Andersons TGR (Turf Growth Regulator) and Turf Enhancer formulations are effective tools for reducing existing Poa annua in turf. Paclobutrazol, the active ingredient in TGR and Turf Enhancer, is a root-absorbed gibberillic acid inhibitor. Since paclobutrazol is a growth regulator, you will see a differential growth regulating effect on Poa annua in the desired turf species. Poa annua is particularly sensitive to paclobutrazol. When using TGR or Turf Enhancer products in a program, Poa annua is weakened, and you will see increased density and tillering in the desired turf, which allows it to gradually replace Poa annua.

Before beginning a Poa annua reduction program, it is helpful to understand the amount of active ingredient that you are applying. This will vary with the product used. Any of Andersons' TGR and Turf Enhancer formulations can be used to successfully reduce and suppress Poa annua.

Since nitrogen is necessary for enhancing the effect of paclobutrazol, Andersons' granular TGR and Turf Enhancer formulations are excellent choices. All of our products are SGN 100 particle sizing and can be used on all turf areas, including bentgrass greens.

**Beginning a Poa annua reduction program**

- It is best to start a program either in the fall or spring when turf is healthy and actively growing. Success is achieved only by continuing on a TGR/Turf Enhancer program until the Poa population is reduced to the desired level.
- TGR and Turf Enhancer application timings and rates vary with the type of turf being grown and the climate it is grown in. Your Andersons' territory manager can provide specific information on the rates and formulations which best suit your area.

**What to expect**

- Poa will begin to turn yellow about two weeks after the first application. Cooler spring temperatures can delay this somewhat. The degree of discoloration of the Poa will vary with the rate of active ingredient applied. Below are two examples of the degree of discoloration of Poa after an initial application.

![](https://example.com/14-0-28-plus-Turf-Enhancer.png) **14-0-28 plus Turf Enhancer**

- .2 ounces of paclobutrazol per acre

![](https://example.com/31-3-7-plus-TGR.png) **31-3-7 plus TGR**

- .5 ounces of paclobutrazol per acre

- Under regulation, the desired turf species will begin to fill in areas of weakened Poa annua. You can enhance this effect by overseeding 10 to 14 days after an application of TGR or Turf Enhancer.
- The length of time that Poa is regulated will depend on the rate of active ingredient that is applied. Generally you will see about three to four weeks regulation at lower rates and eight to 12 weeks at higher rates.
- For additional information on Andersons TGR and Turf Enhancer products, contact your local Andersons distributor or Andersons Territory Manager.


For more information, visit our Web site: [www.andersonsgolfproducts.com](http://www.andersonsgolfproducts.com) or call 800-225-2639.
Treatments were also applied in 2002. However, disease development was slight and no treatments were significantly better than the non-treated control in spring 2003.

Treatments were applied with a CO$_2$-pressurized (40 psi) boom sprayer with XR11004 VS flat-fan TeeJet nozzles using a 52-gallon per acre water carrier rate. Individual plots were 5 feet by 10 feet in a randomized complete-block experimental design with four replications.

Continuous winter snow cover is common at McCall. During both winters, snow cover began late October to mid-November and remained on the plots 132 and 167 days in 2000-2001 and 2001-2002, respectively. Plots were rated for disease in late April 2001 (90 percent gray snow mold, 10 percent pink snow mold) and early May 2002 (essentially 100 percent gray snow mold, 95 percent $T.$ ishikariensis and 5 percent $T.$ incarnata) and turfgrass quality (quality rated 1-9; 9 = excellent turf quality).

Disease infection was severe (nontreated control 94 percent) at McCall during the winter 2000-2001 (Table 3). Several treatments had less disease than the nontreated control. Although not statistically different from several other treatments, numerically the best disease control (10 percent disease) was given by Medallion + Banner MAXX + Daconil Ultrex. Also, the lack of disease control (68 percent disease) when PCNB (Terraclor 75WP) was used alone indicated possible pathogen resistance had developed (Table 3).

In April 2001, several treatments had turfgrass quality better than the nontreated control (Table 3). As with disease control, numerically the best spring turfgrass quality was observed with Medallion + Banner MAXX + Daconil Ultrex and Heritage + Banner MAXX + Daconil Ultrex.

Disease infection also was severe (nontreated control 95 percent) at McCall during the winter 2001-2002 (Table 4). Although not statistically different from several other treatments, numerically the best disease control (less than 7 percent disease) was given by Medallion + Banner MAXX + Daconil Ultrex and Heritage + Banner MAXX + Daconil Ultrex. As in 2001, the lack of disease control (78 percent and 92 percent disease) when PCNB (Turficide 400 and FFII with 14-3-3, respectively) was used alone indicated that possible pathogen resistance had developed (Table 4).

Several treatments had turfgrass quality better than the nontreated control. As with disease control, numerically the best spring turfgrass quality was observed in plots treated with Medallion + Banner MAXX + Daconil Ultrex and Heritage + Banner MAXX + Daconil Ultrex.

**Correct pathogen identification**

Proper pathogen identification is always important prior to making any chemical application. This is especially true when managing the snow mold complex of pathogens in the Intermountain Northwest. It is not uncommon to observe $M.$ nivale, $T.$ incarnata, and $T.$ ishikariensis alone or together on golf greens, which typically depends on the severity and length of snow cover.

Vargas (1994) noted that Trizole fungicides are often effective against $T.$ incarnata but not $T.$ ishikariensis. Yet, Trizole fungicides are sometimes listed as controlling Typhula blight (Christians, 1998).

An interesting observation was that regardless of the dominant pathogen, combinations of products are needed to control snow mold in areas with prolonged snow. This was evident at McCall in 2001-2002 (Table 4). Visual observation in the spring 2002 indicated that the dominant pathogen was $T.$ ishikariensis (95 percent) with minor $T.$ incarnata (5 percent).

As expected, Fungicide V (chloroneb), which is effective against $T.$ incarnata performed poorly (65 percent disease). However, Medallion 50WP (fludioxonil), which in
GRAY SNOW MOLD TYPES

Caused by the pathogen *Typhula incarnata* Lasch ex Fr.
- In the fall, pink upright fruiting bodies.
- Generally associated with snow cover.
- Patches 6 inches to 2 feet in diameter; most 6 inches to 12 inches in diameter.
- Following snow melt, variable patches of gray-white matted turf.
- Sclerotia are large, up to 5 millimeters in diameter, and reddish brown.
- Generally less destructive that *T. ishikariensis*.

Caused by the pathogen *Typhula ishikariensis* Imai
- In the fall, silvery/white, very small fruiting bodies may be present.
- Generally associated with deep prolonged snow cover, often greater than 100 days.
- Following snow melt, bleached patches generally 6 inches to 12 inches in diameter.
- Sclerotia are small, less that 2 millimeters in diameter, and black.
- Generally causes more turf injury than *T. incarnata*.
- Most sever injury occurs with snow over unfrozen soil.

Our testing appeared quite effective against *T. ishikariensis*, gave equally poor control. These results indicate that snow mold can cause considerable turfgrass injury if *T. incarnata* is present even when *T. ishikariensis* is the dominant pathogen and is controlled, at initially low disease pressure or in the absence of *T. ishikariensis*.

Finally, the possible resistance of gray snow mold to PCNB noted at McCall needs to be verified. Unfortunately for research (but fortunate for superintendents), these observations could not be confirmed because of the unusually low development of snow mold disease throughout the Northwest during the winter of 2002-2003. Additional testing during 2003-2004 may provide much needed information for the management of snow mold in the Intermountain Northwest.

Conclusions
Snow mold pathogens must be correctly identified for effective control. During moderate winters, more fungicide options are available to control snow mold. Try combining one new with one old chemistry fungicide.

PCNB resistance may have developed with repeat applications under conditions where severe winters are the norm. In these regions, fungicide combinations of two and possibly three fungicides are a must. Efficacious fungicides that can be rotated with PCNB to mitigate the potential for the development of pathogen resistance are available for use.

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www.turfgrasstrends.com
Ultradwarf Bermudagrasses Exhibit Easy Mutation Tendencies

By Patrick McCullough, Bert McCarty, Vance Baird, Haibo Li and Ted Whitwell

Part 2: Do certain pre-emergent herbicides exacerbate this problem?

In Part 1 of this series, the natural genetic instabilities of dwarf-type bermudagrasses were discussed. Golf courses renovating to the new and improved ultradwarf bermudagrass cultivars hope to avoid previous problems of off-types developing five to 10 years after planting. Obviously, one would want to know management practices that could advance or, more importantly, prevent off-type contaminations.

Because DNA replication and chromosome separation are fundamental parts of mitosis, cell-division-inhibiting herbicides and plant growth regulators (PGRs) have the potential to disrupt genetic replication and even damage DNA sequences, exacerbating bermudagrass genetic instabilities and resulting in off-type mutations.

Currently, no herbicides or PGRs are labeled for use on the new ultradwarf bermudagrass cultivars and concerns exist for incorporating these compounds into management programs.

Turf discoloration and negative rooting responses from herbicides and PGRs may limit their potential for routine ultradwarf bermudagrass maintenance.

Herbicides may cause bermudagrass mutations

Pre-emergent herbicides for summer annual weeds are applied before soil temperatures become favorable for seed germination 54 degrees Fahrenheit (F) to 64 degrees F during spring months. Consequently, timing of spring pre-emergent applications coincide with bermudagrass root regeneration and emergence from winter dormancy.

Safe application of pre-emergent herbicides on dwarf bermudagrass is critical since roots are most vulnerable to herbicide injury during maximum root regeneration in the spring (Engel and Ilnicki, 1969). Additionally, pre-emergent herbicides concentrated in the surface layer of the soil could expose dwarf bermudagrass roots to initial and residual herbicide effects during critical root-growth initiation.

Pre-emergent herbicides affecting mitosis and cell division are commonly used on bermudagrass golf courses.

At one time, approximately one-quarter of all herbicides marketed affected mitosis as a primary mechanism of action (Vaughn and Lehnen, 1991). These herbicides, including dinitroanilines and pyridines, disrupt the nuclear division sequence by interfering with microtubules responsible for chromosome mobilization during mitosis (Ross and Lembi, 1999). Herbicides that prevent mitotic spindle formation by slowing or preventing the assembly of microtubules stunt new cell production, causing the replicated chromosomes to remain unseparated and eventually become enclosed by a nuclear envelope, leaving a polyploidy cell (Ross and Lembi, 1999). This mechanism of action is effective for pre-emergent control of germinating weed seedlings.

However, with the sensitivity of ultradwarf bermudagrasses to herbicides, concerns exist with root-growth inhibition, turf injury and potential genetic alterations from cell division interference.

The dinitroaniline herbicides, oryzalin and
pendamethalin, were recently studied by Goatley et al. (2003) on Champion, TifEagle, Floradwarf, MS-Supreme, Tifdwarf and Tifgreen bermudagrasses. Dinitroanilines are commonly used as pre-emergent herbicides in golf course management for annual grassy weed control. The principle effects of dinitroanilines are mitosis interference and prevention of normal cell wall/plate formation and root development (Callahan, 1994).

Because somatic mutations occur during cell division, the scientists analyzed phenotypic stabilities of these six bermudagrass cultivars during repeated cycles of grow-in with and without chronic exposure to dinitroaniline herbicides. From five studies, four off-type grasses were found in Champion bermudagrass treated with both herbicides. DNA testing revealed the off-types were distinct from Champion, while flow cytometry tests indicated genetic changes at the genome level were likely responsible. No off-types were observed in the untreated bermudagrass.

This experiment shows surprising new possibilities of exposing hybrid bermudagrass to cell division inhibiting herbicides and resulting in somatic mutations and genetic instabilities. Somatic mutations include single gene changes, aneuploidy (the addition or loss of a chromosome) and polyploidy (Wan et al., 1991).

DNA replication and chromosome segregation are fundamental processes required for cell division and are strictly controlled during the cell cycle (Buchanan et al., 2000). Cell cycle disruption from herbicide exposure could therefore alter bermudagrass chromosome segregation and arrangement in DNA sequencing and result in off-type mutations (Figure 1).

As turfgrass cells divide, spreading of genetic information to new cells increases the likelihood of species survival (Buchanan et al, 2000). Mitotic inhibiting herbicides, however, disrupt these events. Cell division inhibitors not affecting DNA synthesis may still affect bermudagrass genetics by increasing the amount of DNA per cell (Devine et al., 1993).

Exposure to these herbicides with prevalent soil persistence and potential residual effects may have deleterious effects on ultradwarf bermudagrass, as exemplified by the study of Goatley et al. (2003). While the researchers examined dinitroanaline herbicides, many other herbicides commonly used in golf course management inhibit mitosis, microtubule assembly, RNA biosynthesis and protein biosynthesis.

Cell division inhibitors are effective for pre-emergent control of Poa annua and summer annual weeds, but will likely not be suitable for ultradwarf bermudagrass putting greens. Dithiopyr has a similar mode of action to the dinitroanilines and has shown bermudagrass injury (Fagerness et al., 2002; Ferrell et al., 2003). Bensulide, a thiocarbamate, has shown minimal to no foliar injury during spring transition and summer growth (Callahan, 1976). However, root mass of actively growing Tifgreen bermudagrass was reduced after 60 days following exposure to bensulide at 8.4 and 16.8 kilograms of active ingredient per hectare (kg a.i. per hectare) (Bingham, 1967), and root mass restrictions of zoysiagrass and Kentucky

Continued on page 76

FIGURE 1

Exposure to herbicides with prevalent soil persistence and potential residual effects may have deleterious effects on ultradwarfs.
Continued from page 75
bluegrass have also been reported (Engel and Callahan, 1967; Fry et al., 1986).
Amide herbicides, such as napropamide, provide effective pre-emergent control of Eleu-
sine indica (Dernoeden et al., 1984). These herbicides will likely not be applicable for ultrad-
warf bermudagrass management because of their inhibitory effects on root growth and
DNA synthesis. DiTomaso et al. (1988) found napropamide reduced root DNA synthesis after
24 hours by up to 89 percent. In addition, it was determined that the protein synthesis necessary
during mitosis was also depressed. The researchers noted the majority of cells exposed to
napropamide were arrested in the G1 phase of the cell cycle.
DNA synthesis occurs during interphase between the G1 and G2 phases of the cell cycle.
DNA synthesis in G2 and early mitosis would lead to a change in ploidy, DNA content and
genoome copy number, and interfere with chro-
mosome segregation (Buchanan et al., 2000). However, assembly of protein complexes that
mediate initiation of DNA synthesis is promot-
ed during the phase preceding DNA synthesis. Protein kinases and regulation subunits, poten-
tial targets for herbicides, control major cell
cycle transitions (Buchanan et al., 2000).
Herbicides, like pronamide, may increase
DNA, RNA, and cell cycle proteins and are
commonly used in golf course management.
Auxin-type postemergent herbicides, such as
the phenoxyalkanoic acids, cause phytotoxic
symptoms such as epinasty, swelling, twisting
and bending of treated plant parts, eventually
causing cell division to cease (Rao, 2000).
The postemergence herbicide, 2,4-D,
affects all types of RNA, as well as DNA and
ribonuclease, while asulam appears to inhibit
cell division and expansion of plant meristems
by disrupting microtubule assembly (Rao,
2000). Interference with RNA and protein syn-
thesis has also been observed with asulam
(Veerasekharan et al., 1977).

It's reasonable to consider a
potentially safe pre-emergent
herbicide for dwarf bermudgrass
that would not inhibit root-cell
division or disrupt mitosis.

Potentially safe pre-emergents
for ultradwarfs
Since pre-emergent herbicides are applied
months before weed-seed germination ceases,
the herbicide must strongly adsorb to soil par-
ticles and remain in sufficient concentrations to
provide effective season-long weed control
(Branham, 1994). Thus, persistence of herbi-
cide exposure is crucial for these herbicides to
be effective.
It is reasonable to consider a potentially safe
pre-emergent herbicide for dwarf bermuda-
grass that would not inhibit root cell division or
disrupt mitosis.
Turf scientists have regarded a protoporph-
yrin inhibitor, oxadiazon, as one of the safest
pre-emergent herbicide for high-quality turf-
grasses (McCarty and Murphy, 1994). This con-
tention is based on the mode of action of this
compound, inhibiting shoot emergence of sus-
cceptible weeds without preventing root cellular
division.
Oxadiazon, an oxadiazole herbicide, has a
similar mode of action to diphenyl ethers. These
herbicides are potent inhibitors of the enzyme
protoporphyrinogen oxidase, commonly
referred to as protox, key to chlorophyll and
cytochrome syntheses (Rao, 2000). Protox oxi-
dizes protoporphyrinogen (PPGIX) to proto-
porphyrin IX (PPIX). When protox is inhibited
by oxadiazon, an uncontrollable accumulation
of PPIX occurs in the thylakoid membrane,
where chlorophyll harvests light energy for
photosynthesis.
An oxidation of molecular oxygen to PPIX
occurs causing an abstraction of hydrogen from
fatty acids (Rao, 2000). Lipid radicals and lipid
peroxidations cause a loss of chlorophyll and
carotenoids and eventually leaky membranes
leads to cellular disintegration.
On Tifgreen bermudagrass, oxadiazon pro-
vided 100 percent control of Digitaria san-
ginalis in three consecutive years (Callahan
and High, 1990). Oxdiazon applied 60 days
before overseeding at 2.2 kg per hectare pro-
vided >90 percent annual bluegrass control in
overseeded bermudagrass putting greens (Toler
et al., 2003). Tifway bermudagrass treated with
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Continued from page 76

single applications of oxadiazon at 1.12 and 2.24 kg per hectare provided 70 percent pre-emergent control of Kyllinga squamulata 18 weeks after initial treatments (Bunnell et al., 2001). Johnson (1980) observed that oxadiazon at 4.5 and 13.4 kg per hectare did not affect Tifdwarf or Tifway bermudagrass rooting.

From these studies, oxadiazon appears to give exceptional control of summer and winter annual weeds in bermudagrass turf. Because mitosis is unaffected in root cellular division, this mode of action will likely be most suitable for ultradwarf bermudagrass management.

As dwarf-type bermudagrasses continue to expand on Southern putting greens, superintendents depend heavily on new research regarding best management practices. Pre-emergent herbicide applications are the basis of turf chemical weed control programs and ideally should not restrict rooting of bermudagrass during spring root regeneration (McCarty et al., 2001).

Currently, turf managers require information regarding herbicide safety on ultradwarf bermudagrass as there are none labeled for use on these grasses.

Superintendents managing ultradwarf bermudagrass should be aware of the potential harmful effects of pre-emergent herbicides, especially with cell division inhibitors. Since cell division and mitosis inhibitors are commonly used in golf course management, turf managers will likely need to explore other herbicide options for successful long-term ultradwarf bermudagrass culture.

McCullough is a graduate assistant; McCarty, Baird; and Whitwell are professors; and Liu is an associate professor in the Department of Horticulture at Clemson (S.C.) University.

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Insect Damage Allows Other Pests to Thrive

By Doug Richmond

Too often, turf managers will point a finger at weeds or poor fertility when turfgrass starts to look bad. In many cases, however, these outward signs are only symptoms of a much less obvious problem — root-feeding insects.

It is important to remember that the foundation for insect management is built on cultural practices that promote healthy turfgrass plants. Healthy plants are more tolerant of insect feeding and are better able to recover from damage.

Proper turfgrass species and cultivar selection, mowing height, fertility, and water management can all decrease the likelihood that insect pests will cause noticeable damage in the first place. For instance, there is strong evidence that proper fertility management can have a disproportionate influence on plant susceptibility to insects, diseases and other environmental stresses. Overfertility or poorly timed fertility can reduce root growth, making plants more vulnerable to drought and damage by root-feeding insects. Excess fertility can also promote the growth of succulent tissues that insects and diseases favor. On the other hand, soil insects can also be at the root of problems that are not so obviously related.

Although root-feeding insects can reduce the vigor of turfgrass plants, only rarely do we make the connection between this damage and other ensuing problems such as diseases or weed invasions. In fact, we tend to think of weeds, insects and diseases as separate and independent concerns in turfgrass management. To a large extent, our approach toward research and extension reflects this thinking. However, most biologists would admit that there can be a great deal of interdependence among these different components, even if the connections aren’t always obvious.

A turfgrass manager who relies on his/her biological knowledge to solve pest problems will have a broader view of how turfgrass systems work and will be more likely to make proper diagnoses.

When turfgrasses are attacked by insects such as billbugs or white grubs, their ability to compete with encroaching weeds is compromised. Worse yet, when turfgrass plants die as a result of insect damage, the new occupant of the formerly turfgrass-covered site will likely be a weed.

In a recent study, I watched weed invasions into stands of Kentucky bluegrass suffering from different levels of billbug damage. At the undamaged site, few weed seedlings were ever present. Continued on page 80
Continued from page 79

and none of these weeds were able to establish. However, at the site where moderate billbug damage was apparent, weeds became a significant problem.

What would be the normal course of action for a turfgrass manager under the previous circumstances? Billbug damage, for example, is often misdiagnosed as dormancy, drought stress, or soil compaction, so it may be easy to overlook a billbug problem. An effort could be made to relieve suspected soil compaction through aerification or to irrigate to revive the turfgrass. However, neither of these measures will completely solve the problem.

I suspect that most of us would reach for the herbicide to get rid of the encroaching weeds, and this would most likely be effective over the short term. Unfortunately, the herbicide will have little effect on the billbugs, which are likely to be a chronic problem. So year after year herbicide applications may be made to an ever-enlarging area, but this will only be treating the symptoms of the problem. The tug-test would have quickly revealed the billbug infestation, and a one-time insecticide treatment would have significantly reduced billbug numbers.

Because billbugs are slow to migrate into new areas, another application would likely not be needed several years. Without the billbugs, the turfgrass would be vigorous and weeds would likely not be a problem.

The previous example is only one in a long list of cases where our one-at-a-time, separate-and-independent management philosophy lacks efficiency. For instance, notable increases in the severity of rhizoctonia have been associated with mole cricket infestation in the South. Root-feeding insects may also compromise the ability of plants to outgrow the symptoms of foliar diseases. Although above-ground feeding insects like cutworms and armyworms can also affect plant performance, grasses are generally well-adapted to the periodic removal of above-ground tissues.

Due to a number of adaptations, including placement of the meristematic zone close to the soil surface and storage of nutrient reserves in underground tissues, grasses can quickly replace foliage as long as basic resources are available. Root-feeding insects, however, impose a different suite of pressures on plants that may have more serious effects than leaf feeding. Root-feeding insects can influence plant growth, biomass production and nutrient status, and may alter the nutrient profile of the rhizosphere. As a result, proper management of root-feeding insects can have dramatic effects on overall turfgrass quality. Keep in mind that these out-of-sight, out-of-mind insects are easy to overlook. It's important to understand that other pest management concerns, such as weeds and diseases, may be more closely linked to their presence than commonly recognized.

Richmond is a post-doctoral researcher in the Department of Entomology at The Ohio State University.

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