Edward J. Trione
ARS, USDA, and Oregon State University, Corvallis

Dwarf Bunt of Wheat and Its Importance in International Wheat Trade

In 1980, over 450 million bushels of wheat were produced in the five northwestern states—Oregon, Washington, Idaho, Montana, and Utah—where dwarf bunt disease commonly occurs. In addition, some of the wheat produced in other western states is transported to and shipped from ports in the Pacific Northwest. About 85% of the wheat produced in this region of the United States is exported, primarily to Asian countries. The largest buyers in recent years have been Japan, Korea, Pakistan, Iran, Bangladesh, and Taiwan. The People's Republic of China is one of the world's largest wheat buyers but has purchased little wheat from the northwestern United States during the last 8 years.

In the early 1970s, wheat trade was resumed between the United States and the People's Republic of China, and we exported about 25 million bushels of wheat to China during 1972–1973. Some of those shipments of wheat were contaminated with spores of the bunt fungi. Common bunts of wheat, caused by *Tilletia caries* (DC.) Tul. and *T. foetida* (Wall.) Liro, are present in China, but dwarf bunt of wheat, caused by *T. controversa* Kühn, has not been reported in that country. Late in 1973, China set a "zero tolerance" on spores of *T. controversa* and rejected several shipments of wheat containing spores of the fungus.

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In 1974, all shipments of wheat from the Pacific coastal states to China were stopped because they could not be certified as free from spores of the dwarf bunt fungus. As a result, the northwestern United States lost a potential wheat market of 80–100 million bushels annually.

Canada, Australia, and Argentina have sold large quantities of wheat to China in recent years. In 1980, wheat supplies from these countries were

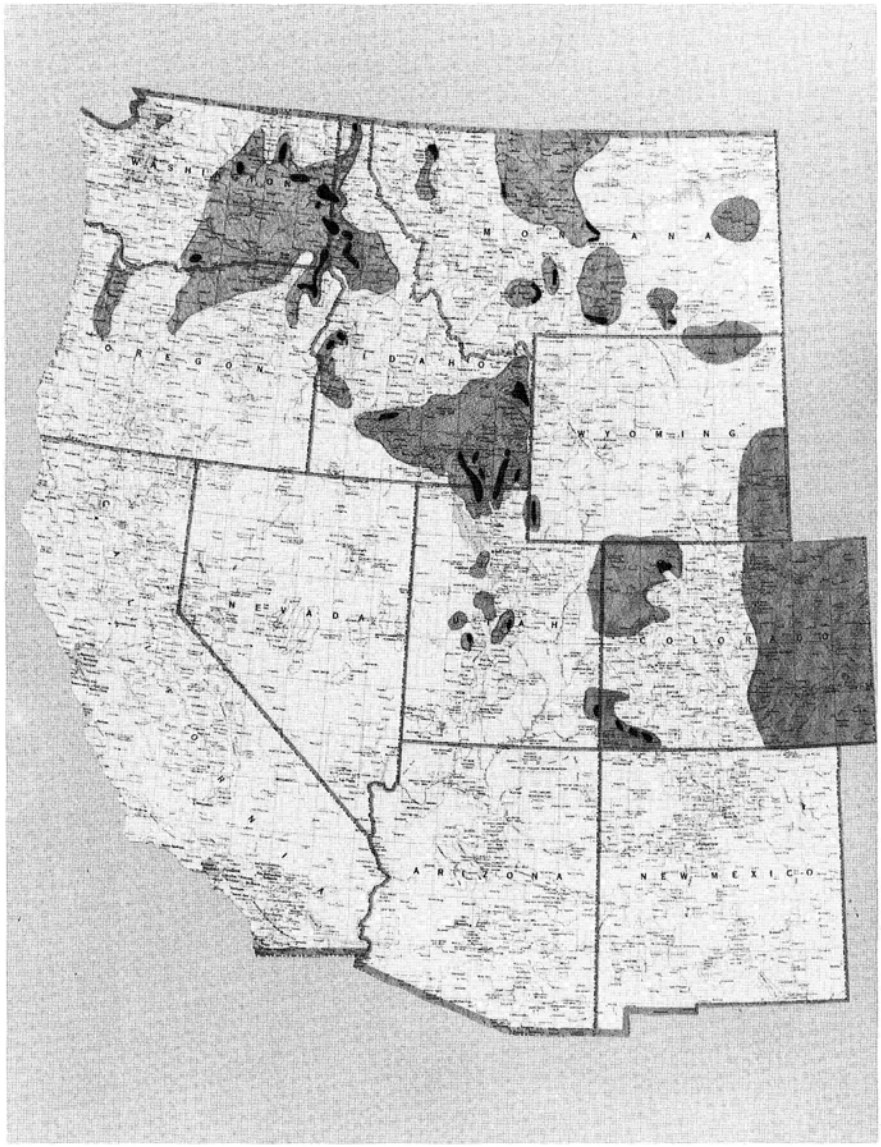


Fig. 1. Locations of dwarf bunt of wheat infestations in the western United States. The gray areas indicate winter wheat regions and the black areas represent dwarf bunt infestations.

limited and wheat prices in the United States were attractive, so China purchased an estimated 13.5 million metric tons of wheat from the United States. A portion of that wheat will come from the Pacific Northwest.

Distribution of Dwarf Bunt

Studies of herbarium specimens have revealed the existence of dwarf bunt disease of wheat in Czechoslovakia in 1847, Germany in 1858, the United States (New York) in 1860, Austria in 1884, Iraq in 1908 (2), and Argentina in 1915 (8). The earliest herbarium records of dwarf bunt of wheat in the United States are: New York 1860, Michigan 1890, Indiana 1917, California 1917, Utah 1918, and Oregon 1921 (2).

The present locations of dwarf bunt infestations in the western United States are shown in Figure 1. In such areas as eastern Washington and southeastern Idaho where heavy infections have occasionally occurred, the disease is found only in certain specific fields and the total infected acreage represents only a small percentage of the total acreage in the infested area. Furthermore, even the wheat fields that are most severely attacked by the pathogen do not have the disease every year. A long, persistent snow cover is an important climatic factor aiding development of the disease.

Although dwarf bunt disease of wheat occurs on relatively few acres in the Northwest, the problem has been greatly magnified because the dustlike spores of *T. controversa* are spread through combines, trucks, railcars, elevators, and ships carrying wheat down the Columbia River for export from Oregon and Washington ports. As a result, clean wheat seeds become contaminated with the spores of *T. controversa* during grain handling and transportation. This widespread distribution of dwarf bunt spores makes it appear that all wheat shipments from Pacific Northwest ports are contaminated with these spores. Therefore, this is not solely a regional problem, as wheat from 10 major wheat-producing states funnels through Pacific Northwest ports.

History of Dwarf Bunt

The bunt diseases of wheat are historically important plant diseases, since they were among the first studied. In 1755, Tillet reported that bunt disease of wheat was caused by wheat seeds becoming contaminated with the dustlike powder (spores) from diseased kernels. In 1807, Prevost demonstrated that the bunt spores germinate and infect the wheat plant and that the infection takes place in very young seedlings. This research on the parasitic nature of wheat bunt preceded Pasteur's historical work in the 1860s demonstrating that microorganisms cause certain animal diseases. Thus, the germ theory of disease, attributed to Pasteur, should rightfully be attributed to Prevost on the basis of his classical research on wheat bunt diseases in 1807.

Before 1935, the dwarf bunt pathogen was considered to be a form of the common smut fungus *T. caries*, but it was later assigned to *T. controversa* (1). Conners (1) suggested that wheat probably first contacted the dwarf bunt disease from native grasses (*Agropyron* spp.) in the mountainous areas of Europe and that diseased seeds were subsequently carried to many wheat-growing regions. Riehm (11) believed that *T. controversa* originated more recently as a mutant of *T. caries*. It is interesting to note, however, that the dwarf bunt disease of wheat is common throughout eastern Turkey, Iran, and northern Pakistan. This region is the center of origin of wheat, and it is quite probable that the bunt diseases have been evolving together with wheat in that region for thousands of years.

Life Cycle

The life cycle of the dwarf bunt fungus begins with germination of the dormant teliospores (Fig. 2). The germination process is slow (1–3 months) and has an obligate requirement for low temperature (1–10 C). The promycelium (germ tube) usually grows to a length 1–3 times the diameter of the teliospore and then produces a whorl of spindle-shaped primary sporidia at its tip. While the sporidia are still attached to the promycelium, compatible sporidia (+ and −) usually fuse in pairs with a conjugation tube. The fused sporidia undergo plasmogamy (but not karyogamy), separate from the promycelium, then germinate to produce a binucleate hypha. The binucleate hyphae are pathogenic to susceptible wheat varieties (5).

Primary sporidia that do not mate germinate to produce haploid, mononucleate hyphae that grow readily on many common media but are not pathogenic to wheat. Although long considered to be obligate parasites, *T. caries* and *T. controversa* have now been cultured axenically through their entire life cycle (12,14). Secondary sporidia are commonly produced in haploid cultures of *T. caries* but rarely in cultures of *T. controversa*.

In the Pacific Northwest, infection of winter wheat by the dwarf bunt fungus takes place from soilborne inoculum several months after the autumn rains begin and the teliospores have received sufficient cold to satisfy the germination requirements. The teliospores do not all germinate at once in a synchronous manner, but rather show a low level of germination over a period of several months. The effect of this long, slow germination period is to extend the time

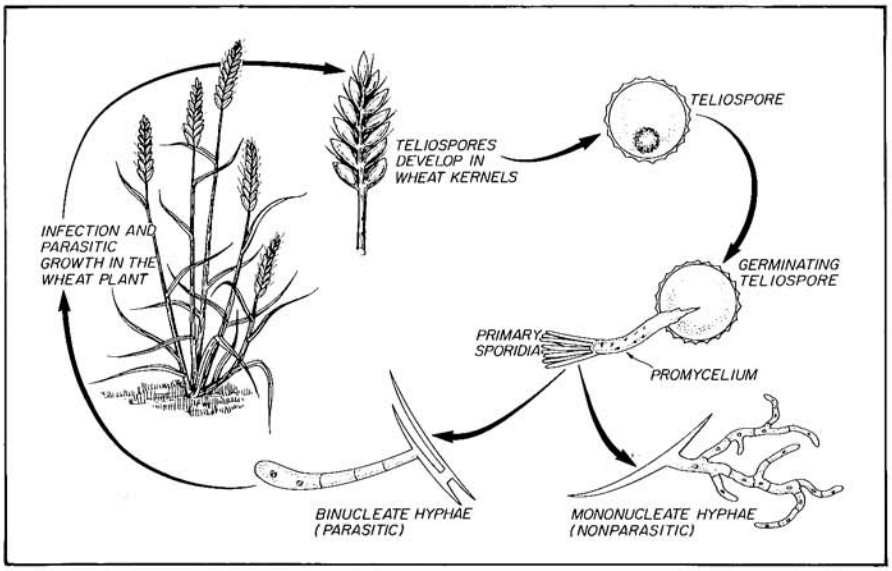


Fig. 2. Life cycle of the dwarf bunt fungus, *Tilletia controversa* Kühn.

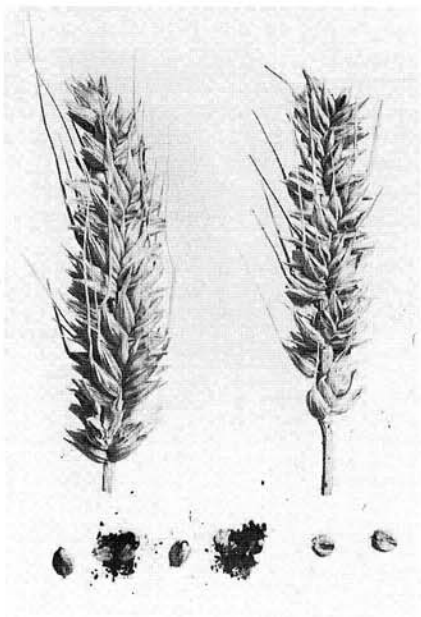


Fig. 3. Dwarf-bunt-infected wheat heads and "bunt balls"; crushed bunt balls illustrate the spores.

of initial seedling infection from December through early April.

The binucleate hyphae of the dwarf bunt pathogen penetrate the young wheat seedlings at multiple points. The pathogenic hyphae grow slowly toward the apical meristematic tissue but remain quiescent in the apex until the wheat plant has received the proper photoperiod for floral induction (4,6). As the bunt-infected wheat plant approaches the heading stage and the kernel tissue begins to enlarge, the pathogen changes from its slow-growing vegetative stage to a very rapid reproductive phase. Fungal teliospores replace the entire contents of the wheat kernel within the seed coat to form the "smut" or "bunt ball." During mechanical harvest and transport of wheat seeds, the fragile bunt balls are commonly ruptured and release the millions of dustlike spores from inside each kernel. In the field, these spores are carried by the wind, but they eventually settle to the soil to set the stage for infection of subsequent wheat crops.

Symptoms

It may be possible to obtain preliminary evidence of dwarf bunt infection by noting two symptoms, increased number of tillers and leaf flecks, before the main stem of the wheat plant elongates. Tillering is usually stimulated by dwarf bunt infection, with infected plants having about twice as many tillers as comparable healthy plants. Some bunt-infected wheat varieties show a flecking or mottling reaction on the seedling leaves. This flecking reaction is not expressed consistently, however, even by the same pathotype on the same variety. Furthermore, the flecking may be caused by infections by the common bunt fungi as well as those by the dwarf bunt fungus.

The most dramatic symptom of dwarf bunt disease after the wheat plants have been induced to flower and stem extension begins is the extreme dwarfing displayed by some infected varieties. Infected plants are always dwarfed to some degree, but the extent of dwarfing varies considerably and is strongly influenced by the particular pathogenic race/host variety combination. Usually the dwarf-bunt-infected plants are reduced to one-third to two-thirds the height of comparable healthy plants. Wheat plants infected with some pathotypes of common bunt may also be reduced in height, eg, to two-thirds the normal height. Thus, there is some overlapping at the upper end of the dwarfing scale in the effects of common bunt and dwarf bunt. Another important point is that some semidwarf wheat varieties show little dwarfing effect even when infected by *T. controversa*. Therefore, the dwarfing symptom cannot be relied on as the sole symptom indicating dwarf-bunt-infected wheat plants.

The dwarf-bunt-infected kernels (sori or bunt balls) are larger and more nearly spherical than kernels infected with the common bunt fungi. These enlarged bunt balls tend to spread the lemma and palea of the spikelets and expose the dwarf bunt sori to a greater extent than in wheat heads infected with common bunt. Fertilization of the wheat ovary is not required for the development of bunt sori, and dwarf-bunt-infected wheat heads commonly contain more bunt sori than comparable healthy wheat heads contain seed. Therefore, since the bunt sori are larger and more abundant than seeds, the dwarf-bunt-infected wheat head is larger in all dimensions than comparable healthy or common-bunt-infected wheat heads (Fig. 3).

Host Range

The host range of the dwarf bunt pathogen is very similar to that of the common bunt fungi. On the basis of data from natural infections and experimentally inoculated plants, the current host list of the dwarf bunt pathogen includes: 6 species of *Aegilops*, 22 of *Agropyron*, 1 of *Agrostis*, 2 of *Alopecurus*, 1 of *Arrhenatherum*, 2 of *Beckmannia*, 5 of *Bromus*, 1 of *Dactylis*, 8 of *Elymus*, 5 of *Festuca*, 1 of *Holcus*, 6 of *Hordeum*, 1 of *Koeleria*, 4 of *Lolium*, 2 of *Poa*, 1 of *Secale*, 1 of *Trisetum*, and 3 of *Triticum* (10).

Although in experimental trials the dwarf bunt fungus can attack many grasses other than wheat, the natural occurrence of dwarf bunt in grasses is rather limited (7), and the small amount of the dwarf bunt disease on wild grasses does not apparently influence the perpetuation of the disease on wheat (10). It is even less likely that *T. controversa* spores produced on wild grasses contribute significantly to the bunt spores found in wheat shipments from the Pacific Northwest.

Factors Favoring Infection, Development, and Spread

Infection by the dwarf bunt fungus originates almost exclusively from soilborne inoculum, whereas infection by the common bunt fungi originates from either soilborne or seedborne spores. The time required for maximum infection by common bunt fungi varies from 1 to 3 weeks after planting, ie, before or shortly after seedling emergence. In comparison, the dwarf bunt fungus does not infect such young wheat seedlings because the spores require a long cold period for germination to begin and an even longer period for the inoculum density to reach the threshold level for infection. In the Northwest, dwarf bunt infections begin in December and extend into early April, with most occurring in January and February (10). The wheat seedlings are beginning to tiller and have several leaves at that stage of development. Some dwarf bunt infections probably occur in late April or May, but stem elongation is in progress at that time and the invading hyphae may not reach the growing point; therefore, the plants may be infected but the wheat heads remain healthy.

After penetrating the wheat tissue, the dwarf bunt pathogen ramifies in the intercellular spaces of the host, but the fungal cytoplasm generally stays with the growing hyphal tips. Little hyphal branching occurs and the total hyphal cytoplasm does not increase markedly. Most of the wheat tissue, even in susceptible varieties, appears to be incompatible for extensive hyphal growth and development. The pathogenic hyphae grow slowly in a directed manner toward the unique, minute tissue in the apical region between the growing point and the developing basal nodes (4,6). In the apical wheat tissue, the pathogenic hyphae remain small and quiescent for months. Once floral development begins, wheat tissue grows very rapidly and the pathogenic hyphae are carried passively upward, in the apical tissue, owing to internode elongation. At this stage, the pathogen is also activated and grows throughout the apical tissues in an almost inconspicuous manner, not disturbing the normal physiology of the host. Stem elongation and the complicated morphological changes of floral development of the wheat plant proceed normally. Once ovarian tissue is differentiated, however, the pathogen changes dramatically from a slow-growing vegetative form to a rapid-growing reproductive form that both consumes and sporulates in the developing kernels. As the wheat plant matures, the pathogenic hyphae and spores are found almost exclusively in the ovarian tissues and rarely in the axis of the head (rachis), glumes, nodes, and leaves.

The early development of the pathogen is a most crucial stage, for if the hyphae do not reach the apex before stem elongation starts, the pathogen will not be present in the kernels. If the hyphae could be slowed or diverted away from the apex, or if the pathogenic dikaryon could be induced to revert to the nonpathogenic monokaryons, the host would appear to be resistant.

Numerous reports have indicated that a persistent snow cover for 30–60 days favors dwarf bunt infection. Many infected sites, however, have adjacent wheat fields with the same wheat variety, similar soil, similar snow cover, and contaminated with *T. controversa* spores, yet free from dwarf bunt disease. Others suggest that any type of organic insulating material (eg, vermiculite or straw) at the soil surface would provide a moist, cold (1–8 C) microenvironment favoring spore germination and allowing sporidial mating to form the pathogenic dikaryotic phase leading to infection. The pathogenic hyphae of *T. controversa*

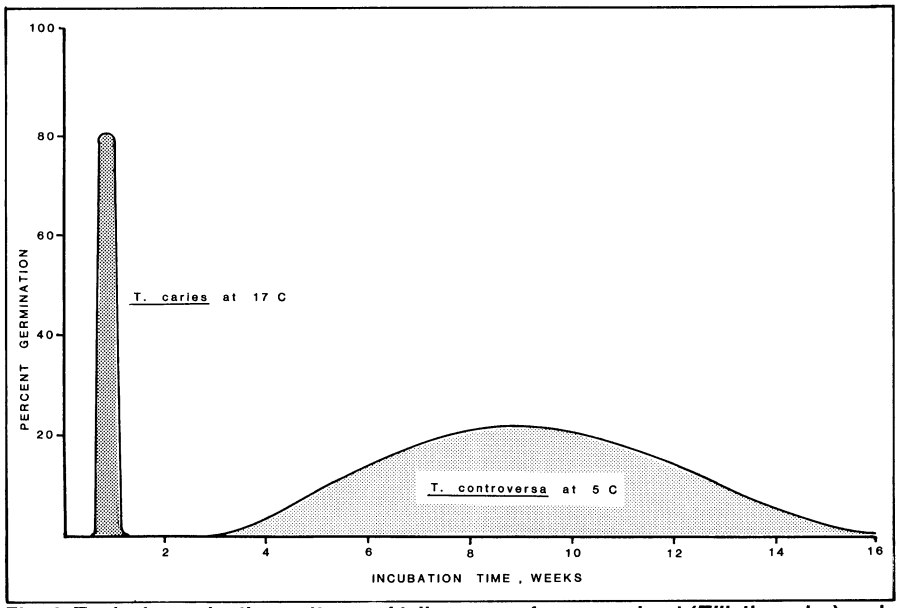


Fig. 4. Typical germination patterns of teliospores of common bunt (*Tilletia caries*) and dwarf bunt (*T. controversa*).

appear to develop in the susceptible host only at low temperatures (3–8 C) and are inhibited at higher temperatures (15–20 C). Unprotected soil surfaces at temperatures below freezing are not favorable sites for germination of the spores, fusion of the sporidia, or infection.

Free individual teliospores of *T. controversa* survive for only about 1 year in the soil of wheat fields that are conducive to the disease, but spores partially protected in bunt balls may remain viable in these fields for 3–10 years. The survival of dwarf bunt spores is unknown in the soil at sites where the disease does not develop. In about one-half of the wheat regions of China, wheat is rotated with rice. Experiments are in progress to determine the capacity of *T. controversa* spores to survive in the anaerobic conditions of a rice field.

Dwarf bunt disease occurs only on fall-planted winter wheat varieties, because a long cold period is required for the spores to germinate and infect young wheat plants. The natural infestations of dwarf bunt disease are limited to small areas in the large winter wheat regions of the northwestern United States (Fig. 1). The spores produced on infected wheat plants in these small areas have been carried throughout the winter wheat regions of the entire country for many decades by wind currents, transportation systems, irrigation systems, etc. Although the spores of *T. controversa* have been widely distributed and remain viable for years, the disease has remained localized in small, isolated areas of the Northwest. Likewise, in other regions of the world where dwarf bunt disease of wheat occurs there have been no restrictions on the movement of *T. controversa* spores, yet the diseased sites have remained small and localized.

It is important to note that the probability of infection occurring is low even if there are many bunt spores in the soil or on each wheat seed. For example, an average of 20,000 spores of the common bunt fungus *T. caries* per wheat seed is required for a high probability of an infected wheat plant. Knowledge of the germination characteristics of *T. controversa* spores leads us to predict that it would take many more than 20,000 spores per wheat seed to obtain infection. That very high inoculum level would rarely, if ever, be reached by the random distribution of dwarf bunt spores.

Another important historical consideration related to the potential spread of this disease is the fact that tremendous quantities of wheat have been shipped from the Pacific Northwest to many countries of the world during the last 40 years. It is likely that most of those shipments contained varying levels of *T. controversa* spores, but no outbreaks of dwarf bunt disease have been reported in any of the countries receiving the wheat.

It is also noteworthy that for decades the scientists who breed for resistance to dwarf bunt disease and the scientists who test new fungicides to control the disease have found it difficult to obtain uniform dwarf bunt infection in their field experiments. Even though they use a dense, viable inoculum on susceptible varieties at field sites known to be conducive to the disease under the most ideal conditions known for infection, good infection is obtained only about 1 year in every 4. Those results, along with the observations that the disease has not spread to similar adjacent wheat fields, suggest there may be undefined biological factors in the soil that either enhance or impede dwarf bunt disease of wheat.

Germination of Spores

During germination of the teliospores of *T. controversa* and *T. caries*, the ultrastructural changes are essentially identical (W. M. Hess, *personal communication*). *Tilletia* teliospores, on germination, produce nonseptate promycelia bearing terminal filiform sporidia that commonly mate in situ, forming H-shaped fused sporidia. These fused sporidia germinate to produce the pathogenic dikaryotic infection hyphae (Fig. 2). These postgermination developmental stages may be the weakest link in the whole life cycle and, thus, the most susceptible to control measures.

Teliospores of the common bunt fungus require 3–5 days for germination, whereas teliospores of the dwarf bunt fungus require up to 3 months under optimal conditions of moisture, oxygen, light, and temperature. Several potent endogenous germination inhibitors have been detected and partially purified from *T. controversa* spores (15). Teliospores of the dwarf bunt fungus do not germinate at temperatures above 12 C. Weak, continuous white light usually enhances germination somewhat if it is applied during the last half of the germination period. It has not been possible to enhance germination of these spores by adding either exogenous nutrients or the germination stimulants that are effective on some dormant bacterial and fungal spores. While many unique treatments and some chemicals have been reported to improve the germination of some collections of *T. controversa* spores, no general germination triggers, chemical or physical, have been found that will stimulate the teliospores to germinate in less than 3 weeks (13).

Teliospores of *T. caries* often show 70–80% germination after 3–5 days of incubation at 17 C. As a population, the germination characteristics of *T. controversa* spores are more variable than those of the common bunt fungi. Although dwarf bunt teliospores may begin to germinate in 3–4 weeks under optimal conditions, this early germination is less than 10%. Typically, the percentage germination increases slowly during the second month and then decreases slowly during the following month. In a population of *T. controversa* spores, germination occurs over a long period of time and rarely reaches a high level of percentage germination (Fig. 4).

These characteristics of *T. controversa* spore populations to germinate very slowly over a long period of time in a cold environment, a few degrees above freezing, are primary features of dwarf bunt disease. In the winter wheat regions, these requirements obviously limit the disease to those geographic sites where teliospore germination and sporidial mating can occur.

Identification of Spores

There are many similarities in the microscopic appearances of spores of fungi responsible for dwarf, common,

and some grass bunts (16). The morphological overlap among these species is so great that when only a few spores are available for observation they cannot always be positively identified by light or scanning electron microscopy. New sensitive, precise, and rapid tests are needed for differentiating spores of *T. controversa* from those of fungi responsible for common and grass bunts when only 5–100 spores are available.

Duran and Fischer (3) used comparative morphology of the teliospores and host symptomatology as the principal basis for species delimitation. They described the teliospores of *Tilletia* species as follows: *T. controversa*, spherical, usually 19–24 μm in diameter, reticulations 1.5–3 μm deep, covered by a hyaline sheath 1.5–5.5 μm thick; *T. caries*, spherical, less frequently aspherical, usually 14–23.5 μm in diameter, reticulations 0.5–1.5 μm deep.

Several new tests were recently described that distinguish the teliospores of the bunt fungi (16). In one test, the dry spores of *T. controversa* were shown to be spherical and normal in appearance in anhydrous propanol, whereas dry spores of the common bunt and grass bunt fungi were aspherical and deformed. In another test, a negative staining reaction, in which the spores were stained with methylene blue and then mixed with India ink, clearly showed the capsule on the spores and aided measurements and identification. New tests are being developed to identify *T. controversa* spores based on the following concepts: specific fluorescent antibodies; mineral element composition by microanalysis, x-ray, and neutron activation analysis; and pyrolysis—mass spectrometric analysis of whole spores.

The characteristics of spore germination are often used to distinguish *Tilletia* spp. If several hundred or more teliospores are available for testing, and if the results are not needed immediately, it is advisable to check the germination characteristics of the spores. If the spores germinate in 1 week on 3% water agar at 17 C, they are not *T. controversa* spores (16).

A standard procedure for determining whether grain is contaminated with spores of *T. controversa* is to shake a 50-g sample of the seed in 100 ml of water containing one drop of Tween 20. The water is poured through a coarse filter and centrifuged at 1,000 *g*. The pellet at the bottom of the centrifuge tube is examined for *Tilletia* spores by one or more of the above tests. This method is sensitive enough to detect one infected wheat head (containing about 200 million spores) in an acre of wheat.

Control of Dwarf Bunt Disease

In the preemergence stage, wheat seedlings are susceptible to infection by the common bunt pathogens. To be effective in blocking the germinating bunt fungi, a seed-treatment fungicide has to be volatile and persistent well beyond the immediate vicinity of the seed. None of the standard seed-treatment chemicals used before 1951, ie, formaldehyde, copper carbonate, and the mercurials, provided that protection. In 1951, hexachlorobenzene (HCB), which had been developed in Europe, was tested in the Pacific Northwest and found to be highly effective in controlling seedborne and soilborne common bunt of wheat. The use of resistant varieties of wheat combined with HCB as a seed-treatment fungicide has provided excellent control of common bunt in the Pacific Northwest for the past 26 years.

Unfortunately, neither HCB nor resistant varieties, or a combination of the two, has been effective in controlling dwarf bunt disease of wheat. HCB is ineffective primarily because infection of winter wheat by *T. controversa* occurs 3–6 months after HCB has been applied, and by that time the concentration of HCB in the seedling zone has dropped below the effective threshold. Several experimental fungicides effectively control dwarf bunt disease. The systemic fungicide thiabendazole is the only one approved for use against dwarf bunt in the United States but is expensive and not widely used in the Northwest.

Although wheat varieties resistant to dwarf bunt disease are available (eg, Luke, Ranger, Franklin, Jeff, Cardon, and Hansel), only about 5% of the winter wheat planted in the Northwest is resistant to *T. controversa*. Good sources of resistance against dwarf bunt disease are available in wheat breeding lines, but a farmer who plants a resistant variety not specifically adapted to his area may get about 25% less wheat yield than from a better adapted susceptible variety. Thus, farmers often plant dwarf-bunt-susceptible varieties and take a chance that *T. controversa* will not attack their wheat crop.

The dwarf bunt fungus is capable of forming new races when it is blocked by resistant varieties. Approximately 30 pathotypes (races) of the common bunt fungi are known, and 17 pathotypes of the dwarf bunt fungus have been characterized. It is interesting to note that the same wheat genes that confer resistance against the common bunt fungi also confer resistance against the dwarf bunt fungus (9).

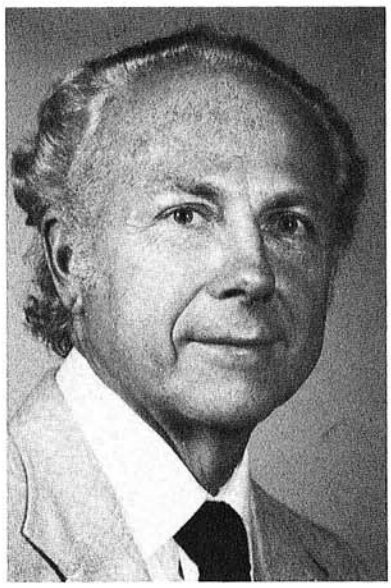
Bunt Spores in Wheat Shipments

The concept of "zero tolerance" implied that spores of *T. controversa* were not present in China and that a few spores of the dwarf bunt fungus getting into the country could start an epidemic of dwarf bunt that would destroy a significant portion of China's wheat crop. Several points are important in considering this concept. The policy was imposed by China in 1974, but before and since that date, many spores of *T. controversa* entered China by various routes. Between 1935 and 1950, millions of bushels of wheat were shipped from Pacific Northwest ports to China. Some of those shipments surely contained dwarf bunt spores, for the disease was more prevalent in those years than it is now. Also, an oceangoing vessel routinely carries many different types of cargoes in its international trade journeys. The ship may at some time transport a load of wheat contaminated with dwarf bunt spores. Some of the fine, dustlike spores remain in the ship after the wheat has been unloaded and settle on subsequent cargoes. Therefore, many spores of *T. controversa* surely enter China every day on miscellaneous cargoes carried on hundreds of ships used randomly to transport many items of international trade. The dwarf bunt spores entering China do not originate only from the wheat of the northwestern United States; they may come from any or all of the countries where dwarf bunt disease is found.

Although many spores of *T. controversa* have undoubtedly entered China during the last 40 years, there is no evidence that dwarf bunt disease ever became established and caused a noticeable problem. This is probably because few, if any, areas in China possess the unique soil, moisture, and temperature requirements needed for dwarf bunt disease to develop. As noted earlier, the probability of infection occurring is very low even when each wheat seed has many spores.

The spores of *T. controversa* have had many opportunities during the past 40 years to be distributed to all of the wheat-growing areas of the world. The fact that dwarf bunt disease is found in only a few limited areas in a few countries illustrates the extremely specific conditions that must be met before the disease can become established.

The biological, pathological, and geographic evidence all suggest that wheat shipments from the northwestern United States, which may contain a low level of *T. controversa* spores, pose no serious threat to wheat production in China. If *T. controversa* did infect a wheat field in China, the infected area would remain small and could probably be eliminated by using fungicides and wheat varieties resistant to the dwarf bunt fungus. *T. controversa* has never caused a widespread, epidemic-type disease. When the dwarf bunt fungus does attack wheat plants, it has always been in small, localized fields. There is no scientific basis to support the "zero tolerance" concept of dwarf bunt spores in the wheat export trade.



Edward J. Trione

Dr. Trione is a research biochemist with the USDA's Agricultural Research Service and a professor of botany and plant pathology at Oregon State University, Corvallis. He received his Ph.D. at Oregon State University in 1957. Most of his research has focused on the bunt diseases of wheat.

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