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SMALL GRAIN PRODUCTION MANUAL PART 14

Troubleshooting Small Grain Production

An Identification Guide to Field Problems in Small Grains

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This publication, *Troubleshooting Small Grain Production*, is the last in a fourteen-part series of University of California Cooperative Extension online publications that comprise the *Small Grain Production Manual*. The other parts cover specific aspects of small grain production practices in California:

- *Part 1: Importance of Small Grain Crops in California Agriculture*, Publication 8164
- *Part 2: Growth and Development*, Publication 8165
- *Part 3: Seedbed Preparation, Sowing, and Residue Management*, Publication 8166
- *Part 4: Fertilization*, Publication 8167
- *Part 5: Irrigation and Water Relations*, Publication 8168
- *Part 6: Pest Management—Diseases*, Publication 8169
- *Part 8: Pest Management—Vertebrates*, Publication 8171
- *Part 9: Pest Management—Weeds*, Publication 8172
- *Part 10: Small Grain Forages*, Publication 8173
- *Part 11: Small Grain Cover Crops*, Publication 8174
- *Part 12: Small Grains in Crop Rotations*, Publication 8175
- *Part 13: Harvesting and Storage*, Publication 8176

It can be difficult to diagnose nutrient deficiencies and toxicities, disease symptoms, insect damage, and herbicide injury in small grains. Symptoms of damage from herbicides, fertilizers, or environmental conditions may develop rapidly or be delayed until later in the growing season. Deficiency symptoms of one nutrient may be masked by the levels (excess or deficiency) of other nutrients, and soil pH affects the availability of some nutrients and influences deficiency and toxicity symptoms. Herbicide injury may cause plant symptoms that mimic nutritional or pathological disorders. Foliar symptoms of disease range in size, color, and configuration and may appear simultaneously with symptoms of pesticide injury, leading one to overlook the real cause of the problem. In order to accurately troubleshoot field problems, you should have a sound knowledge of how small grain growth and development affects plant nutrient uptake and needs and susceptibility to diseases and pests (see part 2, *Growth and Development*).



This publication provides descriptions and photographs of abiotic (environmental) and biotic (pest- or disease-related) disorders of small grains and includes a troubleshooting guide to assist you in correctly identifying the cause or causes of problems in the field.

TROUBLESHOOTING CROP PROBLEMS

- Determine the variety and plant growth stage.
- Check all parts of the plant for symptoms, including the leaves, stems, and roots, as well as the inside of stems and roots.
- Estimate the percentage of plants damaged in the affected part of the field.
- Determine whether the problem in the field matches the pattern of irrigation, mechanical or equipment operations, spray or fertilizer applications, insect or disease outbreaks, or soil characteristics.
- Evaluate whether weeds in or adjacent to the field share similar symptoms in order to eliminate or confirm factors such as diseases and pests, herbicide drift, or nutrients as possible causes.
- Determine the history of the problem, which can often provide the foundation for accurate diagnosis.
- Identify recent environmental events, field operations, previous crop history, and cultural practices.

NUTRIENT DEFICIENCIES

Many of the following brief descriptions of symptoms of various nutrient deficiencies, toxicities, and other common disorders of small grains have been adapted from the publication *Nutrient Deficiencies and Toxicities in Wheat: A Guide to Field Identification* (Snowball and Robson 1991), which contains a wealth of other information on the topic.

Nitrogen

Symptoms of nitrogen deficiency (see [figs. 7 and 8](#)) appear first on older leaves. The older leaves appear pale, with chlorosis beginning at the leaf tip and gradually merging into light green further down the leaf blade, while new leaves remain relatively green. As chlorosis spreads to other leaves, older leaves become totally chlorotic, changing from yellow to nearly white.

Phosphorus

Early symptoms include reduced growth and vigor. Leaves become dull dark green with slight mottling of the oldest leaves. Leaves coil to a greater extent than normal, and old leaves sometimes encase younger leaves. On older leaves chlorosis begins at the leaf tip and moves down the leaf blade. Necrosis of chlorotic areas is fairly rapid; the leaf tip becomes orange to dark brown and shrivels while the remainder of the leaf turns yellow. The base of the leaf remains dark green.

Potassium

Severe deficiency symptoms appear on the oldest leaves, although growth of the whole plant can be affected. Leaves have an unthrifty and spindly appearance. Necrosis on oldest leaves begins as necrotic speckling along the length of the leaf and spreads rapidly to the tip and margins. An arrow of green tissue can remain pointing upward from the base. Chlorotic tissue, generally seen as a mottling, rapidly turns necrotic. Complete death of old leaves is common, and plants in the field may appear to have dried prematurely due to drought stress.

Sulfur

Symptoms of sulfur deficiency are similar to those of nitrogen deficiency except that the whole plant is pale, with greater chlorosis of young leaves. The pattern of chlorosis of new leaves may show gradation in intensity from tip to base, but leaves rapidly become totally chlorotic with a light yellow color. Leaf tip necrosis can appear, but it may indicate nitrate accumulation rather than a direct result of sulfur deficiency.

Magnesium

Young leaves are pale (in contrast to old leaves, which remain dark) and soon become chlorotic and remain unopened, resulting in a twisted appearance similar to that of drought-stressed plants. If the deficiency is severe, the entire length of the leaf remains folded or rolled. Chlorosis of new leaves becomes mottled and finally necrotic; older leaves may develop a mottled chlorosis and in some cases a reddish coloration along the margins.

Iron

Symptoms of iron deficiency are similar to those of magnesium deficiency in that new leaves are affected first and become chlorotic. Iron deficiency differs in that there is a more marked contrast between the green of old leaves and the chlorosis of new growth. Also, leaves show longitudinal interveinal chlorosis, resulting in a pattern of alternate green and yellow striping. Under severe deficiency new growth appears completely devoid of chlorophyll and turns white.

Manganese

Symptoms appear first in new leaves, which become pale and limp in contrast to old leaves. Light gray flecking and striping then appear at the base of the newest fully opened leaf, and, under severe deficiency, flecking and striping appears over the entire length of the leaf.

Copper

Initial symptoms are a general wilting of the plant at early tillering. Plants are light green in color. Withertip, a sudden dying and withering (curling) of the tip end of the blade, appears on young leaves and may extend up to half the length of the leaf. The base of the blade can remain green.

Zinc

Initial symptoms generally appear on middle-aged leaves that show a change in color from green to muddy gray-green in the central regions. These leaf regions appear drought-stressed, and necrotic areas soon develop and extend to leaf margins. Leaves may take on an oily appearance, and necrotic patches become larger and surrounded by mottled yellow-green areas.

NUTRIENT TOXICITIES

Aluminum

Aluminum toxicity may occur on soils with low pH. Retarded root growth is the most characteristic symptom. Plants also have reduced growth above ground and appear unthrifty with thinner than normal leaves. Yellowing occurs along the margin near the tip of the oldest leaf. Brown lesions form in chlorotic regions and work in from the margins, resulting in the formation of indentations. Old leaves become drought-stressed and withered and collapse in the center.

Boron

Initial symptoms, a mottled chlorosis just behind the tip of the oldest leaf and along the margin, are indistinguishable from those of phosphorus toxicity. Eventually, the chlorosis associated

with boron toxicity is less yellow. Mottled chlorotic areas have a dehydrated appearance, and the necrosis of the leaf tip leads down the margins in a fine necrotic edging. Chlorotic spots appear in from the margins and well down the leaf; necrotic areas form within the chlorotic spots and join together, giving much of the leaf a shriveled and dead appearance.

Phosphorus

The initial symptom of phosphorus toxicity is a mottled chlorosis just behind the tip of the oldest leaf and along the margin. The leaf tip becomes necrotic. The chlorosis progresses to a bright yellow along the margins, leaving a green arrow effect and increased necrosis at the tip. The base of the blade remains green.

Manganese

Symptoms appear first on oldest leaves and progress to younger leaves and include chlorosis with little necrosis, chlorosis progressing to necrosis, and in some cases, reddening combined with necrosis and chlorosis. Symptoms first appear on the oldest leaf tips and progress along the leaf with the leaf margins being more affected. A brown blotch or gray flecks of necrotic tissue can appear over the entire leaf.

OTHER COMMON ABIOTIC DISORDERS

Low Temperature and Frost Injury

When frost occurs from emergence through seedling development, cells at the growing point may be killed. This causes bands of yellow or tan on early leaves and can cause leaves to develop without chlorophyll (see figs 2 and 32). These leaves remain white while later leaves usually develop normally. Frost can also damage floral tissues. Frost injury to the developing head tissues can occur before heading, anytime after stem elongation (6- to 7-leaf stage) begins; this may occur as early as late January for early fall-sown small grains. The immature spike becomes increasingly vulnerable to frost and wind chill injury as it grows upward away from the protection of the densely tillered and leafy canopy at the soil surface. Injury generally is associated with minimum temperatures in the 29° to 35°F (-1.7° to 1.7°C) range. Often only one frost is involved, and the duration usually does not exceed a few hours.

Injury that occurs at flowering usually is nonuniform because flowering time can vary widely in a field due to topography, nutrition, and plant density differences. Flowering usually takes place over a three- to five-day period on an individual spike (or panicle in the case of oat), while flowering within a field may extend over a period of 15 to 20 days p due to non-uniform emergence and fertility. During flowering, when the pollen tube is extending, growth may be stopped by unfavorable temperature. At temperatures near 32°F (0°C) there is no killing of glumes, but the male flower parts can be sterilized.

Symptoms of frost injury are most apparent after heading (see fig. 31). The grain spikes may appear tan in color (somewhat like a mature, dry spike ready for harvest), but the grain will not develop into kernels; instead, spikes will be blank because flower development and pollination were disrupted. Spikes that have been frozen and then subjected to wind may shatter, leaving a spike that appears only as a stem (rachis). The risk of frost injury to fall-sown small grains in the Central Valley and surrounding areas can be reduced by delaying sowing until at least mid-November. Late-heading cultivars are recommended for early-fall sowing. Thickly planted stands are more vulnerable to frost damage than are thinner stands since thick stands are not as capable of generating new tillers as thinner stands.

Waterlogging

Small grain plants have a relatively low water demand during early stages of development. Excess water or waterlogging displaces soil oxygen, stunts plants, and turns leaves yellow or reddish. Symptoms usually occur first in low spots within a field where water can collect (see fig. 29).

Salt Damage and Salinity

Affected plants are stunted and dark blue green in color, with tip burn and firing of the leaf margins (see fig. 4). Since soil salinity is rarely uniform within a field, variability in crop growth is one of the first symptoms. Bare spots within a field are common. Wheat is less tolerant of salinity than is barley, but it is more tolerant than rice, field corn, or beans. However, wheat often yields higher under alkaline soil conditions (and the accompanying reduced soil permeability to water) because of its ability to withstand waterlogged conditions better than barley and because of its higher grain yield potential. Wheat yield is reduced when the electrical conductivity (EC) of the soil (saturated extract) exceeds a threshold of about 6.0 mmhos/cm. Yield reduction is proportional to salinity, with a yield decrease of about 3 to 5 percent for each 1 mmhos/cm (the measurement unit of salinity) increase in salinity above the threshold (Maas and Poss 1989).

Herbicide Injury

Contact herbicides

Leaf tissue damage can occur from herbicides used for weed control in cereals or from herbicides used in other crops that may drift long distances. Leaf burn from herbicides (see figs. 3, 5, and 6) is usually prompted by unfavorable environmental conditions such as freezing, long periods of fog, or dry, windy periods that increase susceptibility of the plant to injury. Symptoms on foliage begin with small spots or patches of brown to white bleached tissue. Affected areas eventually darken and dry up. Leaf damage also can occur from herbicide applications as a result of surfactants or fertilizer solutions in the spray mixture.

Phenoxy herbicides

The phenoxy herbicides MCPA and 2,4-D are mainstay herbicides used for broadleaf weed control in small grain production. Proper timing of application based on crop growth stage is critical for maintaining crop safety. If 2,4-D is applied too early (2- to 3-leaf stage) or too late (boot stage) it may cause abnormal kernel development or prevent spikes or awns from freely emerging from the leaf collar. It causes twisted spikes or elongated rachis at heading as well as kernel blanking (see figs. 34, 35, and 36). Dicamba, also a growth regulator herbicide, causes prostrate vegetative growth (fig. 33) and lodging when applied too late (tillering stage and later). It also causes distortion or twisting of grain spikes.

Soil residual herbicides

Soil residual (or soil active) herbicides are used on many crops that are grown prior to small grain crops. Some have a long soil half-life and remain at a high enough concentration in the soil to adversely affect the development of the small grain crop. The extent of injury and symptoms depend on herbicide type, the concentration of herbicide remaining in the soil, and, to some degree, on the variety or type of small grain crop: wheat, barley, and oat have different levels of herbicide susceptibility. Damage may be observable at germination and may inhibit seedling emergence, or damage may appear later when roots reach the herbicide zone, resulting in stunting, growth suppression, and foliage discoloration. Following dinitroanilin herbicides, seedlings germinate but develop short, stubby roots and stunted plants. Residues from acetolactate synthase (ALS) herbicides (rimsulfuron, imazamox) can kill germinating seedlings or cause stunting or yellowing.

DAMAGE BY DISEASES AND INSECT PESTS

Comprehensive descriptions of key disease and pest problems of small grains can be found in part 6, *Pest Management—Diseases*, and in part 7, *Pest Management—Insects*.



Figure 1. Wireworm damage to barley field. Photo by John Prato.



Figure 2. Leaf banding from frost damage. Photo by Jack Kelly Clark.



Figure 3. Leaf burn from herbicide injury. Photo by Ron Vargas.



Figure 4. Leaf tip burn from salinity. Photo by Steve Wright.

TROUBLESHOOTING GUIDE

Once you have a basic understanding of small grain development and can recognize normal plant growth at all stages and identify visual abnormalities, use the following troubleshooting guide to match up many common symptoms and problems with their causes. The guide is organized by plant parts affected, primary symptoms, and causes, and includes photographs of symptoms taken in affected fields.

Troubleshooting Guide for Small Grains

SEEDLINGS (CROP EMERGENCE)

Primary symptoms	Cause
Delayed emergence and nonuniform stand; yellow stems and leaves below soil surface; elongated subcrown.	Deep planting (below 2 in, or 5 cm) in heavy-textured soil; soil crusting.
Dead seedlings; roots of emerging plants are pruned, killing the seedlings and reducing the stand. As surviving plants mature, stems may be girdled, resulting in white heads.	Wireworms (fig. 1).

LEAVES

Primary symptoms	Cause
Leaf banding (narrow white stripes across leaf blade).	Freezing temperature as leaf emerges (fig. 2).
Leaf tip burn. Brown necrotic tissue beginning at leaf tip, usually ½ to 1 inch (12 to 25 mm) long.	Drying wind, moisture stress; contact herbicides; foliar fertilizer burn; soil salinity (figs. 3, 4).
White or tan speckled upper leaves, bleached circular areas to ¼ inch (6 mm) in size.	Herbicide drift (paraquat); or other herbicides/ adjuvants or foliar fertilizer sprays (figs. 5, 6).
Leaf surfaces skeletonized. Plants may senesce early and produce few tillers.	Cereal leaf beetle.
Light to intense yellowing of lower leaves or of entire plant.	Nitrogen deficiency; denitrification from saturated soil (figs. 7, 8).

(continued)



Figure 5. Paraquat spray drift injury. Photo by Jack Kelly Clark.



Figure 7. Nitrogen deficiency from denitrification. Photo by Steve Wright.



Figure 6. Shark herbicide injury. Photo by Ron Vargas.



Figure 8. Nitrogen deficiency. Photo by Steve Wright



Figure 9. Leaf yellowing of barley from barley yellows dwarf virus. Photo by Lee Jackson.



Figure 10. Leaf yellowing of wheat from barley yellows dwarf virus. Photo by Lee Jackson.



Figure 11. Leaf reddening of oat from barley yellows dwarf virus. Photo by Jack Kelly Clark.



Figure 12. Barley yellow dwarf virus infected area. Photo by Steve Wright.



Figure 13. Oat bird cherry aphid. Photo by Jack Kelly Clark.



Figure 14. Silvering of leaves due to mite feeding. Photo by Jack Kelly Clark.



Figure 16. "Hooked spike" from Russian wheat aphid. Photo by Jack Kelly Clark.

Primary symptoms	Cause
Lower leaves pale green; upper leaves yellow to white.	Sulfur deficiency.
Yellow, red, or purple leaves; uneven, blotchy leaf discoloration progressing from leaf tip to base and margin to midrib. Wheat and barley leaves usually become yellow, while oat leaves usually become red. Plants also can be stunted. Oat panicles can be blasted (florets become sterile). Aphids may be present.	Barley yellow dwarf virus (figs. 9, 10, 11, 12, 13).
Leaves first appear silvery and later take on a scorched appearance; leaves may appear yellowish and plants stunted, similar to cold temperature injury; webbing may be present.	Mites (fig. 14).
Leaves curled like a soda straw (tubelike); white, yellow, or purple vertical leaf stripes. Plants can be stunted and sometimes prostrate; awns may be trapped in the curled flag leaf, distorting the spike which assumes a fish-hook appearance; aphids may be found inside the curled leaves.	Russian wheat aphid (figs. 15, 16).
Irregularly shaped necrotic leaf spots, reddish-brown with gray-brown ashen centers and small black (pepper-size) specks (wheat).	Septoria tritici leaf blotch fungus (fig. 17).

(continued)



Figure 15. Leaf curling and streaking from Russian wheat aphid. Photo by Jack Kelly Clark.



Figure 17. Septoria tritici blotch lesions. Photo by Jack Kelly Clark.



Figure 18. Barley net blotch lesions. Photo by Jack Kelly Clark.



Figure 19. Early symptoms of leaf scald of barley. Photo by Jack Kelly Clark.



Figure 20. Late symptoms of leaf scald of barley. Photo by Jack Kelly Clark.



Figure 21. Wheat leaf rust pustules. Photo by Jack Kelly Clark.



Figure 22. Wheat leaf rust uredial and telial pustules. Photo by Jack Kelly Clark.

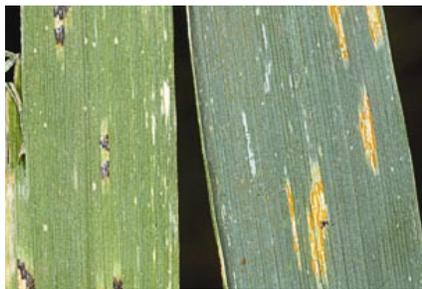


Figure 23. Oat crown rust pustules. Photo by Jack Kelly Clark.



Figure 24. Wheat stripe rust symptoms. Photo by Jack Kelly Clark.



Figure 26. Patches of powdery mildew. Photo by Jack Kelly Clark.

Primary symptoms	Cause
Small brown spots to narrow brown blotches with a netted or cross-hatched appearance; surrounding tissue becomes yellow. Lesions can expand, spread over the entire leaf (barley).	Barley net blotch fungus (fig. 18).
Irregularly shaped to oval leaf spots with bluish gray centers and dark brown margins; spots can coalesce, giving the appearance of rapid scalding (barley).	Barley scald fungus (figs. 19, 20).
Yellow-brown (barley) to reddish-orange (wheat) pustules on leaves and sheaths. Pustules on barley are small and round; pustules on wheat are scattered or clustered on upper leaf surface. As the plants mature, the pustules turn dark and shiny as teliospores are formed.	Leaf rust fungi (figs. 21, 22).
Oblong, orange-colored pustules primarily on leaves and sheaths (oat). As the plant matures, the pustules turn dark and shiny as teliospores are formed.	Oat crown rust fungus (fig. 23).
Yellow-orange pustules form conspicuous stripes primarily on leaves (wheat and barley). As plant matures, the pustules turn dark and shiny as teliospores are formed.	Stripe rust fungi (figs. 24, 25).
Patches of white cottony growth (mycelium and spores) develop opposite chlorotic spots on leaf surfaces. These patches later turn dull gray-brown. Small dark-brown structures (cleistothecia) develop among the cottony patches.	Powdery mildew fungus (figs. 26, 27).



Figure 25. Stripe rust symptoms on wheat kernels. Photo by Milton D. Miller.



Figure 27. Patches of powdery mildew with cleistothecia. Photo by Jack Kelly Clark.



Figure 28. Plants dying due to lagoon water. Photo by Steve Wright.



Figure 29. Plant death due to waterlogged soil. Photo by Steve Wright.

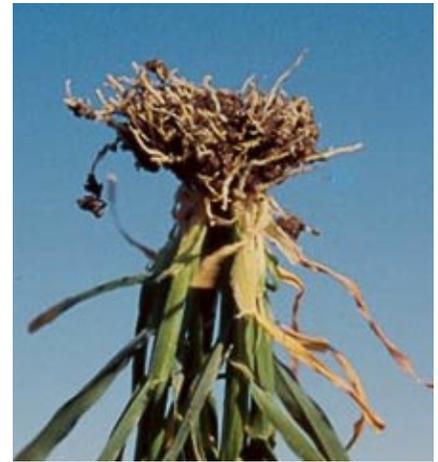


Figure 30. Stunted wheat roots from compacted soil. Photo by Ron Vargas.

WHOLE PLANT

Primary symptoms	Cause
Areas of dying, yellow to reddish brown plants usually at ends of fields or in low spots in field.	Saturated soils; lagoon water with high organic matter content (figs. 28, 29).
Stunted plants, poor tiller development; leaves purple; delayed heading.	Cold soils; low phosphorus availability.
Uneven, shallow, horizontal root growth; stunted plants in rows or strips; visible traffic patterns.	Compacted soil (fig. 30).
Leaves appear water soaked and dark green; bleached white spikelets appear later. Absence of kernels at different positions in the spike; injury may vary through field.	Freezing temperatures during heading, at pollination, or during grain fill (figs. 31, 32).
Prostrate growth, sharp bends at stem nodes; kinked heads and curled awns.	Dicamba herbicide injury (fig. 33).



Figure 31. Freezing injury to wheat spike. Photo by Jack Kelly Clark.

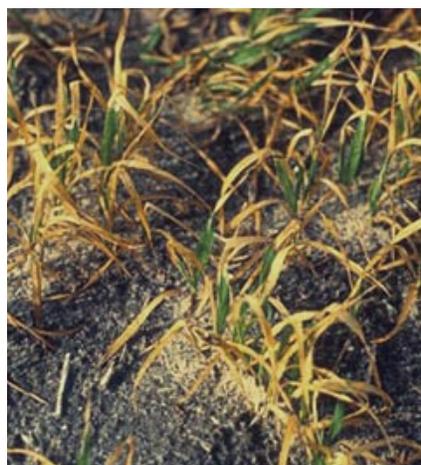


Figure 32. Frost injury to barley seedlings. Photo by Ron Vargas



Figure 33. Prostrate growth from Dicamba herbicide injury. Photo by Mick Canevari.



Figure 34. Distorted spikes from phenoxy herbicide injury. Photo by Jack Kelly Clark.



Figure 35. Distorted spike and kinked leaf from phenoxy herbicide injury. Photo by Mick Canevari.



Figure 36. Leaf twisting from phenoxy herbicide injury. Photo by Mick Canevari.

HEAD TISSUES AND/OR SEEDS

Primary symptoms	Cause
Bulging, malformed spikes and curled awns; nonuniform grain symmetry; leaf twisting.	Misapplication (timing or rate) of phenoxy herbicides; frost injury (figs. 34, 35, 36).
Blanking and bleaching of spikes (white heads). Lower leaf sheaths, culms, crowns, subcrown internodes, and roots are discolored (brown or black) and rotted.	Root and foot rot fungi (figs. 37, 38, 39).

(continued)



Figure 37. White heads caused by common root rot. Photo by Jack Kelly Clark.



Figure 38. Root rot of wheat. Photo by Milton D. Miller.



Figure 39. Browning of wheat crown and roots. Photo by Lee Jackson.



Figure 40. Blackening of stems caused by take-all. Photo by Jack Kelly Clark.



Figure 41. Wheat stem maggot inside stem. Photo by Jack Kelly Clark.

Primary symptoms	Cause
Blanking and bleaching of spikes (white heads); blackening of roots, crown, and under lower leaf sheaths. The presence of a layer of dark brown or black fungal mycelium underneath the lower leaf sheaths distinguishes take-all from common root rot.	Take-all root rot fungus (fig. 40).
Blanking and bleaching of spikes (white heads). The spike turns white and is easily pulled free from the stem where it has been chewed through; legless maggots may be found inside the stem.	Wheat stem maggot (fig. 41).
Normal spike tissue is replaced by olive-black masses of spores enclosed in fragile membranes that rupture near flowering time, releasing the spores and leaving only a bare rachis at maturity (barley, wheat).	Loose smut fungi (fig. 42).
Kernels are replaced by round, seedlike bodies that contain masses of dark spores. Glumes on infected spikes spread apart. The spore masses have a distinctive odor, similar to that of decaying fish, when they are crushed.	Covered smut (barley, oat) and stinking smut or bunt (wheat) fungi (fig. 43).



Figure 42. Loose smut of wheat, spike infection. Photo by Jack Kelly Clark.



Figure 43. Covered smut of barley. Photo by Jack Kelly Clark.

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