospore population can be used throughout, inoculum may be provided or enhanced by applying teliospores as a water suspension to the soil surface after seeding or after seedling emergence. The effectiveness of this procedure, however, depends on weather conditions, particularly a persistent snow cover. Various artificial ground covers, including vermiculite, wood shavings, oat straw, and polyethylene sheeting, have been used to simulate a snow cover (1). Such covers usually enhance dwarf bunt infection, but their use on a large scale may be prohibitively costly or laborious. Moreover, under some conditions, the increase in bunt incidence may be offset by a loss of plant stands.

In studies requiring inoculation with different teliospore collections or where only limited quantities of spores are available, seed or seedlings are inoculated

with germinating teliospores. To reduce contamination during the long germination period, the teliospores are suspended in a 5% Clorox solution for about 1.5 minutes, then rinsed with two changes of sterile water. The teliospores in a water suspension are placed on SEA and incubated at 5 C under light. When the spores attain maximum germination, the seed to be inoculated and a small amount of water are placed in the dish with germinating spores and stirred to thoroughly cover the seed with spores and spore germination products. The inoculated seed is placed in moist vermiculite and incubated at 10–15 C until the seedlings are 4–6 cm long. The seedlings are then planted in soil in peat pots, vernalized, and transplanted to the field in spring. Generally, this procedure results in high incidences of infection in susceptible wheat cultivars. The number of inoculations is limited by the time and labor involved, but this has been the inoculation method used in our pathogenic race studies with dwarf bunt since 1961.

Dwarf bunt infection can be obtained in susceptible wheat cultivars and wild grasses by spraying a suspension of germinating teliospores onto plants in the 2–3 leaf stage. Following inoculation, the plants (usually in pots) are enclosed in plastic bags and placed at 10–15 C for 10–14 days. The plants are then vernalized (if necessary) and grown to maturity in the greenhouse or transplanted to the field.

Both of these methods employing teliospore germination products as inoculum are adaptations of methods developed by Meiners (15), and both work equally well with common bunt and other bunt fungi. Generally, any procedure that places spore germination products in the vicinity of susceptible host tissue under appropriate conditions of temperature and moisture will result in infection.

Inoculation of seed or seedlings has a considerable disadvantage in that several months must elapse between inoculation and production of sori in the mature host plant. This period may be shortened to 3–4 weeks by an inoculation procedure recently described by Fernandez and Durán (2). They obtained some bunt sori, but not completely bunted spikes, by injecting a suspension of spore germination products into the region of the spike of wheat plants in the flag-leaf stage of development. This procedure may be useful for rapidly producing successive generations of teliospores in genetic studies with bunt fungi.

**Control by resistant cultivars.** Because of the lack of effective seed treatments, host resistance has been the primary means of dwarf bunt control. Control by host resistance is complicated by a high degree of pathogenic specialization in both the common and the dwarf bunt species. New combinations of virulence

genes are produced as a result of hybridization, and virulent types are rapidly selected from the bunt populations by host screening. Consequently, bunt resistance in a wheat cultivar has become accepted as a transient attribute, its longevity depending on the dynamics of the bunt population to which it becomes exposed.

Development of wheat cultivars resistant to dwarf bunt is an important aspect of the winter wheat breeding programs of most northwestern states. Up to about the mid-1960s, breeders relied primarily on the Martin (*Bt1*), Turkey (*Bt4*), and Redit (*Bt3*) sources of bunt resistance, first singly and then in combination. However, races virulent against the combined Martin, Turkey, and Redit resistance appeared about 1954 in widely separated areas, necessitating the use of entirely new resistance sources for control of dwarf bunt. Fortunately, screening of the USDA World Wheat Collection for bunt resistance sources (13) had identified several that were resistant to all known bunt races, including those virulent against *Bt1*, *Bt3*, and *Bt4*. Among these new resistance sources was a wheat collection from eastern Turkey, designated PI 178383. In addition to having excellent bunt resistance, this wheat has resistance to stripe rust and flag smut and a useful degree of tolerance to snow mold. Consequently, it has been widely used in the wheat breeding programs of the Northwest. Subsequent genetic studies (17) have identified three genes for bunt resistance in PI 178383, designated *Bt8*, *Bt9*, and *Bt10*. These genes, singly and in combination, provide the resistance to dwarf bunt in all the winter wheat cultivars grown in the Northwest at the present time, including the hard-red cultivars Cardon, Crest, Franklin, Hansel, Jeff, Manning, Ranger, and Weston and the soft-white cultivars Luke and Moro.

Virulence against the resistance of PI 178383 has already been detected in the dwarf bunt population and most, if not all, of the currently grown cultivars are susceptible to one or more races. Consequently, the need for other new sources of bunt resistance in the wheat breeding programs is urgent. At the present time, the amount and diversity of useful bunt-resistant germ plasm appear to be dangerously limited. Less than a dozen sources are known and the number of different resistance genes represented in these sources may be considerably less than that. Wheats currently recommended as potentially useful sources of bunt resistance include CI 14106, PI 119333, PI 166910, PI 211657, and PI 245579.

Most new sources of bunt resistance that have been identified have originated from eastern Turkey, where both common and dwarf bunt are endemic. A germ plasm collection expedition

conducted in eastern Turkey in 1979 provided seed of several hundred additional native wheats and wheat relatives. Screening of this material, as well as other new entries in the World Wheat Collection and other germ plasm repositories, hopefully will identify new and different sources of bunt resistance.

Among the resistance genes used for control of dwarf bunt, *Bt1* and *Bt3* have shown greater durability than others, at least in the northwestern United States. Moreover, preliminary observations suggest that the frequency of cultures expressing virulence against *Bt1* and *Bt3* declines when selection pressure is removed. However, a decrease in frequency of cultures having virulence against *Bt2*, *Bt4*, *Bt6*, and *Bt7* has not been observed despite reduced selection pressure. In this context, it is interesting to note that virulence against *Bt9* and *Bt10* was detected in both the common and the dwarf bunt fungi before these resistance genes were used in breeding programs.

The use of general or nonspecific resistance to bunt has been largely ignored in most wheat breeding programs, and apparently little has been done to identify sources of general resistance or to determine its potential usefulness. That general resistance to bunt occurs is suggested by additive interactions for increased resistance from genes that are not effective singly and by different levels of bunt incidence expressed by specific resistance genes in different genetic backgrounds (18). Although it may be argued that general resistance alone would not provide an adequate level of control, its use would perhaps prolong the effectiveness of what appears to be a limited supply of specific bunt resistance genes.

The mechanisms of host resistance to the bunt fungi remain a fertile area for investigation. Studies involving artificial inoculation of the wheat coleoptile with common and dwarf bunt fungi indicate that both susceptible and resistant cultivars are penetrated by hyphae and initially invaded (3,5). In susceptible cultivars, the fungus reaches the floral primordia before internodal elongation and ultimately sporulates. In resistant cultivars, the fungus hyphae invade the juvenile tissue of the crown to a greater or lesser extent but usually fail to reach the floral primordia. In any case, the highly variable expression of the host-parasite interaction suggests that several mechanisms of resistance may be operative, at least in dwarf bunt. Different host-parasite combinations may express differences in amount of leaf flecking or mottling, culm dwarfing, tiller stimulation, bunted spikes per plant, or bunted florets per spike. Sometimes, these differences may be a reflection of the stage of plant development at the time of infection that determines the duration of the host-

pathogen association. Differences in symptom expression, on the other hand, can be correlated also with specific host-parasite genotypes. For example, certain wheat cultivars show a severe flecking reaction in the seedling stage, which is an obvious and demonstrable indication of bunt infection, but no bunt sori will be produced in the spike. Other cultivars may show little or no flecking as seedlings but will have completely bunted spikes at maturity. Under conditions promoting a high infection potential, even highly resistant cultivars will often have a few bunted florets.

**Pathogenic specialization.** Identification and classification of pathogenic races of dwarf bunt were hampered initially owing to the lack of inoculation methods that would produce consistently high levels of infection. Eight races were reported in the first classification of dwarf bunt races in 1962 (9) and 17 were reported in 1976 (10). Since then, numerous additional pathogenic types have been identified. Some of these are virulent against the combined *Bt9* and *Bt10* resistance genes from PI 178383. Recent preliminary results indicate virulence in the dwarf bunt population also against *Bt8*, the third bunt resistance gene in PI 178383. Since all the dwarf-bunt-resistant cultivars currently grown in the Northwest derive resistance from this source, all are vulnerable to these new races.

Recent results indicate that few, if any, dwarf bunt races express virulence against the Hohenheimer resistance (*Bt5*). Previous reports indicating virulence against *Bt5* were based on the reaction of an Elgin × Hohenheimer selection that apparently contained only a part of the Hohenheimer resistance. Tests using the cultivar Hohenheimer itself indicate little, if any, virulence against *Bt5* in the dwarf bunt population in the western United States. Up to the present time, Hohenheimer has not been used as a source of bunt resistance because of its undesirable agronomic characteristics. Moreover, it is susceptible to several races of the common bunt fungi.

Virulence has been detected in *T. controversa* against nine of the 10 bunt resistance genes thus far identified, and combined virulence against eight resistance genes has been found in several spore collections. Thus, accumulation of virulence genes in the dwarf bunt fungus appears to be unlimited with regard to number or diversity. The rapidity with which different pathogenic types are detected suggests that a diversity of virulence combinations is already present in a highly heterogeneous and/or heterozygous population. It is likely that the ascendance of new pathogenic types only awaits the selective action of the appropriate combination of resistance genes in the host.

Pathogenic races of the dwarf bunt fungus appear to be less stable than those of the common bunt fungi. Frequently, teliospores from a single dwarf-bunted spike will yield several pathogenic types on reselection from differential wheat cultivars. Also, what initially appear to be stable pathogenic races may later show changes in virulence. The propensity for variation in virulence, as well as other characteristics, in *T. controversa* compared with *T. caries* or *T. foetida* may be due to several features peculiar to the dwarf bunt organism that would seemingly lead to a greater degree of outcrossing. The fact that dwarf bunt spore germination and infection occur at the soil surface rather than in the soil, as is the case with common bunt, would presumably allow a greater opportunity for interteliosporic crossing. Also, a larger number of primary sporidia and the multiallelic mating system in *T. controversa* (8) would also seem to favor a greater degree of outcrossing in this species than in the common bunt species. Perhaps also, nuclear exchanges may occur among genetically diverse hyphae in the host. This, however, has not been experimentally demonstrated. Indeed, too little is known of the genetics of the bunt fungi to provide sound hypotheses for explaining the mechanisms of variation.

Because of the longevity of *T. controversa* teliospores in soil and the difficulty in obtaining dwarf bunt infection naturally or artificially, initial screening for host resistance is best accomplished by inoculation with appropriate races of the common bunt fungi. Such studies have been greatly aided by the development of new races of common bunt fungi that carry virulence genes equivalent to those carried by the more broadly virulent races of *T. controversa*. These were obtained by inoculating a Hohenheimer × Elgin selection with common bunt races, either singly or in combination. The genetic mechanism involved in the production of new races by this means is obscure, but five new races of *T. caries* have been obtained in this manner (16).

**Chemical control.** The contrasting effectiveness of nonsystemic fungicides used as seed treatments against common and dwarf bunt emphasizes the basic differences in the biology of the causal organisms. Control by chemicals is contingent upon bringing the chemical to bear in the immediate vicinity of the point of infection. Consequently, seed treatment may be effective against both seedborne and soilborne common bunt, but unless the chemical becomes systemic, it has no value as a protectant against infection from dwarf bunt. On the other hand, application of certain chemicals (eg, HCB, PCNB) to the soil surface after seeding does provide control of dwarf bunt. Apparently, this has been the

primary means of dwarf bunt control in Europe but has not been adopted in the United States because of economic or environmental considerations.

The potential for controlling dwarf bunt by chemical seed treatment became apparent with the development of systemic fungicides. Routine testing of systemic fungicides in 1969 showed that thiabendazole was effective as a seed treatment not only against seedborne and soilborne common bunt but also against dwarf bunt, at least under certain conditions. Subsequent studies showed that effectiveness of thiabendazole varied with seeding date, i.e., effectiveness was increased by late seeding and reduced by early seeding (7). It was concluded that the increased plant growth resulting from early seeding reduced the concentration of fungicide within the plant to an ineffective level.

Thiabendazole was registered for use on wheat in five western states in 1979. Use of the material to control dwarf bunt has not been great, however, because of its relatively high cost and the availability of dwarf-bunt-resistant cultivars. Moreover, thiabendazole's effectiveness in commercial use has been less than satisfactory because of grower's insistence on early seeding and the uneven distribution of the chemical on seed obtained with commercial seed treaters. The only other materials that have shown efficacy comparable to that of thiabendazole as seed treatment against dwarf bunt are triadimenol and CGA-64251. However, CGA-64251 has shown phytotoxicity at effective rates and neither material is registered for use on wheat.

In view of the elapsed time between seeding and dwarf bunt infection (3-4 months), it is surprising that systemic fungicides applied as seed treatments are even partially effective against dwarf bunt. Because of the numerous problems involved, it may be expecting too much of any seed treatment material to provide the dependable control realized with common bunt and other smut diseases.

The application of effective systemic fungicides in spring after infection has occurred may represent the most ideal form of chemical control for dwarf bunt. Often, dwarf bunt infection can be recognized in spring by the flecking or yellowing of leaves of infected plants (Fig. 6). Also, dwarf bunt infection can be predicted with considerable reliability on the basis of weather conditions during winter. Consequently, chemical treatment would be used only when necessary, sparing the grower the expense of treatment when conditions are not conducive to dwarf bunt development, as is the case in most years. Unfortunately, none of the systemic fungicides tested thus far has shown efficacy as an eradicator of dwarf bunt when applied to wheat plants in the spring.

**Cultural control.** In addition to



resistant cultivars and chemicals, certain cultural practices may reduce the incidence of dwarf bunt. Spring-sown wheat can be grown instead of winter wheat, but the yield of winter wheat may be greater even with losses from dwarf bunt.

As mentioned previously, stages of winter wheat development most susceptible to dwarf bunt infection may be avoided by either very early or very late seeding. Extreme variation from the normal seeding time, however, may increase the potential for other disease problems or reduce yields. A considerable reduction in dwarf bunt incidence usually results from seeding deeper than 6-8 cm, although the reasons for this remain obscure. Soil compaction favors dwarf bunt incidence either as an indirect effect on seeding depth or as a direct effect on physical factors that promote spore germination or infection. Thus, soil compaction should be avoided.

The effects on dwarf bunt incidence of such other management practices as spring or fall plowing or discing of stubble or minimum or no tillage have not been investigated. These practices could have significant effects on spore longevity as well as on other factors involved in dwarf bunt epidemiology and may suggest additional options for control.

### Future Considerations

It has been more than 200 years since Tillet discovered the contagious nature of *T. caries*. In that length of time, one could surmise that there is little left to be discovered about the biology and control of the bunt fungi. Most definitely, that is not the case. There are yet significant gaps in our knowledge of these important and extensively studied pathogens. For example, the details of mitotic nuclear

divisions have been reported only recently (4), and the details of meiosis remain hidden within the thick-walled teliospore. The chromosome number of not only *Tilletia* but of all the smut fungi is still a subject of controversy.

Not much is really known of the genetics of the bunt pathogens. It would appear that the *Tilletia-Triticum* host-parasite system would be ideally suited to studies of the genetics of virulence and mechanisms of resistance. Intraspecific and interspecific hybridization of the pathogens is easily initiated and near-isogenic wheat cultivars having single bunt resistance genes are available. However, in spite of the fact that bunt dikaryons have been induced to sporulate in culture (21), the bunt fungi are obligate parasites and production of teliospores independent of a living host is not a matter of routine. Production of generations of teliospores in the host plant is time-consuming, and most artificially produced hybrids lack vigor or aggressiveness. Consequently, genetic studies with the bunt fungi have not kept pace with such studies in other host-parasite systems. Much more needs to be done.

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**James A. Hoffmann**

Dr. Hoffmann is a research plant pathologist in the USDA-ARS Crops Research Laboratory, Logan, Utah, and adjunct professor in the Plant Science Department of Utah State University. His research focuses on the biology and control of the bunt diseases of cereals and wild grasses. After receiving his Ph.D. from Washington State University in 1961, he was with the USDA Regional Cereal Disease Research Laboratory at Pullman, Washington. He relocated to Logan in 1972.