Seed Treatments for Small Grain Cereals

R. Smiley, R.J. Cook, and T. Paulitz

Seed treatment is an important component of integrated pest management (IPM) systems. Seed treatment is acceptable environmentally and economically for reducing damage from insects and diseases, and for promoting uniform stand establishment and seedling vigor.

This publication explains why seed treatment is important, what kinds of treatment compounds are available, and how they work.

What is a seed treatment?

A seed treatment includes any compound or process applied to seed in order to reduce damage by organisms that infest the seed surface, infect tissue inside the seed, live in soil where the seed will be placed, or attack young seedlings.

Compounds discussed in this publication include organic chemicals and microbial formulations that are applied to suppress damage by pathogenic fungi, insects, or parasitic nematodes. Other biological and chemical products enhance seedling vigor in the following ways:

• Provide plant nutrition (e.g., Seed Life®)
• Encourage growth of native organisms that kill fungi (e.g., YEA!®)
• Protect seedlings from herbicides (e.g., activated charcoal)
• Supply symbiotic nitrogen-fixing bacteria (e.g., inoculants for legumes)

Processes can be chemical and physical. They include treatment of seed with surface sterilizing substances (e.g., hot water or Chlorox®) or sanitizing energy in the form of microwaves, magnetic energy, or electron bombardment (e.g., E-Dressing®).

History of seed treatment

Since the mid-1700s, smut diseases have been a primary target for seed sanitizing procedures. Fifty years before it was proved that common bunt was caused by a living organism, it was discovered that damage could be reduced by treating wheat seed with solutions of sea salt, saltpeter, and lime. More recent treatments included hot water and application of compounds containing formaldehyde, copper, or mercury. These treatments killed smut spores on the seed surface, but most did not protect against smut fungi that live in soil or inside the seed.
Prior to the mid-1950s, common bunt caused severe damage in the Pacific Northwest. Until that time, common bunt could not be controlled adequately by genetic resistance or fungicides. Common bunt was brought under sustained control with the development of hexachlorobenzene, which controls soil-borne as well as seed-borne pathogens.

The next major advance occurred during the late 1960s, when carboxin was registered to control flag smut, loose smut, and seed-borne inocula of common bunt. Carboxin became a major component of a highly efficient integrated smut management strategy that combined genetic resistance and chemical treatment. This dual-control strategy stabilized the Pacific Northwest wheat industry so successfully that it led to a reduction in emphasis on breeding wheat with resistance to these pathogens.

Up to 95 percent of the winter wheat seed planted in the Pacific Northwest was treated with a single smut-control fungicide (carboxin) for more than 20 years. Near total reliance on one fungicide and increasing releases of susceptible varieties caused concern during the 1980s. Smut fungi readily develop resistance to a fungicide if it is used too frequently. Resistance has occurred in several regions, but apparently not in the Pacific Northwest.

In 1990, difenoconazole was registered as a triazole fungicide effective for controlling soil-borne inocula for common bunt and dwarf bunt in addition to most diseases already controlled by carboxin. This was an important advance in smut-control technology.

**Smut control today**

As was true in Europe 250 years ago, the most important reason to treat wheat and barley seed in the Pacific Northwest today is to control smut diseases. Growers rely on fungicide seed treatments more heavily now than at any other time in the history of the small grains industry in the Pacific Northwest. Smut control practices in some regions now depend almost entirely on chemical seed treatments, because many profitable varieties are susceptible to one or more of the smut pathogens.

During the 1999–2000 crop year, winter wheat varieties susceptible to common bunt were planted on 82 percent and 26 percent of the acreage in Oregon and Washington, respectively. Ninety-six percent of the acreage in these states was planted to varieties susceptible to flag smut. Most spring wheat and barley varieties are susceptible to at least one smut disease. Smut control procedures in some areas now depend almost entirely on chemical seed treatment.

Common bunt, dwarf bunt, flag smut, and loose smut can be observed most years in a few plants and locations. But, economic damage occurs only in rare instances where untreated seed of susceptible varieties is planted during several successive years in an individual field.

Smut cannot be controlled by application of fungicides to foliage or soil. Therefore, wheat and barley seed in the Pacific Northwest must be treated with a smut-control fungicide. Fungicides available in the Pacific Northwest include carboxin, difenoconazole, tebuconazole, triadimenol, thiabendazole, and quintozene.

Seed treatment technology also has developed strategies to reduce damage by fungi that cause seed decay, seedling damping-off, and root rot, and damage by insects and nematodes. Mixtures of chemical treatments are now common and important. They generally include a smut-control fungicide mixed with another fungicide or insecticide to expand the spectrum of protection. These other fungicides include thiram, captan, metalaxyl, mefenoxam, fludioxonil, and imazalil.

Insecticides applied as seed treatments to small grain cereals to reduce damage from wireworms, aphids, and barley yellow dwarf virus include imidacloprid (Gaucho®) and thiamethoxam (Cruiser®). Benzene hexachloride (Lindane®) is an older insecticide that reduces damage from wireworms.

Biological fungicides are being developed. None are used widely on cereals in the Pacific Northwest at this time.
Chemical families of fungicides

Minor differences in the molecular structure of closely related chemicals can have a major impact on the performance and characteristics of plant protection products. Though each product is distinct, it is important to recognize how “families” of chemicals are related, particularly if a fungus or insect develops resistance to a previously effective pesticide.

When an organism develops resistance to a specific chemical, it is common for that resistance to extend to other products in the same chemical family. Table 1 lists families of closely related fungicides that are applied as seed treatments to small grains. Note that metalaxyl and mefenoxam belong to the same chemical family. If a fungus develops resistance to metalaxyl, it is likely to have resistance also to mefenoxam. Another closely related family of chemicals is the demethylation-inhibiting triazole fungicides, including difenoconazole, tebuconazole, and triadimenol.

A new class of protein compounds act as fungicides by inducing systemic activated resistance (SAR). Chemicals having SAR activity are not toxic to fungi but induce plants to activate internal defense mechanisms throughout the plant. This results in chemically induced plant resistance.

<table>
<thead>
<tr>
<th>Chemical family</th>
<th>Chemical name</th>
<th>Representative trade names</th>
</tr>
</thead>
<tbody>
<tr>
<td>Benzimidazole</td>
<td>thiabendazole</td>
<td>Mertect 340, TBZ, Agrosol</td>
</tr>
<tr>
<td>Phenylpyrrole</td>
<td>fludioxonil</td>
<td>Maxim</td>
</tr>
<tr>
<td>Phenylamide</td>
<td>metalaxyl</td>
<td>Apron, Allegiance</td>
</tr>
<tr>
<td></td>
<td>mefenoxam</td>
<td>Apron XL</td>
</tr>
<tr>
<td>Demethylation-inhibiting:</td>
<td>Imidazole</td>
<td></td>
</tr>
<tr>
<td></td>
<td>imazalil</td>
<td>Flo-Pro, Nu-Zone</td>
</tr>
<tr>
<td></td>
<td>difenoconazole</td>
<td>Dividend</td>
</tr>
<tr>
<td></td>
<td>tebuconazole</td>
<td>Raxil</td>
</tr>
<tr>
<td></td>
<td>triadimenol</td>
<td>Baytan</td>
</tr>
<tr>
<td></td>
<td>flutriafol*</td>
<td>Vincit*</td>
</tr>
<tr>
<td></td>
<td>hexaconazole*</td>
<td>ProSeed*</td>
</tr>
<tr>
<td></td>
<td>triticonazole*</td>
<td>Charter*, Premis*, Real*</td>
</tr>
<tr>
<td>Carboxamide</td>
<td>silthiophan*</td>
<td>Latitude*</td>
</tr>
<tr>
<td>EBDC-like</td>
<td>thiram</td>
<td>Thiram</td>
</tr>
<tr>
<td>Aromatic</td>
<td>quintozene</td>
<td>Pentachloronitrobenzene, PCNB</td>
</tr>
<tr>
<td>Phthalimide</td>
<td>captan</td>
<td>Captan</td>
</tr>
<tr>
<td>Oxathiiin</td>
<td>carboxin</td>
<td>Vitavax</td>
</tr>
<tr>
<td>Protein</td>
<td>harpin</td>
<td>Messenger</td>
</tr>
</tbody>
</table>

* Indicates active ingredients and products that are not registered for cereals in the U.S.

Always follow the guidelines and precautions on the product label to assure personal and crop safety when handling these products.
Biofungicides

Beneficial microorganisms that are intended to replace or complement chemical seed treatments are being developed. Biological treatments consist of living organisms selected to protect plants from infection while multiplying and spreading in the seed-zone and root-zone. Beneficial bacteria and fungi can reduce plant stress in the following ways:

- Compete with pathogens for nutrients at the root surface
- Produce toxins that inhibit growth or reproduction of the pathogens and pests
- Produce biologically stimulatory compounds that are absorbed by roots
- Parasitize fungal pathogens, insects, or nematodes

Table 2 lists existing biological seed treatments for small grains. More biological products are being developed for small grain cereals.

Biofungicides can be applied as supplements to chemical smut-control fungicides, because current biologicals do not protect adequately against smut pathogens. Biological treatments have the potential to persist longer in an active form than current chemical treatments.

Biological treatments show potential for moving down roots to control root diseases, insects, and nematodes. While this is of tremendous interest, it is difficult to achieve in practice, because of the myriad factors that influence microbial growth and functional efficiency in soil. As with chemical treatments targeted for controlling root diseases, the performance of biological treatments has been inconsistent thus far. However, promising formulations continue to be developed.

Zones of protection

When a seed is placed into moist soil, it absorbs moisture, and cells begin to respire actively. Pathogens in, on, or near the seed also are stimulated into active growth.

Some seed treatment chemicals protect by killing fungal spores on the seed surface and in soil a short distance from the seed surface (surface protectants). Other seed treatment chemicals are absorbed into the internal seed tissue and, depending on their chemistry, either are restricted to movement only within the seed tissue (local systemics) or are mobilized to move upward into

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**Table 2. Biological seed treatments for small grains.**

<table>
<thead>
<tr>
<th>Microbe type</th>
<th>Genus and species</th>
<th>Common trade names</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bacteria</td>
<td><em>Bacillus subtilis</em></td>
<td>Kodiak, System 3, Subtilex</td>
</tr>
<tr>
<td></td>
<td><em>Burkholderia cepacia</em> (= <em>Pseudomonas cepacia</em>)</td>
<td>Deny, Precept, Intercept</td>
</tr>
<tr>
<td></td>
<td><em>Pseudomonas chlororaphis</em></td>
<td>Cedoman</td>
</tr>
<tr>
<td></td>
<td><em>Streptomyces griseoviridis</em></td>
<td>Mycostop</td>
</tr>
<tr>
<td></td>
<td><em>Pseudomonas species</em></td>
<td>Several are being developed, but none are available at this time.</td>
</tr>
<tr>
<td></td>
<td>Mixtures of species</td>
<td>SC27; perhaps other products without specified ingredients</td>
</tr>
<tr>
<td>Fungi</td>
<td><em>Paecilomyces lilacinus</em></td>
<td>Paecil, Bioact (both are nematicides)</td>
</tr>
<tr>
<td></td>
<td><em>Trichoderma harzianum</em></td>
<td>T-22G, T-22 Planter Box, Bio-Trek</td>
</tr>
</tbody>
</table>
Figure 1. Zones of protection from chemical seed treatments.

the coleoptile and leaves (systemically translocated compounds). Thus, chemical seed treatments are categorized according to their method for fighting diseases. See Figure 1 for an illustration.

**Surface protectants**

Surface protectant fungicides kill a broad spectrum of fungi that infest the seed surface and cause seed decay, seedling damping-off, and seedling blight. These fungicides also diffuse a short distance into soil around the seed and extend the range of protective activity against soil fungi that attack the seed and first roots.

Surface protectants are applied to cereals in a mixture with a smut-control fungicide.

Surface protectants include the fungicides captan, thiram, and fludioxonil, and the insecticide benzene hexachloride. Captan and thiram are older fungicides. Fludioxonil is a new fungicide applied at lower rates than the older compounds.

**Local systemics**

Local systemic fungicides are absorbed to a limited extent into the seed tissue, but they serve mostly as surface protectants.

Local systemic fungicides include quintozene and imazalil. Quintozene is an older chemical that controls common bunt of wheat, covered smut of barley and oats, and early infections by fungi that cause Rhizoctonia root rot. Imazalil is a newer fungicide that reduces damage from common root rot and barley leaf stripe.

**Systemically translocated compounds**

Systemically translocated compounds have curative as well as surface protectant abilities. They are absorbed into seed and plant tissues and can kill (fungicides) or immobilize (fungistats) fungal pathogens that have already invaded the seed. Some are translocated into the coleoptile to protect against pathogens that attack young plants.
Nearly all systemic movement of these fungicides is upward in the plant xylem. So, some of these compounds can suppress early damage from foliar diseases like stripe rust, powdery mildew, and Septoria leaf blotch. None efficiently translocates downward in the phloem. Thus, all are inefficient for protecting roots.

While all systemic compounds are called fungicides here, some are true fungicides that kill fungi but others are fungistats that prevent fungal growth without killing the pathogen. Pathogens held in check by fungistats can start growing again after the chemical concentration in plant tissue dips below the minimum required to restrain fungal growth.

New compounds that induce systemic activated resistance have the potential to affect the entire plant, including the roots. Further testing is underway to determine if this technology can improve control of root diseases.

**The chemicals and their activity**

Systemically translocated fungicides include metalaxyl, mefenoxam, thiabendazole, carboxin, and the triazole family of demethylation inhibitors.

Metalaxyl and mefenoxam have a narrow but important spectrum of toxicity limited to fungi that cause Pythium damping-off and root rot in cereals.

Thiabendazole protects against common bunt of wheat and reduces pressure from fungi that cause seed decay, seedling damping-off, and Fusarium foot rot.

Carboxin controls common bunt, loose smut and flag smut of wheat, and loose and covered smuts of oats and barley.

The triazole fungicides are active against a wider spectrum of pathogenic fungi and are applied at a fraction of the rates required for thiabendazole and carboxin. Triadimenol, difenoconazole, and tebuconazole protect against the list of diseases shown for carboxin. They also are active against many fungi that cause foliar diseases of seedlings, including powdery mildew, net blotch, and stripe, crown, and leaf rust.

Difenoconazole, tebuconazole, and triadimenol can suppress early infections by the root- and crown-infecting fungi that cause take-all, Rhizoctonia root rot, common root rot, and Fusarium foot rot. Difenoconazole also has the unique ability to control dwarf bunt (TCK smut).

Other triazole fungicides are registered in Canada or overseas but not in the U.S. These include hexaconazole, triticonazole, and silthiophan. The latter was tested as MON 65500 and is exceptionally effective for controlling take-all.

Systemically translocated insecticides include imidacloprid and thiamethoxam. They are especially active against wireworms and aphids that colonize seedlings.

**Concentration and duration**

**Concentration**

All pesticides have a dose:response relationship. A minimum concentration is required to achieve a specific level of protection. The effective concentration for seed treatments diminishes over time as the active ingredient decomposes in soil, dilutes as the chemical diffuses or leaches away from the seed surface, dilutes in plant tissue as the volume of tissue increases, or is otherwise broken down in the plant.

**Duration**

Infection sites for smut pathogens occur at or above the seed. Some smut pathogens are carried inside the seed and can grow upward at nearly the same rate as the emerging coleoptile. Smut pathogens carried on the seed surface or residing as spores in soil have infective periods that coincide with coleoptile emergence, which can be as few as 7 days. The longest infective period for a smut pathogen (dwarf bunt) occurs over 2 to 3 months on near-dormant, snow-covered winter wheat. Because of relatively short infection time and closely grouped infection sites for most smut pathogens, the smut diseases can be controlled effectively by seed treatment fungicides.
In contrast, most root-infecting pathogens can infect young roots that grow outside the zone of protection made by contact protectants or compounds translocated upward in the xylem. Many root pathogens can infect young roots during most of the growing season. For autumn-sown cereals in the Pacific Northwest, the infective period for root pathogens can be as long as 9 months. Typically, most damage occurs from infection in the autumn, but damage to roots can continue during the spring (Figure 2). Chemical fungicides don't persist long enough to be efficient through the extended infective interval on winter cereals.

Spring cereals produce most of their growth over a 3- to 4-month period. Seed treatment concentrations stay comparatively higher through a longer phase of the growth cycle for cereals planted in the spring than those planted during the autumn. Thus, pest management and yield responses are more common on spring cereals than winter cereals.

Limitations of seed treatments

Seed treatments cannot fully offset effects from inferior seed lots or poor planting conditions. They do not reverse poor germination due to mechanical damage or seed stored too long or under adverse conditions.

No seed treatment can control all diseases or insects. The level of protection diminishes over a relatively short time period. Treatments are most effective for controlling smut diseases and for protecting seed planted under conditions that delay germination and seedling establishment, such as hot, cold, dry, or wet soil, or high-residue systems.

Adverse responses to chemical seed treatments

Surface protectants have the lowest incidence of adverse effects, because they are not absorbed into the seed. Local systemics and systemically translocated pesticides are absorbed into the seed. They directly can influence plant physiological processes essential for seed germination and seedling growth.

Some of these pesticides are effective plant growth regulators if they are applied to seed at rates higher than listed on the product label. An entire seed lot can receive an amount of compound higher than the label rate if it is treated uniformly in excess, or if two systemically translocated fungicides are both applied as a mixture. Individual seeds can be treated in excess if the fungicide is not mixed uniformly through the seed lot. Excess pesticide may disrupt geotropism (shoots don't grow up and roots don't grow

<table>
<thead>
<tr>
<th>Winter Cereals: 11 months</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sep</strong></td>
</tr>
<tr>
<td>planting</td>
</tr>
<tr>
<td>harvest</td>
</tr>
<tr>
<td>infective interval for one or more pathogens</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Spring Cereals: 6 months</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sep</strong></td>
</tr>
<tr>
<td>planting</td>
</tr>
<tr>
<td>harvest</td>
</tr>
<tr>
<td>infective interval</td>
</tr>
</tbody>
</table>

*Figure 2. Generalized production cycle for small grain cereals in the PNW.*
down), reduce the rate of coleoptile growth (slow or reduced emergence), or distort foliar tissue (thickened leaves).

The occurrence and extent of these abnormalities are influenced by complex interactions among the rate of chemical applied, quality of seed, planting depth, and soil environment. Adverse effects for some of these fungicides are most apparent when seed germination is slowed by planting into soil that is hot, cold, dry, or wet. Seed treatment also can have a negative influence on plant growth when seed is old, has mechanical damage, or has been stored for prolonged periods, especially if it was not dried thoroughly.

“Disease trading”

A compound effective against one pathogen may control that pathogen, but it also can create a biological void that favors more aggressive infection by another pathogen that is insensitive to that chemical. This phenomenon is called “disease trading.” For example, if seed is treated only with PCNB, the treatment can reduce infections by pathogens that cause smuts and Rhizoctonia root rot. However, under certain conditions, the treatment also may encourage more root- and crown-rot by Pythium and Fusarium. Damage from the latter pathogens can be more severe than if seed had not been treated, or if a broad spectrum fungicide had been applied.

Seed treatment is an important component of integrated pest management programs for producing small grain cereals in the Pacific Northwest. Protection against a broad spectrum of organisms is required. It is common for mixtures of several fungicides and an insecticide to be applied to small grain cereals in our region. One must know the treatments available, why each is important, and how they protect different parts of plants growing from treated seed.

For more information

OSU Extension publications

*Dwarf Bunt of Winter Wheat in the Northwest*, PNW 489 (reprinted 1996). $1.00

*Pacific Northwest Insect Management Handbook* (2002). $35.00

*Pacific Northwest Plant Disease Management Handbook* (2002). $35.00

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