J. B. Matanguihan and K. M. Murphy
Washington State University, Pullman

S. S. Jones
Washington State University, Mount Vernon

Control of Common Bunt in Organic Wheat

During the last two decades, common bunt has re-emerged in low-input and organic wheat, most notably in northern and western Europe. Agriculture in Europe has been moving toward organic and sustainable, low-input farming systems with reduced chemical inputs in crop production (74). Fundamental changes in agricultural production systems, such as the lack of chemical seed treatments, have caused the resurgence of many seedborne diseases, including common bunt, that were previously controlled with chemicals. In the United Kingdom, organic seed lots are predominantly contaminated with common bunt spores (94). In the Czech Republic, a 4-year monitoring of bunt incidence from grain samples showed an increase of bunt spores from various locations. This increased bunt incidence was observed in low-input and organic farms, and can also be related to changes that forced farmers to grow winter wheat at a higher percentage in crop rotations (124). Contamination of wheat with common bunt spores has resulted in considerable loss of yield and seed quality. In Romania, if untreated seeds are used, the incidence of common bunt can reach 70 to 80%, with yield losses up to 40% (21). Typically, yield losses almost equal disease incidence because wheat kernels have been replaced with bunt spores. Even cleaning the seed and sowing at higher soil temperatures cannot totally prevent the occurrence of common bunt (126). Given the epidemiology of the disease, it has the potential to cause economic devastation to low-input and organic farmers.

The legal requirement for organic seed has compounded the bunt problem in Europe. For many years, it was possible to use conventionally produced seed as long as the cultivars were not of transgenic origin and the seed had not been treated after harvest with synthetic fungicides. All of this changed with Commission Regulation (EC) No. 1452/2003, which stipulated that beginning January 2004, all plant materials used for organic agriculture must be produced under organic farming conditions. With this regulation, a high level of expertise in disease management became requisite for organic seed production. It is now crucial that seed and planting materials be pathogen-free and of superior quality, since most forms of synthetic chemical protection are not allowed. However, organic seed lots frequently do not make the grade and are often discarded because of contamination with common bunt (76). If this trend continues, there could be shortages of organic and certified seed. The limited supply of organic and certified seed might cause farmers to use seed saved from previous seasons. If farm-saved seed is contaminated with common bunt, the disease will build up further (124), especially if farmers do not monitor each successive crop for this disease (95).

In conventional agriculture, common bunt is often exclusively controlled with chemical seed treatments. Given that these seed treatments are prohibited under organic certification standards, alternative treatments are being explored to manage common bunt under organic conditions. Tillecur is one of a few organic seed treatments that are effective, but these seed treatments also vary in efficacy, increase production costs (73), and often cannot be applied on a large scale. Under organic systems, the use of host resistance is a major component for sustainable disease management. However, there are limited numbers of wheat cultivars highly resistant to bunt that are adapted to organic systems. Most of the resistant cultivars have been bred under conventional agricultural systems, and might not be the best cultivars to use in organic farming. Studies have shown that these cultivars could lack important traits required under organic and low-input cropping systems (75,100,129). It is imperative, then, that selection for bunt resistance is conducted under organic farming conditions.

Now, more than half a century after common bunt was thought to be vanquished, it has re-emerged in organic wheat. Today's farmers and scientists, like those in the past, are faced with the challenge of managing common bunt, but this time without chemical seed treatments. In this review, we present two main approaches that have been taken in managing the disease under organic systems: host resistance and seed treatments. Much of the research described here was conducted in Europe, Canada, West Asia, and North Africa. Even though common bunt has not yet re-emerged in organic wheat in the United States, we believe that it is inevitable if conventionally produced seed will no longer be allowed on organic farms. We conclude this article by making recommendations for the control of common bunt consistent with the principles of organic agriculture.

Corresponding author: S. S. Jones, Department of Crop and Soil Sciences, Washington State University Mount Vernon Northwestern Washington Research and Extension Center, Mount Vernon, WA 98273; E-mail: joness@wsu.edu

doi:10.1094/PDIS-09-10-0620

Fig. 1. Wheat head infected with common bunt, showing kernels replaced by sori or bunt balls. Photo by Margaret Gollnick.

The Pathogen and Disease

Common bunt is caused by two closely related fungi, *Tilletia caries* (D.C.) Tul. & C. Tul. (syn. *Tilletia tritici* (Bjerk.) G. Winter) and *T. laevis* J.G. Kühn (syn. *T. foetida* (Wallr.) Liro). The teliospores of *T. caries* have reticulated walls, whereas those of *T. laevis* have smooth walls. Although morphologically different, the two species are similar in germination requirements, life cycles, and disease symptoms produced. *T. caries* and *T. laevis*, together with *T. controversa*, the causal agent of dwarf bunt, could be variants of the same species, as proposed by several genetic, biochemical, and molecular studies (18).

Common bunt is one of the most destructive diseases of wheat worldwide, causing considerable yield loss and reduction in seed quality. Common bunt is also called stinking smut, due to the production of trimethylamine, which gives the disease a distinct fishy odor even at contamination levels as low as 0.1% by volume (77). There is optimum infection when soil temperatures range from 5 to 10°C, but infection is reduced when soil temperatures are at 22°C (108). Teliospores on the seed or in the soil germinate and produce hyphae that infect the wheat coleoptiles before emergence. The fungus grows systemically in the plant and proliferates in the spikes when ovaries begin to form. The pathogen sporulates in the endosperm tissue until the entire kernel is converted into a bunt ball (sorus) consisting of a dark mass of teliospores. The bunt balls often break during harvest and grain handling, releasing teliospores that contaminate the seed and soil, thus initiating another cycle of infection.

History of Bunt Control

In 1750, the Royal Academy of Literature, Science and Arts of Bordeaux announced that a prize would be given for the best investigation into the smutting of wheat. Mathieu Tillet, Keeper of the Mint at Troyes, entered the contest. In his seminal experiments, Tillet planted wheat seed that he had dusted with the black spores and other seed that he had not. From the seed coated with black dust, he observed 50% or more smutted heads, while in the rows of clean seed, little or no smut developed (33). Tillet had found the answer. His experiments proved that the smut spores were infective: *"The outcome of the different experiments I have presented seems sufficient to persuade me that the disease [bunt] was contagious and that the virus was resident in the dust of the bunt balls"* (122). Tillet not only found the cause of the disease, but also some way to prevent it. He washed the seed grain in water, cattle urine, lye solutions, lime and salt, and finally, copper sulfate. Although none of these eliminated smut entirely, each helped to suppress it. For his groundbreaking experiments, with their remarkable scientific underpinnings, he won the prize. Unknowingly, he also laid the foundation of a new science, plant pathology, and had his name forever linked to smut of wheat: *Tilletia*.

W. J. Farrer is acknowledged to be the first to apply systematic breeding methods to develop wheat cultivars resistant to bunt. He released 'Florence', which E. F. Gaines crossed with 'Turkey' to produce 'Ridit', the first bunt-resistant cultivar in the Pacific Northwest (PNW) region of the United States (33). In the early 1900s to the 1960s, common bunt was the most destructive disease of wheat in the PNW, and its management was intensively studied. Pathogen genetics, pathogenic races, survival of spores in soil and spore germination, and the effect of seeding dates, tillage methods, and seed treatments on disease management, were studied (16). In Pullman, WA, work on wheat resistance to bunt began in 1914 (41). There was a concentrated search for resistant cultivars to form the core of a bunt breeding program (123), since the specificity of bunt resistance in wheat was long recognized before the discovery of specialization in the pathogen. In addition to screening wheat germplasm for bunt resistance, Gaines studied the genetics of bunt resistance (39,40). He also established the existence of physiologic races of the pathogen (42,43), as did Flor (34). Their work started decades of effort to gain the upper hand in bunt control. Plant

breeders and plant pathologists spent years developing bunt-resistant varieties. However, each resistant variety released in the PNW would be subsequently attacked by new, virulent races of bunt (117). In turn, the resistant genes in the wheat cultivars influenced the racial population dynamics of the bunt pathogens (54). From these observations, it became clear that there is a gene-for-gene interaction between wheat and the common bunt pathogens. Dominant bunt resistance genes (*Bt*) in wheat hosts have corresponding dominant avirulence genes (*avr* genes) in the fungus. The genetic specificity of the wheat–bunt pathogen interaction made it difficult to control the disease by host resistance alone, especially since all of the resistance at that time was race-specific.

Since Tillet's time, numerous seed treatment methods have been used to control common bunt, such as salt brine, lime, mixtures of lime, salt, saltpeter, wood ashes, copper sulfate, formaldehyde, copper carbonate, and liquid mercury, all of which were either ineffective or too toxic to the seed, or to humans (91). In addition to these seed dressings, other physical seed treatments were tried, such as hot water treatment, originated by Jensen in 1888, and later, heat treatments in the form of steam (33).

As early as 1807, Prevost had demonstrated that bunt could be controlled to some degree by copper sulfate. But it was not until the latter half of the nineteenth century that chemical disease control really started to gain ground (113). The development of the polychlorobenzenes, notable for their high specificity for certain fungi, specifically hexachlorobenzene (HCB), proved to be a powerful weapon in the control of common bunt. HCB was so effective against both seedborne and soilborne spores of bunt (60,107) that efforts toward its integrated control slowed dramatically after the introduction of HCB (16). In a matter of years, the new chemicals were widely adopted. Throughout the PNW and in much of the world, common bunt was finally controlled. The dreaded black harvest was no more. This classic, textbook disease was rarely seen in farmers' fields, observed only when untreated or improperly treated seed was used (54,95). Common bunt had become a forgotten disease—until its re-emergence in organic wheat.

Host Resistance

Breeding programs for common bunt resistance no longer exist in most wheat-growing countries. Under the assumption that the disease could be simply controlled by a single chemical seed treatment, breeding for bunt resistance has been given low priority in the United States, Europe, North Africa, and West Asia. Organic and low-input farmers must largely depend on crop cultivars produced for conventional farming (101), for which there is little information on bunt resistance. There is also limited knowledge on pathogenic variability. Therefore, current research on bunt resistance in organic wheat echoes research performed early in the twentieth century: monitoring bunt incidence and pathogen races, screening cultivars for bunt resistance, conducting studies on the mode of inheritance of bunt resistance, and searching for new sources of resistance. Employing molecular techniques, genes involved in resistant host response have been identified (84,85), and resistance genes have been mapped (35,96,127). Quantitative bunt resistance has also been investigated (35).

New races and virulence patterns of common bunt isolates. Due to the gene-for-gene interaction that exists between specific bunt avirulence genes and bunt resistance genes in wheat, it is necessary to identify and monitor races of the pathogens. These races can be identified by inoculating them on differential cultivars, monogenic for bunt resistance genes. Their ability to infect specific cultivars within the set of differential cultivars will give a virulence pattern. This virulence pattern is analyzed and compared to the unique virulence patterns of the known races, as reported by Hoffman and Metzger (55). If the virulence patterns are unlike those of the known races, new races could then be postulated. The presence of new races in a certain area or the prevalence of known races in an area would help plant breeders determine what bunt resistance genes to deploy. It would also inform them what resistance genes to use or pyramid when developing new, resistant cultivars.

Mamluk (86) reviewed reports on the prevalent common bunt races in Turkey, Egypt, Syria, Tunisia, Lebanon, Iran, and Morocco. In Turkey, 37 races were reported in 1981, and 88 in 1983. Five of the prevalent races in Turkey and two in Syria correspond to the North American races. Three isolates from Syria had new combinations of virulence patterns, and were reported to be new races (62). There are more recent reports of new bunt races from Iran, with four new races of *T. laevis* from the Khorasan Province (2) and nine new races from the Kermanshah Province (22). A systematic survey of common bunt incidence in the different agroecological zones in Iraq was conducted during the 2002–2003 season. High disease incidence was observed in the central and southern regions of Iraq for the first time, although common bunt was thought to be restricted to the northern region. Movement of the disease to the central and southern regions could be ascribed to the use of contaminated wheat seed. Results of the survey also showed that *T. caries* was more widespread in the north, and *T. laevis* in the south (1).

T. laevis is more prevalent in Romania, especially in the south, while *T. caries* is more common in the northwest, coming with the seed from Europe (104). Due to increased wheat monoculture, inappropriate use of chemical seed treatments, and the continual and rapid evolution in pathogen races, epidemics have become more common in small farms in Romania (104). In Ukraine, the dominant pathogen is *T. caries*, with the population consisting of 12 races. Seven of these races have virulence patterns similar to those of the known North American races T-1, T-2, T-3, T-7, T-9, T-17, and T-20. Most of the wheat cultivars grown in the country are susceptible to these races (3).

Other European workers have reported the virulence of the local bunt populations to the bunt resistance genes present in their germplasm collection, and against the differential cultivars (Fig. 1, Table 1). Germplasm resistance screenings were conducted for several years, and showed that most of the European bunt populations were virulent against the *Bt* resistance genes *1, 2, 3,* and *7*, while these could not attack the *Bt* genes *5, 8, 9, 10,* and *11* (Table 1). In the United States, only five races are virulent on the *Bt* genes *5, 9,* and *10*, and none on *Bt8, 11,* and *12* (48).

Screening of wheat germplasm for bunt resistance. Due to limited information on the resistance of registered wheat cultivars to bunt, several resistance screening studies have been performed in the last two decades. For these studies, wheat seed are inoculated by dusting with teliospores before sowing. Inoculated seed

Table 1. Virulence of local populations of common bunt against resistance genes (*Bt*) from differential cultivars and wheat germplasm

Source of common bunt population	Years of screening	*Bt* genes effective against the local bunt population	Reference
Hungary	1991-1997	*Bt5, Bt6, Bt8, Bt9, Bt10*	125
Europe	2000-2002	*Bt3, Bt5, Bt6, Bt8, Bt9, Bt11, Bt12, Bt13*	5
Austria and Germany	2005-2006	*Bt4, Bt5, Bt6, Bt8, Bt9, Bt10, Bt11, Bt12, Bt14*	61
Poland	1998-2000, 2004-2005	*Bt4, Bt8, Bt11*	72
Romania	2005-2006	*Bt5, Bt8, Bt9, Bt10, Bt11, Bt12, Bt13*	104
Latvia	2008-2009	*Bt4, Bt5, Bt6, Bt8, Bt9, Bt11, Bt12*	106

are sown when the soil temperatures are 5 to 10°C, and bunt incidence is observed at plant maturity as the percentage of infected heads. In Canada, Gaudet and Puchalski (46) tested the field reaction of triticale, hard red spring, durum, and soft white spring wheat to common bunt, and found that triticale (wheat × rye hybrid) was the most resistant among the cereals tested. Of the classes of wheat, durum (*Triticum durum*) was the most resistant, followed by hard red, soft white, and Canadian prairie spring wheats. Out of 22 CIMMYT lines and six Canadian wheat cultivars tested for bunt resistance, only four CIMMYT lines and one Canadian cultivar had low infection levels (47). In the PNW of the United States, wheat breeding lines have been screened for bunt resistance for over 25 years (6). However, most of these lines have not been specifically bred for organic farming and had been screened under non-organic conditions. The disease reaction of the cultivars should not change whether the screening was conducted under organic conditions or not. However, in Europe, most of the bunt resistance screening was conducted under organic conditions since the lines were being developed for organic farming.

Most of the widely grown, local cultivars in Europe are susceptible to common bunt. In Serbia and Montenegro, only four out of the 12 most widely grown cultivars were resistant to common bunt (109). In the Czech Republic, Dumalasová and Bartoš (25–27) screened winter wheat and spring wheat cultivars for bunt resistance and found that it varied among cultivars, and also across years and locations. Spring wheats generally had less disease, probably due to the warmer soil conditions when the seeds are sown. Recently, they screened 17 newly registered wheat cultivars, and none of these were resistant to common bunt (29). In Lithuania, Liatukas and Ruzgas (78) determined that out of the 26 winter wheat cultivars registered in their country, none was highly resistant, and only two were moderately resistant. In order to initiate a breeding program for organic wheat, they increased the number of cultivars screened for bunt resistance, screening more than 2,000 cultivars over a period of 12 years (1993 to 2004), with some of the cultivars continuously screened for up to 8 years (79). More than 1,000 germplasm lines were screened during 2006 to 2008 against local populations of the pathogen. Their tests showed that only 1% of the genotypes tested were resistant to common bunt (81). In a separate screening of 347 breeding lines, only two lines were resistant (114). Moreover, the resistant lines were agronomically poor and could only serve as donors of resistance genes, indicating the need for a more intensive search for resistant germplasm. Their tests also showed that the local pathogen population in Lithuania possessed virulence to the majority of the genes studied (114).

For most of these resistance screenings, workers have observed variation in bunt incidence among replicates and over years of screening. This variation is attributed to differences in weather conditions over the years. If the inoculum is obtained from field isolates, variation in bunt incidence could also be due to the presence of different virulence genes in the pathogen populations (26,124). Thus, to achieve a more precise assessment of resistance to common bunt, there should be at least 3 years' testing under high disease pressure, with a minimum of three replicates and the inclusion of cultivars with known resistance levels (26). The inoculum dose could also be standardized, as the amount of inoculum has an effect on disease incidence of susceptible cultivars, although not on resistant cultivars (28). The source of inoculum should also be uniform over the years of screening, and a variety of tests should be organized at several locations. The bunt races occurring in different regions need to be monitored, since the emergence of new races would change the interpretation of resistance tests.

Inheritance of bunt resistance. The genetic control of bunt resistance has been studied in order to deploy bunt resistance genes into new cultivars. Knowledge of the mode of inheritance can also assist in the identification of resistance genes and the genetic markers which can be used in marker-assisted selection. Past genetic studies have shown that the inheritance of common bunt resistance in various crosses can be controlled by single genes. Of the 15 known bunt resistance genes, 14 are dominant (98,99,128). Although the majority of bunt resistance genes are completely dominant, there have been reports of incomplete dominance, as reviewed by Holton and Heald (59).

Knox et al. (66) determined that bunt resistance in lines SC8O21V2 and L8474D1was due to a single major gene with incomplete dominance. They used doubled haploid and random inbred populations of wheat, both androgenetically derived, to remove segregation as a source of variation. In another study, the authors He and Hughes (52) investigated the genetic control of common bunt resistance by testing different populations from crosses of the resistant wheat cultivar Triple Dirk and the spelt wheat cultivars RL5407 and SK0263, with susceptible cultivars Laura and Genesis. They found that Triple Dirk appeared to carry three major genes for common bunt resistance that conferred resistance to each of the three races T-1, T-13, and L-7. The spelt wheat RL5407 possibly carried a single major gene or closely linked genes for resistance to both races T-13 and L-7, and another major gene for resistance to T-1. In addition, bunt incidence in the F_1 and F_2 generations also suggested that the resistance to race T-1 was due to additive gene action in these two crosses. They concluded that selection of resistant lines could be developed by incorporating the resistance from Triple Dirk and RL5407.

In Romania, Coţa et al. (21) screened F_2 populations of wheat derived from seven crosses of resistant and susceptible cultivars to establish the mode of inheritance to common bunt. The analysis of variance showed significant differences among the F_2 wheat progenies in their reaction to field samples of *T. caries* and *T. laevis*. Genetic analysis showed that resistance to *T. caries* and *T. laevis* in seven F_2 segregating populations is inherited as a single dominant gene, although the gene conditioning resistance has not been identified.

Identification of bunt resistance genes. There are 15 known bunt resistance genes (48), but for many cultivars, it is not known which genes they possess. Gaudet and Puchalski (46) attempted to identify the bunt resistance genes in 23 spring wheat cultivars. They inoculated single races of *T. caries* and *T. laevis* on the cultivars and compared the virulence patterns of the known races to those of the differential cultivars, as published by Hoffman and Metzger (55). The high specificity of avirulence genes to resistance genes made it possible to do gene postulation. The reaction of the cultivars to single races indicated that only a few cultivars contained specific genes for resistance. They postulated the presence of *Bt1* in the cultivar Canuck and *Bt10* in the line BW-553. Resistance in the other cultivars of hard red spring wheat appeared to be race-non-specific, and the resistance genes they possess could not be identified by the method used.

Molecular markers associated with major bunt resistance genes. Molecular markers associated with bunt resistance genes could aid the development of resistant cultivars by facilitating screening for resistance and the introgression of bunt resistance genes in wheat cultivars with good agronomic genotypes (20). Since bunt disease symptoms become obvious only at plant maturity, screening cultivars for resistance is time-consuming (4). Variable disease infection levels due to environmental effects, in addition to disease escapes, also complicate the evaluation of resistance in wheat. Researchers often find it difficult to classify the lines precisely as resistant or susceptible, especially in field results when disease incidence is low (127). Molecular markers associated with resistance genes could hasten the process of resistance screening by circumventing the complications of field screening.

The first molecular markers associated with bunt resistance were RAPD markers for the *Bt10* gene in wheat (23) and spelt (53). *Bt10* is a major gene from 'PI 178383' and is resistant to 35 of the 40 known bunt races. Later, Laroche et al. (77) developed a highly specific polymerase chain reaction (PCR) marker, FSD_RSA, associated with *Bt10.* Using wheat microsatellite (SSR) markers, Menzies et al. mapped *Bt10* on wheat chromosome 6D (96). This is the sixth gene, out of the known 15 *Bt* genes, to be mapped to its chromosomal location. *Bt1* is located on 2B (92,116), *Bt7* on 2D (92), *Bt4* and *Bt6* on 1B (115), and *Bt5* on 1B (92). The location of

Bt8 is unknown, but by genetic analysis, it is known not to be on 5A, 1B, or 2D (128). Wang et al. (127) mapped the resistance gene of 'Blizzard' to 1BS. Blizzard is a winter wheat cultivar resistant against known races of common bunt in western Canada. The resistance gene in Blizzard has not been identified, but since it has been mapped to 1BS, it is different from *Bt1*, *Bt7*, *Bt8*, and *Bt10*. Further study is needed to determine if it is different from the other named genes with unknown chromosomal location (127). Another bunt resistance gene has been mapped to chromosome 1B, but it is derived from *Aegilops cylindrica* (44).

Ciucă and Săulescu (20) used the *Bt10* markers developed by Demeke et al. (23) and Laroche et al. (77) to screen Romanian cultivars for the presence of the *Bt10* gene. All lines had been previously screened to be bunt resistant. Only one line, obtained from the cross between 'PI 554118' (source of *Bt10*) and 'Dropia' (susceptible line) showed the 275-bp fragment associated to *Bt10*. The 13 lines derived from crosses with PI 178383 as one of the parents did not show the fragment, even though PI 178383 is a carrier of *Bt8, 9,* and *10*. It is presumed that when it was crossed to a susceptible parent, either *Bt8* or *Bt9* was retained, or both were retained, while *Bt10* was lost due to selection for adaptation and plant type. As expected, none of the lines known to carry other bunt resistance genes showed the 275-bp amplification product. This is a confirmation that this amplification product is specific only to *Bt10*.

Markers could also be powerful tools in pyramiding bunt resistance genes to achieve durable bunt resistance. Although scientists are making progress, much work remains to be done toward marker assisted selection for bunt resistance. At present, only the *Bt10* gene has markers associated with it, and of the 15 known bunt resistance genes, only six have been mapped to their chromosomal location. Moreover, it is still not known which resistance genes are present in several resistant cultivars in the United States, Canada, and Europe. The genes of many landraces from Turkey and Iran known to be resistant to common bunt (6) have not been studied.

Quantitative bunt resistance genes. Gaines first mentioned the concept of quantitative resistance to common bunt when he observed that there is a wide range of disease incidence in the F_3 population under field conditions (39). He concluded that bunt resistance in wheat is not a simple Mendelian trait, and that if it is Mendelian, it is composed of multiple factors, "for a continuous series ranging from complete immunity to complete susceptibility has been observed". From 1926 to 1945, six major genes—(*Martin (M_1)* or *Bt1, Hussar (H)* or *Bt2, Ridit (rd)* or *Bt3, Turkey (T)* or *Bt4, Rio (R)* or *Bt6,* and *Martin (M_2)* or *Bt7)*—and five minor genes—*(U, V, W, X, Y)*—for bunt resistance had been identified (33).

Gaudet and Puchalski (46) also mentioned the phenomenon of field resistance. They observed that many hard red spring wheats had adequate resistance in the field but were susceptible to all bunt races in controlled environment studies. The failure of field resistance to be expressed under controlled environment conditions suggested to them that most of the hard red spring wheat cultivars screened possessed none of the race-specific resistance genes. Instead, field resistance appears to be race-non-specific and is weakened or lost under controlled conditions, but the resistance mechanism is unknown. The authors hypothesized that the race-non-specific resistance in these cultivars probably involves the phenomenon of disease escape, where genetically susceptible plants do not become infected due to the absence of factors necessary for disease development, or these factors do not coincide long enough for disease to develop.

There is strong evidence that general resistance to bunt exists. In dwarf bunt, there are reports of increased resistance due to additive interactions from genes that are not effective singly, and of varying levels of bunt incidence expressed by specific resistance genes in different genetic backgrounds (54). Rusgaz and Liatukas (114) had the same observations in common bunt. They noted that different cultivars that have the same resistance genes had significantly different disease incidences, even though these possessed the same genes or the same combination of genes. They hypothesized that modifying genes accounted for the difference in virulence patterns and could affect the introgression of resistance genes into different wheat backgrounds.

Fofana et al. (35) were the first to map quantitative trait loci (QTL) associated with common bunt resistance in the wheat doubled haploid population derived from the cross RL4452 × AC Domain. Their results showed three genomic regions located on two chromosomes (1B and 7A) that explained 32% of the phenotypic variation contributed by the AC Domain alleles. This type of resistance appears to be race-non-specific, and could be valuable in providing durable resistance against shifts in virulence in the pathogen population.

Molecular and physiological basis of bunt resistance. Lu et al. (84,85) conducted initial investigations into the mechanisms of host resistance to bunt fungi. Lu et al. (84) characterized defense-related genes that were preferentially up-regulated during a resistant or incompatible interaction involving the bunt resistant gene *Bt10* and race T-1 of *T. caries*. A total of 168 differentially up-regulated and 25 down-regulated genes were identified and sequenced. The majority of these sequences (71%) had significant homology to genes of known function, namely genes responsible for cellular metabolism and development (69%), abiotic/biotic stress response (28%), and transcription and signal transduction processes (3%). Two putative resistance genes and a transcription factor were identified among the up-regulated sequences.

The expression of several candidate genes, including a lipase, two nonspecific lipid transfer proteins (ns-LTPS), and several wheat pathogenesis-related proteins (PR-proteins) was evaluated in susceptible (compatible) and resistant (incompatible) interactions. Results confirmed the higher overall expression of these genes in the resistant line compared to the susceptible line. In a follow-up study, Lu et al. (85) demonstrated that the stage of seedling development can also affect the expression of some PR-proteins induced by jasmonic acid (JA) and salicylic acid (SA). For some PR-proteins, responsiveness to induction by these signaling compounds decreased with plant age. In addition, tillers initiated later were most likely to become infected.

Using fluorescence and confocal microscopy, Gaudet et al. (45) compared host responses in compatible and incompatible interactions between *T. caries* races T-1 and T-27, and the *Bt10* gene. They reported that initial host perception of pathogen invasion was similar in both susceptible and resistant cultivars and occurred as early as 5 to 6 days after seeding. However, after 9 days, the bunt fungus had grown past the coleoptiles and through the first embryonic leaf of a susceptible cultivar, while it remained restricted to the coleoptiles of the resistant cultivar. There was also a rapid accumulation of callose around the invading fungal hyphae, confirmed further when the expression of callose synthase transcripts was examined and found to be more abundant in the resistant cultivar. Their study confirmed earlier observations involving artificial inoculation of wheat coleoptiles, where both susceptible and resistant cultivars are penetrated by hyphae and initially invaded. However, after initial invasion, pathogen ingress differs in susceptible and resistant cultivars. In resistant cultivars, the fungal hyphae colonize juvenile tissue but fail to reach floral primordia, while in susceptible cultivars, the fungus reaches the flora primordia before internodal elongation and sporulates as seed develops (54). The authors conclude that callose accumulation was only one of several host defense responses and was shown to be insufficient in terminating infection. Rather, the expression of a pathogenesis-related protein (PR) protein, *PR-1.1*, *chitinase 2*, and a lipase, together with other PR proteins, could be more important factors in stopping fungal growth and spread through a resistant host (45).

New sources of bunt resistance. The study of Martynov et al. (90) emphasized the severe narrowness of the genetic base for common bunt resistance in wheat cultivars. They analyzed resistance to common bunt by tracing the cultivar pedigrees of resistant and susceptible cultivars from two regions: North America (Canada and the United States) and the former Soviet Union (Russia and Ukraine). In North America, the contributions of cultivars possess-

ing the resistance genes *Bt1*, *Bt3*, *Bt4, Bt, Bt6*, and *Bt7* appear to be significantly higher in the resistant cultivars, since these have been used as sources of resistance for several decades. The contribution of PI 178383, a Turkish landrace collected by Jack R. Harlan in 1948, is considerably higher in the group of resistant cultivars bred after 1965. Most of the currently grown cultivars are susceptible to one or more of the common bunt races (90). Virulence against the resistance of *Bt9* and *Bt10* has already been detected, and the number and diversity of useful germplasm appear to be critically limited. In the United States, less than a dozen sources for bunt resistance are known, and the number of resistance genes represented in these cultivars may be considerably less than that (54).

Hoffman pointed out the urgency to identify new sources of bunt resistance to provide more genetic diversity for breeding programs (54). Nearly 30 years later that urgency has grown. Bonman et al. (6) suggested that we explore the geographical centers of bunt resistance to broaden the genetic base of resistance. Results of the systematic characterization of the USDA-ARS National Small Grains Collection (NSGC) in terms of disease and insect resistance show that there is a clear center of concentration for bunt resistance. This geographical center extends from Serbia and Montenegro through Macedonia, Turkey, and Iran. The highest frequency of resistance occurred in Kosovo Province in Serbia and Montenegro (36%) and Bakhtaran Province in Iran (40.8%). Collections of landraces from these regions might be sources of new genes or new gene combinations for bunt resistance.

Related wheat species and genera have long been recognized as valuable sources of bunt resistance genes. The spelt wheat cultivars RL5407 and SK0263 could be potential new sources of bunt resistance (52). There could be specific resistance genes in the tetraploid *T. durum* cultivars that are different from those originating from the hexaploid *T. aestivum* cultivars (46). Since 13 of the 15 differential cultivars are hexaploid wheats, virulence patterns of the standard bunt races on *T. durum* cultivars would not produce a known pattern. It is significant that individual bunt resistance genes that have been studied are located on the B and D genomes in *T. aestivum* cultivars. Because most durum wheat is highly resistant to bunt under field conditions, but is susceptible to the majority of races under controlled environment conditions, it is likely that durum wheat also possesses high levels of race-non-specific resistance. Mamluk and Nachit (87) detailed similar results when they screened 42 durum wheat genotypes for bunt resistance. They identified 26 resistant genotypes from the ICARDA breeding program, originating from Italy, France, Turkey, Syria, and Tunisia. Dumalasová and Bartoš (29) also studied the resistance of 17 triticale cultivars, one emmer, two spelt, and seven durum wheat cultivars. All triticale cultivars were resistant, while disease incidences in the emmer, spelt, and durum wheats were lower compared to the susceptible bread wheats.

Wild relatives and progenitors of wheat could also be sources of bunt resistance. Accessions of wild einkorn, *T. boeticum (T. monococcum* L. subsp. *aegilopoides*), and wild emmer, *T. dicoccoides (T. turgidum* L. subsp. *dicoccoides*), from ICARDA were screened for bunt resistance, and 51% of the *T. boeticum* and 49% of the *T. dicoccoides* accessions were resistant. Of the 328 accessions of 23 *Aegilops* species screened, only two species were infected, and at very low levels (86). Oncică and Săulescu (105) tested the resistance of 26 winter wheat lines derived from crosses with related species or genera, bred at the National Agricultural Research and Development Institute (NARDI), Romania. They identified several resistant lines, one of which had *T. monococcum* in its pedigree. In addition, there were three lines selected from the cross *Triticale*/2*wheat which had high levels of resistance in 3 years' testing. The pedigree of these lines does not suggest the presence of known bunt resistance genes, which means they could be new sources of resistance.

Nonwheat relatives were also tapped as sources of bunt resistance. *Hordeum chilense* is a diploid wild barley native to Chile and a small area in Argentina. It is resistant to common bunt and Karnal bunt, and to other smuts, *Ustilago nuda* and *U. tritici*, plus several fungal pathogens causing rusts and powdery mildew, suggesting that it is a nonhost to these pathogens (112). When *H. chilense* is crossed with diploid, tetraploid, and hexaploid wheats, intergeneric amphidiploids are obtained and named tritordeums. The hexaploid tritordeums, from the cross of *H. chilense* durum, has potential as a new crop (88). Accessions of *H. chilense* and hexaploid tritordeums were screened against common bunt and found to be very resistant. Almost complete resistance was conferred by a gene(s) present in chromosome 7, and a slight but significant level of resistance was conferred by chromosome 6 in the wheat–barley addition lines (110,111). On the other hand, several cultivars carrying translocations from rye (*Secale cereale*) did not possess effective bunt resistance (80).

"The power of alien sources of resistance is not only to expand existing genetic variation in wheat but more importantly, to introduce truly novel variation" (64). The challenge is to introgress bunt resistance genes from wheat and wheat relatives while eliminating poor agronomic traits (51). Most sources of bunt resistance have poor agronomic performance, and some susceptibility alleles are not completely expressed. Even after several cycles of crossing with high-yielding cultivars, the association of bunt resistance with some unfavorable traits remains difficult to break. Advancements in the field of genomics, including the use of DNA markers, DNA sequencing technologies, and marker-assisted selection, could be applied to pyramid resistance genes and maintain recessive alleles in backcrossing pedigrees (51). The fact remains that, whether improvements are achieved through classical breeding or with the aid of molecular techniques, considerable time and effort will be required before the release of new, bunt-resistant cultivars adapted to organic conditions. Meanwhile, existing resistant cultivars should be treated with alternative seed treatments for integrated management of common bunt.

Seed Treatments

Research on alternative seed treatments has been limited, mainly because for decades, there was no perceived need for nonchemical seed treatments. With the resurgence of common bunt in organic wheat, the urgent need for nonchemical seed treatments compelled research on seed treatments compatible with the philosophies of organic agriculture. There is renewed interest in using hot water, heat treatments, and plant-based substances to manage common bunt. More novel seed treatments have been explored, including biological control, the use of volatiles from microbes, and other innovative physical seed treatments.

Establishment of threshold levels. Before seed treatments are initiated, the seed lot has to be subjected to a phytosanitary analysis. The spore thresholds currently prescribed or recommended for common bunt in organic agriculture vary in different countries. The threshold range is 20 spores/seed in Germany, 10 spores/seed in Austria and Switzerland, and only one spore/seed in Scotland (126) and in the United Kingdom (94). In Denmark, intervention is recommended at the first detection of spores. There should be no spores in certified seed, while in organic seed the threshold is 10 spores/g seed (102). In order to meet this recommendation, there should be less than one infected tiller per 1,000 m^2 in a field used for seed production (13). To produce certified seed in Lithuania, not more than seven infected spikes/150 m^2 can be tolerated, precluding the use of susceptible cultivars in organic seed production (78). However, these are general recommendations, and the treatment threshold levels could be different for certain cultivars, according to their level of bunt resistance, and also for different environments. In their study, Waldow and Jahn (126) determined the treatment threshold levels for three cultivars, two highly susceptible and one moderately susceptible. They inoculated these cultivars with three inoculum levels: 20, 100, and 1,000 spores/seed and then treated these with Tillecur or hot water before planting. Independent of site and year, 5 to 20 spores/seed were sufficient to produce a distinct infection in susceptible cultivars. The results show that a threshold of 20 spores/seed, as was proposed in Germany, is too high for more susceptible cultivars. The threshold for

susceptible cultivars should be 1 to 5 spores/seed, and 20 spores/seed for moderately susceptible cultivars (126). Establishing the correct treatment thresholds for specific cultivars and environments is important to avoid the build-up of common bunt, especially in organic seed production.

Reduction of spore load by physical methods. Common bunt incidence in the field could be reduced if bunt contamination in seed lots is decreased. Borgen (9) studied the effectiveness of brush cleaning, preceded by conventional air cleaning of seed, to reduce bunt contamination. The conventional air-screen precleaning reduced the number of spores by 69.4%, while the brush cleaner, when used alone, reduced contamination by 83.9%. However, if the seed lot was first cleaned by an air-screen cleaner, and then cleaned with the brush cleaner, 99.8% of the spores were removed from the seed lot. Borgen (9) recommended that seed should be treated within a short duration with a low load of seed in the brush cleaner (ø = 400 mm). However, if the bunt contamination is low, the efficacy of this treatment could be difficult to assess, since the threshold level for bunt contamination might be close to the detection level.

Since the end of the nineteenth century, hot water has been used to control seedborne diseases of wheat and barley. Nielsen et al. (103) soaked seeds contaminated with bunt spores in water at different temperatures and at different durations. After soaking in water, the seeds were subjected to heat treatment at higher temperatures and short treatment times. Adequate bunt control was only achieved by soaking seeds at 45°C and subsequent treatment at 55°C for 2 or 4 minutes.

However, the traditional hot water method is costly and complicated, especially with large quantities of seed which need to be dried afterward. Due to this constraint, several institutions developed different types of equipment for thermal treatment of seed. Experiments on a combination of vapor and microwaves were carried out at the Göttingen University in Germany, while experiments with vapor treatment were conducted at the PlanteForsk in Norway. In Germany, grains are irradiated with electro-rays of the same type as those used in television tubes (8). Borgen et al. (12) tested the effect of surface heat sterilization on the control of common bunt in wheat and spelt. They exposed contaminated seed to a combination of steam and ultrasound. Air molecules will fluctuate in the air chamber due to the ultrasound, thereby increasing the exposure of the seed surface to the hot steam. Under this treatment, spores in wheat were eliminated after 4 seconds, and in spelt after 8 seconds. This technology (SonoSteam) was initially used to eliminate pathogens on food surfaces (11). A high-precision treatment with hot, humid air to kill seedborne fungi, including *T. caries*, was developed at the Swedish University of Agricultural Sciences (36,37) and is now marketed as ThermoSeed (38).

Organic seed treatments. Tillecur is a yellow mustard powder product that is applied as slurry to seeds before sowing. In two replicated field experiments, the highest level of bunt control was obtained with Tillecur compared with nine noncommercial biological control agents and 11 commercial products (67). Waldow and Jahn (126) compared hot water to Tillecur in their efficacy against common bunt. Seeds of three cultivars (two highly susceptible and one moderately susceptible) were inoculated with different spore concentrations and treated with Tillecur or hot water before planting. Bunt infection rates in the treated plants varied according to inoculum dose, cultivar, and treatment method, with additional variation between respective years and sites. Tillecur provided the most effective bunt control, with the number of infested spikes low to zero for all sites and inoculum doses. Hot water was less effective, and its effect was not significant except at the high inoculum level. Although Tillecur did not always suppress bunt infection completely, it did reduce disease development.

El-Naimi et al. (31) used powdered skimmed milk, hucket (local skimmed milk), and wheat flour as alternative seed treatments in West Asian and North African countries. They conducted field tests over 4 years at ICARDA and observed an average of 96% reduction of common bunt incidence when skimmed milk was applied to inoculated seeds before planting. When hucket was used, there was a 93% reduction in bunt compared to the control, and 62% when wheat flour was used. The results were consistent over seasons and against both *T. caries* and *T. laevis.* The authors hypothesized that although these substances did not kill the teliospores, they could have increased the antagonistic potential of unknown soilborne microorganisms. They could also have produced toxic metabolites that inhibited teliospore germination. Further studies are needed to determine the effect of skimmed milk powder and hucket on seed germination, field emergence, and grain yield, as well as on the application technique and economic return of these substances.

Borgen and Nielsen (14) treated seed inoculated with bunt spores with 5% acetic acid (AA) solutions at 20 ml kg^{-1} or higher under field conditions. They observed that the bunt incidence of plants grown from treated seeds was reduced by 91.5 to 96.2% in winter wheat and 83% in spring wheat. They did not observe any negative effects of the AA on seed germination and seedling vigor. Instead of acid solutions, Sholberg et al. (119) used acetic acid vapors as fumigants to control common bunt. They inoculated seeds of the highly susceptible cultivar Laura with composite races of *T. caries* and *T. laevis*, then fumigated the inoculated seeds with 2 and 4 g kg^{-1} AA vapor for 1 hour at 20°C. Results from 3-year field trials showed that both rates were as effective as Vitavax, but the higher rate reduced bunt incidence more than the lower rate of fumigant. Fumigation with acetic acid could be an organic alternative to fungicides, as long as commercial facilities for fumigating grain with acetic acid vapors can be constructed.

Biological control. There are several reports on the biological control of common bunt. As early as 1975, Kollmorgen and Jones (69) demonstrated that isolates of *Streptomyces* and *Bacillus* species can cause marked reductions in teliospore germination of *T. caries* and *T. laevis* in vitro. In a follow up study, Kollmorgen (68) observed that *Bacillus* species reduced disease incidence of common bunt under field conditions. McManus et al. (93) reported that some strains of *Pseudomonas fluorescens* inhibited the germination of *T. laevis* teliospores and reduced bunt incidence by 65% when wheat seeds were inoculated with these strains. Hokeberg et al. (56) and Johnsson et al. (63) found that one *P. chlororaphis* isolate, MA 342, is a potent inhibitor of *T. caries* in the greenhouse and in the field. This strain has been developed into the commercial biopesticides, Cedomon and Cerall. Cedomon is used in barley and oats against the seedborne pathogen *Pyrenophora* spp., while Cerall has been approved for use in wheat, rye, and triticale against seedborne pathogens including *T. caries*. It is registered in Sweden, Austria, Finland, Switzerland, and Lithuania. Dromph and Borgen (24) studied the effect of collembolans on the viability of soilborne inoculum of common bunt. Teliospores of *T. caries* were fed to five species of collembolans, and they observed that ingestion by collembolans almost completely inhibits germination of teliospores, thus reducing wheat infection. Aside from this study, the use of soil microflora against common bunt spores remains unexplored.

Kosted et al. (70) investigated the possibility of using the mating pheromones of *Ustilago hordei*, causal agent of covered smut of barley, to reduce teliospore germination in *T. caries.* It has been shown that the mating pheromones of *U. hordei* break down to smaller peptide compounds that act as powerful inhibitors of mating and germination in several fungal species (118). Synthetic peptide analogs of the pheromone derivatives were farnesylated, methyl esterified, or both, and tested for preventing mating in *U. hordei* and teliospore germination in *T. caries*. Greenhouse studies showed that using selected antagonists to inhibit mating and teliospore germination decreased the incidence of covered smut of barley and common bunt of wheat, although the level of control was inconsistent. It is critical that the pathogens are exposed to the compounds at the right time and place to achieve more reliable control.

Goates and Mercier (49) evaluated the effect of volatiles from *Muscodor albus,* a fungal endophyte (120), on the viability of teliospores of several *Tilletia* species, including *T. caries*. The vola-

tiles of *M. albus* have been shown to possess fungicidal activity against *Rhizoctonia solani* and *U. hordei,* among other species (97,120). After fumigation, the teliospores were incubated at 20 to 22°C on agar for up to 5 weeks to assess their viability. Results showed that *T. caries* teliospores and those of the other *Tilletia* species were highly susceptible to volatiles of *M. albus* when they are physiologically active or in the process of germination. However, these volatiles do not inhibit dry dormant teliospores. Further work demonstrated that *Muscodor albus* was effective when applied as a seed treatment or when applied in furrow during planting (50).

The Challenge Remains

Common bunt is one of the most destructive diseases of wheat in the United States, Canada, Europe, and the semiarid regions of western Asia and northern Africa. Its management was intensively studied for decades until an effective chemical seed treatment was discovered. Once bunt was effectively controlled with chemicals, it dwindled in significance, essentially becoming a forgotten disease. Research on host resistance and pathogen population biology was given low priority until the disease re-emerged in organic wheat. In Europe, the regulation that only organically produced seed may be used in organic farms has caused common bunt to be one of the major threats in organic wheat. Given the dearth of information on host resistance and pathogen biology, scientists have to start from the basics: surveying for bunt incidence, monitoring pathogenic races, screening wheat germplasm for bunt resistance, identifying new sources of resistance, and initiating a breeding program for bunt resistance.

The use of resistant cultivars is one of the cornerstones of managing common bunt. It is economical and has ecological benefits, and can limit disease outbreaks (30). However, most modern cultivars are highly susceptible to common bunt because breeding programs designed for conventional agriculture have little interest in breeding for resistance to the disease. In addition, existing resistant cultivars possess only one or two major race-specific genes for resistance. Due to the gene-for-gene interaction that exists between specific bunt avirulence genes and bunt resistance genes in wheat (48,54), the resistance of a cultivar is frequently overcome. Virulent types of the pathogen are rapidly selected from the bunt populations by host screening, changing the race populations. Because of the dynamics of host and pathogen interactions, the resistance of a cultivar is sometimes short-lived, its durability depending on the genetic plasticity of the bunt population with which it interacts (54). Thus, a continuing program of breeding bunt-resistant cultivars is vital to the sustainable control of common bunt.

Given the heterogeneity of organic farming systems, it is essential that bunt-resistant cultivars be adapted to organic and low-input cropping systems. Cultivars to be used in organic systems should possess certain traits lacking in cultivars bred under conventional agriculture (100). Most of these traits, such as improved nutrient-use efficiency, weed competitiveness, and disease resistance, are often compensated for by inputs such as inorganic fertilizers and chemical pesticides in conventional agriculture (73). In organic agriculture, these traits must be directly selected. Thus, breeding for bunt resistance should be done under organic conditions and needs a different approach since organic and low-input farms are more heterogeneous compared to conventional farms. Also, organic farming is more dependent on local conditions than conventional agriculture. The greater diversity of weed and insect pests and diseases calls for more variable management systems, resulting in significant genotype × environment × management interactions (73). It must also be emphasized that breeding for disease resistance in organic agriculture differs from the conventional agriculture approach, particularly with regard to the diseases targeted (82). Often, organic systems require resistance to specific diseases that are not essential to conventional management due to control by pesticides. Organic breeding programs will be forced to target resistance to diseases that lack a certified organic cultural control. At present, there are seven winter wheat cultivars, selected under organic conditions, released in Austria (82) and 10 in Germany (73).

Work on bunt resistance has focused on major genes probably due to the very low threshold levels allowed for spore contamination, particularly in organic seed. Also, given the potential of the disease to build up over time, even low initial bunt inoculum can increase significantly in the succeeding years, especially if untreated, farm-saved seed is used every season. A study in Denmark showed that the number of spores in the seed lot is proportional to the number of infected plants in the field (13). Because of the extremely low tolerance for spore contamination, efforts on screening for bunt resistance are still focused on major, race-specific resistance. The use of minor genes with additive effects can be effective in reducing disease severity, as seen in leaf and yellow rust (30). However, apart from the study of Fofana et al. (35), there has been little work on identifying sources of quantitative or nonspecific resistance to bunt and evaluating their potential usefulness. Realistically, nonspecific resistance alone cannot give the level of bunt control desired by organic farmers. But combined with race-specific resistance genes, general resistance genes could prolong the effectiveness of a limited number of major genes (54). The incorporation of minor genes could produce a durable type of resistance that can prevent the "boom and bust" cycle that has been observed in the past, especially in the PNW.

Since the early twentieth century, it has been recognized that the existence of races in the common bunt pathogens is the single most important impediment to long-term bunt management (19). What is needed is genetic diversity for resistance that would prevent selection of more virulent races. For other host–pathogen systems, the use of cultivar mixtures and multilines has diversified resistance genes, thereby restricting pathogen spread and directing pathogen evolution. However, the use of cultivar mixtures and multilines for functional diversity has not been investigated in common bunt, probably because it is a seedborne, monocyclic disease. Cultivar mixtures and multilines have been found to be effective in polycyclic, foliar diseases.

Another route to increase diversity for resistance is to integrate it into the breeding process, with the use of evolutionary breeding methods to create composite cross populations (CCP) (10,32) and modern landraces (101). CCP are genetically diverse populations created from crossing, in all possible combinations, several or many parental lines. The hybrids are bulked together for propagation and subjected to mass selection. These lines or populations have a broad genetic base and can adapt to the natural or artificial selection pressures imposed during the breeding process. As a result, they have improved fitness to the local environment, i.e., organic farming conditions, which are more heterogeneous due to greater landscape diversity (83) and are more difficult to characterize than conventional farming systems. On the other hand, "modern landraces" are bulk populations developed from superior germplasm and further subjected to local selection (101). Both CCP and modern landraces invite farmer participation in the process of selection, and promise the advantage of farm suitability and profitability. A new project in Denmark will use CCP and marker assisted selection to achieve more durable bunt resistance (15).

Aside from resistant cultivars, lines, or populations adapted to organic agriculture, it is also imperative that disease-free seed be available to farmers. Common bunt has the potential to increase rapidly to economically intolerable levels within a few growing seasons, and alternative seed treatments should give satisfactory bunt control, especially for organic seed production. Seed treatments compatible with the principles of organic agriculture have been developed, such as novel methods of using heat treatments, the application of substances of natural origin like milk and whey powder, acetic acid, plant extracts or microorganisms, and volatile compounds from fungi. However, not all of these methods have been tested sufficiently and need further refinement to be practical and effective on a large scale. The proprietary products Tillecur, Cerall, and ThermoSeed also need to be included in independent

tests conducted by researchers outside of the companies. Studies have to be conducted over years, across locations, and against various bunt race populations, and in combination with resistant wheat cultivars, for more general recommendations to be made.

Cultivar-specific treatment thresholds also have to be established for each country, according to the end-use of the seed. Sensitive detection methods of bunt spores in organic seeds could help in the establishment of treatment thresholds. McNeil et al. (94) developed a real-time PCR assay to quantify the level of bunt contamination in UK seed lots. The technology has increased test output and sensitivity of detection, and the authors recommend an advisory threshold of one spore per seed in the UK. Kellerer et al. (65) developed PCR and immunological methods to detect *Tilletia* species from organic seed lots. Using Western blotting, they could verify infection by *T. caries* from freshly harvested grains within 5 hours and by PCR within 3 hours. These methods were developed to identify *Tilletia* species in cases where there are not enough teliospores to make positive identification. Normally, *T. caries* and *T. laevis* are easily distinguished with light microscopy. However, identification using individual teliospores could be difficult, because there is a 10% overlap in the morphology of *T. caries* and *T. controversa*, the causal agent of dwarf bunt (48). Also, studies have shown that *T. caries* can hybridize with *T. laevis* and with *T. controversa*, resulting in a full range of morphological variants (57,58) which could make identification to the species level difficult. Understanding pathogen variability across geographical areas, including their evolution over time, is critical for disease management (30). However, there are still significant gaps in our knowledge of the common bunt pathogens. There are very few studies on the genetic variability and population biology of the common bunt pathogens. Because of the difficulty of hybridizing *T. caries* isolates, genetic studies with the common bunt fungi have not kept pace with similar studies in fungi under the *Ustilaginales* (89,121) and other host–parasite systems (54). At present, not much is known of the genetics of the pathogens to provide sound hypotheses to explain the mechanisms of variation, especially of variation leading to the production of new pathogenic races. In a review of the genetics of the smut fungi, Thomas (121) said that little is known of the function of virulence genes, nor have any been mapped. More than 10 years after his article was published, there are still no studies on the effectors or virulence determinants of the pathogens.

In the United States, common bunt is not yet a major problem in organic wheat. This is probably because conventionally produced seed, as long as it has not been treated with synthetic chemicals, can still be used in organic farms. But should organic standards require that only organically produced seed may be used in organic farms, the bunt problem will inevitably return. In anticipation of this, scientists at Washington State University (WSU) in Pullman have started breeding wheat under organic conditions, and are screening breeding lines for bunt resistance (K. M. Murphy and S. S. Jones, *unpublished*). Identification of the resistance genes in U.S. modern wheat cultivars and their chromosomal locations, as well as studies into the genetic diversity and population structure of *T. caries,* are also underway (J. B. Matanguihan and S. S. Jones, *unpublished*).

Management of common bunt under organic agriculture and low-input systems must be holistic and integrated. We can no longer rely on a single control measure. In addition to resistant cultivars and organic seed treatments, measures to reduce airborne and soilborne inoculum could be integrated with sowing dates and crop rotation to prevent common bunt (7,71). Other factors involved in the epidemiology of the disease and pathogen biology may suggest additional options for a systems perspective in disease management. Management of common bunt in organic wheat will need an interdisciplinary approach conducted on organic farms with strong grower participation.

Chemical seed treatments had been so effective against both seedborne and soilborne teliospores of *T. caries* and *T. laevis* that common bunt virtually disappeared in the United States in the late 1950s (48). Bruehl, a professor in the Department of Plant Pathology, WSU, once lamented that plant pathologists and plant breeders no longer have the challenge of keeping up with the common bunt pathogens (17). They moved on to other diseases, and common bunt was relegated to the status of an ancient disease that is no longer a threat. Less than 50 years later, the disease has come back. In organic wheat, the challenge remains.

Literature Cited

1. Al-Maaroof, E. M., Shams Allah, S. A., and Hassan, M. S. 2006. Current status of wheat bunt disease in Iraq. Czech J. Genet. Plant Breed. 42(Special Issue):45-50.
2. Atta Hosaini, S. M., Shams Allah, S. A., and Hassan, M. S. 2003. Physiologic races of *Tilletia laevis* in Khorasan. Seed Plant 18:383-393.
3. Babayants, L. T., Babayants, O. V., Baranovskaya, V. L., and Dubinina, L. A. 2006. *Tilletia caries* and resistance of wheat to this pathogen in Ukraine. Czech J. Genet. Plant Breed. 42(Special Issue):33-36.
4. Bartoš, P., Šip, V., Chrpová, J., Vacke, J., Stuchlíková, E., and Blažková, V. 2002. Achievements and prospects of wheat breeding for disease resistance. Czech J. Genet. Plant Breed. 38:16-28.
5. Blažkova, V., and Bartoš, P. 2002. Virulence pattern of European bunt samples (*Tilletia tritici* and *T. laevis*) and sources of resistance. Cereal Res. Comm. 30:335-342.
6. Bonman, J. M., Bockelman, H., Goates, B., Obert, D., McGuire, P., Qualset, C., and Hijmans, R. 2006. Geographic distribution of common and dwarf bunt resistance in landraces of *Triticum aestivum* subsp. *aestivum*. Crop Sci. 46:1622-1629.
7. Borgen, A. 2000. Perennial survival of common bunt (*Tilletia tritici*) in soil under modern farming practice. J. Plant Dis. Prot. (Z. Pflanzenk. Pflanzen.) 107:182-188.
8. Borgen, A. 2003. Strategies for regulation of seedborne diseases in organic farming. Seed Test. Int. 127:19-21.
9. Borgen, A. 2005. Removal of bunt spores from wheat seed lots by brush cleaning. ICARDA Seed Info. No. 29, July 2005.
10. Borgen, A. 2006. Introductory considerations on crop diversity. Pages 9-12 in: Proc. COST SUSVAR Workshop Cereal Crop Diversity: Implications Crop Production Products, June 13-14, La Besse, France.
11. Borgen, A. 2010. SonoSteam heat treatment to control common bunt in wheat and spelt. XVI Bienn. Workshop on the Smuts and Bunts, Lethbridge, Alberta, Canada.
12. Borgen, A., Krebs, N., and Langkjær, C. 2005. Novel development of heat treatment techniques for seed surface sterilization. Page 28 in: SHC Seed Health Symposium, 5th, Angers, France.
13. Borgen, A., and Kristensen, L. 2010. Spore contamination of *Tilletia tritici* in seed lots as affected by field disease incidence. XVI Bienn. Workshop on the Smuts and Bunts, Lethbridge, Alberta, Canada.
14. Borgen, A., and Nielsen, B. 2001. Effect of seed treatment with acetic acid for control of seedborne diseases. Pages 135-140 in: Proc. BCPC Symposium No. 76, Seed Treatment: Challenges and Opportunities, Feb. 26-27, UK.
15. Borgen, A., Rasmussen, S. K., and Backes, G. 2010. BioBreed – a new project on marker assisted population breeding in wheat with resistance to common bunt. XVI Bienn. Workshop on the Smuts and Bunts, Lethbridge, Alberta, Canada.
16. Bruehl, G. 1989. Integrated control of soil-borne pathogens: An overview. Can. J. Plant Pathol. 11:153-157.
17. Bruehl, G. 1990. Cereal Research at Pullman. In: History of the Department of Plant Pathology. Available from http://plantpath.wsu.edu/aboutplantpath/history.htm
18. Carris, L. M. 2010. Common bunt (stinking smut). Pages 60-61 in: Compendium of Wheat Diseases and Pests. 3rd ed. W. Bockus, R. Bowden, R. Hunger, W. Morrill, T. Murray, and R. Smiley, eds. American Phytopathological Society, St. Paul, MN.
19. Christensen, C. M., Stakman, E. C., and Christensen, J. J. 1947. Variation in phytopathogenic fungi. Annu. Rev. Microbiol. 1:61-84.
20. Ciucă, M., and Săulescu, N. 2008. Screening Romanian winter wheat germplasm for presence of *Bt10* bunt resistance gene using molecular markers. Romanian Agric. Res. 25:1-5.
21. Coţa, L. C., Botez, C., Grigoraş, M., and Curticiu, D. 2009. Screening for resistance to artificial infection by common bunt (*Tilletia caries* and *Tilletia foetida*) in F_2 populations of wheat (*Triticum aestivum* L.). Bull. Univ. Agric. Sci. Vet. Med. (USAMV) 66:24-31.
22. Dariaee, A., Biglar, H., and Haghparast, R. 2006. Identification of new wheat common bunt pathotypes (*Tilletia laevis* Kühn). Comm. Agric. Appl. Biol. Sci. 71:1093-1101.
23. Demeke, T., Laroche, A., and Gaudet, D. A. 1996. A DNA marker for the *Bt-10* common bunt resistance gene in wheat. Genome 39:51-55.

24. Dromph, K. M., and Borgen, A. 2001. Reduction of viability of soilborne inoculum of common bunt (*Tilletia tritici*) by collembolans. Soil Biol. Biochem. 33:1791-1795.
25. Dumalasová, V., and Bartoš, P. 2006. Reaction of winter wheat cultivars registered in the Czech Republic to common bunt *Tilletia tritici* (Bjerk.) Wint. and *T. laevis* Kühn. Cereal Res. Comm. 34:1275-1282.
26. Dumalasová, V., and Bartoš, P. 2006. Resistance of winter wheat cultivars to common bunt *Tilletia tritici* (Bjerk.) Wint. and *T. laevis* Kühn. J. Plant Dis. Prot. 113:159-163.
27. Dumalasová, V., and Bartoš, P. 2007. Reaction of winter wheat cultivars to common bunt *Tilletia tritici* (Bjerk.) Wint. and *T. laevis* Kühn. Plant Prot. Sci. 43:138-141.
28. Dumalasová, V., and Bartoš, P. 2008. Effect of inoculum doses on common bunt infection on wheat caused by *Tilletia tritici* and *T. laevis*. Czech J. Genet. Plant Breed. 44:73-77.
29. Dumalasová, V., and Bartoš, P. 2010. Reaction of wheat, alternative wheat and triticale cultivars to common bunt. Czech J. Genet. Plant Breed. 46:14-20.
30. Duveiller, E., Singh, R. P., and Nicol, J. M. 2007. The challenges of maintaining wheat productivity: Pests, diseases, and potential epidemics. Euphytica 157:417-430.
31. El-Naimi, M., Toubia-Rahme, H., and Mamluk, O. F. 2000. Organic seed treatment as a substitute for chemical seed-treatment to control common bunt of wheat. Eur. J. Plant Pathol. 106:433-437.
32. Finckh, M. 2008. Integration of breeding and technology into diversification strategies for disease control in modern agriculture. Eur. J. Plant Pathol. 121:399-409.
33. Fischer, G., and Holton, C. 1957. Biology and Control of the Smut Fungi. Ronald Press Company, New York.
34. Flor, H. 1933. Studies on physiologic specialization in *Tilletia tritici* and *T. levis* in the Pacific Northwest. J. Agric. Res. (Washington, D.C.) 47:193-213.
35. Fofana, B., Humphreys, D. G., Cloutier, S., McCartney, C. A., and Somers, D. J. 2008. Mapping quantitative trait loci controlling common bunt resistance in a doubled haploid population derived from the spring wheat cross RL4452 × AC Domain. Mol. Breed. 21:317-325.
36. Forsberg, G. 2001. Heat sanitation of cereal seeds with a new, efficient, cheap and environmentally friendly method. Pages 69-72 in: BCPC Symposium Proc. No. 76, Seed Treatment, Challenges and Opportunities.
37. Forsberg, G. 2004. Control of cereal seedborne diseases by hot, humid air seed treatment. Ph.D. diss. Swedish University of Agricultural Sciences, Uppsala.
38. Forsberg, G., and Sanchez-Sava, V. 2009. ThermoSeed treatment – a novel disinfection technology for vegetable seeds. Pages 1-6 in: BCPC Symposium Proc. Seed Production Treatment in a Changing Environment, Feb. 24-25, West Midlands, UK.
39. Gaines, E. F. 1920. The inheritance of resistance to bunt or stinking smut of wheat. J. Am. Soc. Agron. 12:124-132.
40. Gaines, E. F. 1923. Genetics of bunt resistance in wheat. J. Agric. Res. (Washington, D.C.) 23:445-479.
41. Gaines, E. F. 1925. The inheritance of disease resistance in wheat and oats. Phytopathology 15:341-349.
42. Gaines, E. F. 1928. New physiological forms of *Tilletia levis* and *T. tritici*. Phytopathology 18:579-588.
43. Gaines, E. F. 1928. New physiologic forms of *Tilletia tritici* in wheat. Phytopathology 18:139.
44. Galaev, A., Babayants, L., and Sivolap, Y. 2006. Molecular mapping and marking of the bunt resistance gene transferred from *Aegilops cylindrica* to soft wheat. Cytol. Genet. 40:65-70.
45. Gaudet, D. A., Lu, Z.-X., Leggett, F., Puchalski, B., and Laroche, A. 2007. Compatible and incompatible interactions in wheat involving the Bt-10 gene for resistance to *Tilletia tritici*, the common bunt pathogen. Phytopathology 97:1397-1405.
46. Gaudet, D. A., and Puchalski, B. J. 1989. Status of bunt resistance in western Canadian spring wheat and triticale. Can. J. Plant Sci. 69:797-804.
47. Gaudet, D. A., Puchalski, B., and Kozub, G. C. 1995. Reaction of CIMMYT and Canadian Red spring wheat cultivars to common bunt (*Tilletia tritici* and *T. laevis*). Cereal Res. Comm. 23:141-146.
48. Goates, B. J. 1996. Common bunt and dwarf bunt. Pages 12-25 in: Bunt and Smut Diseases of Wheat: Concepts and Methods of Disease Management. R. D. Wilcoxson and E. E. Saari, eds. CIMMYT, Mexico City.
49. Goates, B. J., and Mercier, J. 2009. Effect of biofumigation with volatiles from *Muscodor albus* on the viability of *Tilletia* spp. teliospores. Can. J. Microbiol. 55:203-206.
50. Goates, B. J., and Mercier, J. 2009. Control of common bunt of wheat under field conditions by seed and in furrow treatments with the biofumigant fungus *Muscodor albus*. (Abstr.) Phytopathology 99:S44.
51. Hajjar, R., and Hodgkin, T. 2007. The use of wild relatives in crop improvement: A survey of developments over the last 20 years. Euphytica 156:1-13.
52. He, C., and Hughes, G. R. 2003. Inheritance of resistance to common bunt in spelt and common wheat. Can. J. Plant Sci. 83:47-56.
53. He, C., and Hughes, G. R. 2003. Development of RAPD markers associated with common bunt resistance to race *T1* (*Tilletia tritici*) in spelt wheat. Plant Breed. 122:375-377.
54. Hoffman, J. A. 1982. Bunt of wheat. Plant Dis. 66:979-987.
55. Hoffman, J. A., and Metzger, R. J. 1976. Current status of virulence genes and pathogenic races of the wheat bunt fungi in the northwestern USA. Phytopathology 66:657-660.
56. Hokeberg, M., Gerhardson, B., and Johnsson, L. 1997. Biological control of cereal seed-borne diseases by seed bacterization with greenhouse selected bacteria. Eur. J. Plant Pathol. 103:25-33.
57. 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.
58. Holton, C. S. 1954. Natural hybridization between common and dwarf bunt as related to the problem of delimitation of species of *Tilletia* occurring on wheat. Phytopathology 44:493.
59. Holton, C. S., and Heald, F. D. 1941. Bunt or stinking smut of wheat: A world problem. Burgess Publishing Co., Minneapolis, MN.
60. Holton, C. S., and Purdy, L. H. 1954. Control of soil-borne common bunt of winter wheat in the Pacific Northwest by seed treatment. Plant Dis. Rep. 38:753-754.
61. Huber, K., and Buerstmayr, H. 2006. Development of methods for bunt resistance breeding for organic farming. Czech J. Genet. Plant Breed. 42(Special Issue):66-71.
62. Ismail, S. F., Mamluk, O. F., and Azmeh, M. F. 1995. New pathotypes of common bunt of wheat from Syria. Phytopathol. Mediterr. 34:1-6.
63. Johnsson, L., Hokeberg, M., and Gerhardson, B. 1998. Performance of the *Pseudomonas chlororaphis* biocontrol agent MA 342 against cereal seedborne diseases in field experiments. Eur. J. Plant Pathol. 104:701-711.
64. Jones, S., Murray, T., and Allan, R. 1995. Use of alien genes for the development of disease resistance in wheat. Annu. Rev. Phytopathol. 33:429-443.
65. Kellerer, T., Sedlmeier, M., Rabenstein, F., and Killerman, B. 2006. Development of immunochemical and PCR methods for qualitative detection of *Tilletia* species in organic seeds. Czech J. Genet. Plant Breed. 42(Special Issue):72-74.
66. Knox, R. E., Fernandez, M. R., Brule-Babel, A. L., and DePauw, R. M. 1998. Inheritance of common bunt resistance in androgenetically derived doubled haploid and random inbred populations of wheat. Crop Sci. 38:1119-1124.
67. Koch, E., Well, B., Wächter, R., Wohlleben, S., Spiess, H., and Krauthausen, H.-J. 2006. Evaluation of selected microbial strains and commercial alternative products as seed treatments for the control of *Tilletia tritici*, *Fusarium culmorum, Drechslera graminea* and *D. teres*. J. Plant Dis. Prot. 113:150-158.
68. Kollmorgen, J. F. 1976. Studies on biological control of bunt of wheat (*Tilletia caries*, *T. foetida*). Aust. Plant Pathol. Soc. Newsl. 5:48-51.
69. Kollmorgen, J. F., and Jones, L. C. 1975. The effects of soil-borne microorganisms on the germination of chlamydospores of *Tilletia caries* and *T. foetida*. Soil Biol. Biochem. 7:407-410.
70. Kosted, P. J., Gerhardt, S. A., and Sherwood, J. E. 2002. Pheromone-related inhibitors of *Ustilago hordei* mating and *Tilletia tritici* teliospore germination. Phytopathology 92:210-216.
71. Kristensen, L., and Borgen, A. 2000. Reducing spread of spores of common bunt disease (*Tilletia tritici*) via combining equipment. Biol. Agric. Hortic. 19:9-18.
72. Kubiak, K., and Weber, Z. 2008. Virulence frequency of *Tilletia caries* and the occurrence of common bunt on 20 winter wheat cultivars. Phytopathol. Pol. 47:11-19.
73. Lammerts van Bueren, E. T., Jones, S. S., Tamm, L., Murphy, K. M., Myers, J. R., Leifert, C., and Messmer, M. The need to breed crop varieties suitable for organic farming, using wheat, tomato and broccoli as examples: a review. NJAS-Wageningen J. Life Sci. (2010), doi:10.1016/j.njas.2010.04.001.
74. Lammerts van Bueren, E. T., Østergård, H., Goldringer, I., and Scholten, O. 2008. Plant breeding for organic and sustainable, low-input agriculture: Dealing with genotype-environment interactions. Euphytica 163:321-322.
75. Lammerts van Bueren, E. T., Struik, P. C. and Jacobsen, E. 2002. Ecological concepts in organic farming and their consequences for an organic crop ideotype. Neth. J. Agric. Sci. 50:1-26.
76. Lammerts van Bueren, E. T., Struik, P. C., and Jacobsen, E. 2003. Organic propagation and planting material: An overview of problems and challenges for research. NJAS-Wageningen J. Life Sci. 51:263-277.
77. Laroche, A., Demeke, T., Gaudet, D., Puchalski, B., Frick, M., and McKenzie, R. 2000. Development of a PCR marker for rapid identification of the *Bt10* gene for common bunt resistance in wheat. Genome 43:217-223.
78. Liatukas, P., and Ruzgas, V. 2005. Genetic resources for organic wheat breeding: Impact on resistance to common bunt. BIOLOGIJA 3:62-64.
79. Liatukas, Z., and Ruzgas, V. 2006. Peculiarities of selection for winter wheat resistance to common bunt. Agron. Res. 4(Special Issue):257-261.
80. Liatukas, Z., and Ruzgas, V. 2007. The effect of alien translocations on winter wheat resistance to *Tilletia tritici*. BIOLOGIJA 53:59-62.
81. Liatukas, Z., and Ruzgas, V. 2008. Resistance genes and sources for the control of wheat common bunt (*Tilletia tritici* (DC.) Tul.). BIOLOGIJA 54:274-278.

82. Löschenberger, F., Fleck, A., Grausgruber, H., Hetzendorfer, H., Hof, G., Lafferty, J., Marn, M., Neumayer, A., Pfaffinger, G., and Birschitzky, J. 2008. Breeding for organic agriculture: The example of winter wheat in Austria. Euphytica 163:469-480.
83. Lotter, D. W. 2003. Organic agriculture. J. Sustainable Agric. 21:59-128.
84. Lu, Z.-X., Gaudet, D., Frick, M., Puchalski, B., Genswein, B., and Laroche, A. 2005. Identification and characterization of genes differentially expressed in the resistance reaction in wheat infected with *Tilletia tritici*, the common bunt pathogen. J. Biochem. Mol. Bio. 38:420-431.
85. Lu, Z.-X., Gaudet, D. A., Puchalski, B., Despins, T., Frick, M., and Laroche, A. 2006. Inducers of resistance reduce common bunt infection in wheat seedlings while differentially regulating defense-gene expression. Physiol. Mol. Plant Pathol. 67:138-148.
86. Mamluk, O. F. 1998. Bunts and smuts of wheat in North Africa and the Near East. Euphytica 100:45-50.
87. Mamluk, O. F., and Nachit, M. M. 1994. Sources of resistance to common bunt (*Tilletia foetida* and *T. caries*) in durum wheat. J. Phytopathol. 142:122-130.
88. Martin, A., Alvarez, J. B., Martin, L. M., Barro, F., and Ballesteros, J. 1999. The development of tritordeum: A novel cereal for food processing. J. Cereal Sci. 30:85-95.
89. Martinez-Espinoza, A. D., Garcia-Pedrajas, M. D., and Gold, S. E. 2002. The Ustilaginales as Plant Pests and Model Systems. Fungal Genet. Biol. 35:1-20.
90. Martynov, S., Dobrotvorskaya, T., and Sorokin, O. 2004. Comparative genealogical analysis of the resistance of winter wheat to common bunt. Russian J. Genet. 40:410-424.
91. Mathre, D. E., Johnston, R. H., and Grey, W. E. 2001. Small Grain Cereal Seed Treatment. The Plant Health Instructor. DOI: 10.1094/PHI-I-2001-1008-01. Updated, 2006.
92. McIntosh, R., Hart, G., Devos, K., Gale, M., and Rogers, W. 1998. Catalogue of gene symbols for wheat. Pages 143-144 in: Proc. Int. Wheat Genet. Symposium, 9th. University Extension Press, University of Saskatchewan.
93. McManus, P. S., Ravenscroft, A. V., and Fulbright, D. W. 1993. Inhibition of *Tilletia laevis* teliospore germination and suppression of common bunt of wheat by *Pseudomonas fluorescens* 2-79. Plant Dis. 77:1012-1015.
94. McNeil, M., Roberts, A. M. I., Cockerell, V., and Mulholland, V. 2004. Real-time PCR assay for quantification of *Tilletia caries* contamination of UK wheat seed. Plant Pathol. 53:741-750.
95. Menzies, J., and Gaudet, D. 2009. The surprising bunts and smuts: Still a threat after all these years. Manitoba Agronomist Conference, Dec. 15-16, University of Manitoba, Winnipeg, Manitoba, CA.
96. Menzies, J., Knox, R., Popovic, Z., and Procunier, J. 2006. Common bunt resistance gene *Bt10* located on wheat chromosome 6D. Can. J. Plant Sci. 86:1409-1412.
97. Mercier, J., and Manker, D. C. 2005. Biocontrol of soil-borne diseases and plant growth enhancement in greenhouse soilless mix by the volatile-producing fungus *Muscodor albus*. Crop Prot. 24:355-362.
98. Metzger, R. J., Schaller, C. W., and Rohde, C. R. 1979. Inheritance of resistance to common bunt in wheat, C.I. 7090. Crop Sci. 19:309-312.
99. Metzger, R. J., and Silbaugh, B. A. 1971. A new factor for resistance to common bunt in hexaploid wheats. Crop Sci. 11:66-69.
100. Murphy, K. M., Campbell, K. G., Lyon, S. R., and Jones, S. S. 2007. Evidence of varietal adaptation to organic farming systems. Field Crops Res. 102:172-177.
101. Murphy, K. M., Lammer, D., Lyon, S., Carter, B., and Jones, S. S. 2004. Breeding for organic and low-input farming systems: An evolutionary-participatory breeding method for inbred cereal grains. Renewable Agric. Food Systems 20:48-55.
102. Nielsen, B. J. 2004. Threshold levels for seed borne diseases in organic cereals. DARCOFenews (Newsletter from Danish Research Centre for Organic Farming), September 2004, No. 3.
103. Nielsen, B. J., Borgen, A., and Kristensen, L. 2000. Control of seedborne diseases in production of organic cereals. Pages 171-176 in: BCPC Conference – Pest Diseases, Nov. 13-16, Brighton, UK.
104. Oncică, F., Păunescu, G., and Olaru, L. 2008. Identification of bunt resistance winter wheat lines at the Agricultural Research Development Station-Simnic. Pages 345-348 in: Proc. 43rd Croatian 3rd Int. Symposium Agric., Feb. 18-21, Opatija, Croatia.
105. Oncică, F., and Săulescu, N. 2008. Potentially new sources of genes for resistance to common bunt (*Tilletia* spp.) in winter wheat (*Triticum aestivum* L.). Pages 97-100 in: Proc. Romanian Academy-Series B: Chemistry, Life Sciences Geosciences.
106. Priekule, I. 2010. Virulence pattern of common bunt (*Tilletia caries*), Latvian population. XVI Bienn. Workshop on the Smuts and Bunts, June 14-18, Lethbridge, Alberta, Canada.
107. Purdy, L. H. 1955. Regional seed treatment tests for the control of seedborne and soil-borne common smut of winter wheat in the Pacific Northwest. Plant Dis. Rep. 39:844-849.
108. Purdy, L. H., and Kendrick, E. L. 1963. Influence of environmental factors on the development of wheat bunt in the Pacific Northwest. IV. Effect of

Janet Barroga-Matanguihan

Ms. Barroga-Matanguihan is a Ph.D. student at the Department of Crop and Soil Sciences at Washington State University (WSU). She is investigating the genetic diversity and population structure of *Tilletia caries* isolates and is mapping common bunt resistance genes in wheat. She received a Masters degree in plant pathology from WSU in 1997 and then accepted a faculty position at the Department of Plant Pathology of the College of Agriculture at the University of the Philippines, Los Baños. Her current research interests are host–pathogen interactions in the context of organic and sustainable farming systems.

K. M. Murphy

Dr. Murphy received his Ph.D. in plant breeding and genetics in 2007 from Washington State University (WSU), where he worked in the organic wheat breeding program led by Dr. Stephen Jones. His dissertation focused on enhancing weed suppression and nutritional value in organic wheat cultivars. Dr. Murphy is currently an assistant research professor in the Department of Crop and Soil Sciences at WSU, where his research interests include organic hop production, and plant breeding and agronomy for the alternative grains, quinoa and buckwheat.

S. S. Jones

Dr. Jones has a Ph.D. in genetics from the University of California at Davis. He is a professor in the Department of Crop and Soil Science at Washington State University and the director of the Northwestern Washington Research and Extension Center in Mount Vernon, WA.

soil temperature and soil moisture on infection by soil spores. Phytopathology 53:416-418.
109. Rajković, S., and Dolovac, N. 2006. Common bunt (*Tilletia tritici*) in different wheat genotypes. Czech J. Genet. Plant Breed. 42(Special Issue):16-19.
110. Rubiales, D., and Martin, A. 1999. Chromosomal location in *H. chilense* and expression of common bunt resistance in wheat addition lines. Euphytica 109:157-159.
111. Rubiales, D., Moral, A., and Martin, A. 2001. Chromosome location of resistance to septoria leaf blotch and common bunt in wheat-barley addition lines. Euphytica 122:369-372.
112. Rubiales, D., Niks, R. E., Carver, T. L., Ballesteros, J., and Martin, A. 2001. Prospects for exploitation of disease resistance from *H. chilense* in cultivated cereals. Hereditas 135:161-169.
113. Russell, P. E. 2005. A century of fungicide evolution. J. Agric. Sci. 143:11-25.
114. Ruzgas, V., and Liatukas, Z. 2009. Response of Lithuanian winter wheat advanced lines to common bunt (*Tilletia tritici* (Bjerk.) Wint). Proc. Latvian Acad. Sci. Section B. 63:51-56.
115. Schmidt, J., Morris, R., and Johnson, V. 1969. Monosomic analysis for bunt resistance in derivatives of Turkey and Oro wheats. Crop Sci. 9:286-288.
116. Sears, E., Schaller, C., and Briggs, F. 1960. Identification of the chromosome carrying the Martin gene for resistance of wheat to bunt. Can. J. Genet. Cytol. 2:262-267.
117. Shepherd, J. F. 1980. The development of new wheat varieties in the Pacific Northwest. Agric. Hist. 54:52-63.
118. Sherwood, J. E., Kosted, P. J., Anderson, C. M., and Gerhardt, S. A. 1998. Production of a mating inhibitor by *Ustilago hordei*. Phytopathology 88:456-464.
119. Sholberg, P. L., Gaudet, D. A., Puchalski, B., and Randall, P. 2006. Control of common bunt (*Tilletia tritici* and *T. laevis*) of wheat (*Triticum aestivum* cv. 'Laura') by fumigation with acetic acid vapour. Can. J. Plant Sci. 86:839-843.
120. Strobel, G. A., Dirkse, E., Sears, J., and Markworth, C. 2001. Volatile antimicrobials from *Muscodor albus*, a novel endophytic fungus. Microbiology 147:2943-2950.
121. Thomas, P. L. 1991. Genetics of small-grain smuts. Annu. Rev. Phytopathol. 29:137-148.
122. 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. Translated from the French by H. B. Humphrey. 1937. Phytopathological Classics Number 5, 191 pp.
123. Tisdale, W. H., Briggs, F. N., Mackie, W. W., Woolman, H. M., Stephens, D. E., Gaines, E. F., and Stevenson, F. J. 1925. Relative resistance of wheat to bunt in the Pacific Coast States. U.S. Dep. Agric. Bull. 1299:1-30.
124. Váňová, M., Matušinský, P., and Benada, J. 2006. Survey of incidence of bunts (*Tilletia caries* and *Tilletia controversa*) in the Czech Republic and susceptibility of winter wheat cultivars. Plant Prot. Sci. 42:21-25.
125. Veisz, O., Szunics, L., and Szunics, L. 2000. Effect of common bunt on the frost resistance and winter hardiness of wheat (*Triticum aestivum* L.) lines containing *Bt* genes. Euphytica 114:159-164.
126. Waldow, F., and Jahn, M. 2007. Investigations in the regulation of common bunt (*T. tritici*) of winter wheat with regard to threshold values, cultivar susceptibility and non-chemical protection measures. J. Plant Dis. Prot. 114:269-275.
127. Wang, S., Knox, R., DePauw, R., Clarke, F., Clarke, J., and Thomas, J. 2009. Markers to a common bunt resistance gene derived from 'Blizzard' wheat (*Triticum aestivum* L.) and mapped to chromosome arm 1BS. Theor. Appl. Genet. 119:541-553.
128. Waud, J., and Metzger, R. 1970. Inheritance of a new factor (Bt8) for resistance to common bunt in wheat. Crop Sci. 10:703-704.
129. Wolfe, M., Baresel, J., Desclaux, D., Goldringer, I., Hoad, S., Kovacks, G., Löschenberger, F., Miedaner, T., Østergård, H., and Lammerts van Bueren, E. T. 2008. Developments in breeding cereals for organic agriculture. Euphytica 163:323-346.