Table 1 *(Continued)*

Snow Mold and Phytotoxicity Ratings Recorded on March 28th, 2007 at Gateway GC

Treatment	Rate	Timing[a]	% Snow Mold[b]	Phytotoxicity[c]
54 AND6245	6.66 lb/M	Late	5.7 def	7 bc
55 AND6246	6.66 lb/M	Late	9.3 def	7 bc
56 AND5017	6.66 lb/M	Late	1.7 ef	7 bc
57 AND6247	6.66 lb/M	Late	0 f	7 bc
58 AND6248	10 lb/M	Late	3.3 def	7 bc
59 AND6259	10 lb/M	Late	10 def	7 bc
60 AND6249	10 lb/M	Late	6.7 def	7 bc
61 AND6251	10 lb/M	Late	10 def	7 bc
62 AND6252	10 lb/M	Late	4.3 def	7 bc
63 AND6254	10 lb/M	Late	3.3 def	7 bc
64 AND6253	10 lb/M	Late	6.7 def	7 bc
65 AND6255	10 lb/M	Late	8.3 def	7 bc
66 Prophesy	5 lb/M	Early	1.7 ef	7 bc
AND6257	10 lb/M	Late		
67 Prophesy	5 lb/M	Early	3.3 def	7 bc
AND6258	10 lb/M	Late		
68 Prophesy	5 lb/M	Early	1.7 ef	7 bc
AND6259	10 lb/M	Late		
69 Prophesy	5 lb/M	Early	1.7 ef	7 bc
AND6260	10 lb/M	Late		
70 AND6261	9 lb/M	Late	8.3 def	7 bc
71 AND6262	9 lb/M	Late	2.7 def	7 bc
72 AND6263	9 lb/M	Late	6.7 def	7 bc
73 AND6264	9 lb/M	Late	18.3 c-f	7 bc
74 AND6265	9 lb/M	Late	6.7 def	7 bc
75 AND6266	9 lb/M	Late	3.3 def	7 bc
76 AND6267	9 lb/M	Late	1 ef	7 bc
77 AND6268	9 lb/M	Late	6.7 def	7 bc
78 Prophesy	5 lb/M	Early	1.7 ef	7 bc
AND6269	9 lb/M	Late		
79 Prophesy	5 lb/M	Early	1.7 ef	7 bc
AND6270	9 lb/M	Late		
80 Prophesy	5 lb/M	Early	5 def	7 bc
AND6271	9 lb/M	Late		
81 Prophesy	5 lb/M	Early	0 f	7 bc
AND6272	9 lb/M	Late		
82 Prophesy	5 lb/M	Early	1.7 ef	7 bc
AND6273	9 lb/M	Late		
83 Prophesy	5 lb/M	Early	4.3 def	7 bc
AND6274	9 lb/M	Late		
84 Prophesy	5 lb/M	Early	3.3 def	7 bc
AND6275	9 lb/M	Late		
85 Prophesy	5 lb/M	Early	3.3 def	7 bc
AND6276	9 lb/M	Late		
86 Daconil WeatherStik	5.5 FL OZ/M	Late	0.7 ef	7 bc
26GT	4 FL OZ/M	Late		
87 Daconil WeatherStik	5.5 FL OZ/M	Late	1.7 ef	7 bc
Medallion	0.5 OZ/M	Late		

Means followed by same letter do not significantly differ (P=.05, Student-Newman-Keuls)

[a] Early and late fungicide treatments were applied on Oct. 16, 2006 and Nov. 5, 2006, respectively

[b] Mean percent diseased area

[c] Phytotoxicity was rated on a scale of 1-9 where 1 = straw colored, 6 = acceptable, 9 = dark green

2006-07 Snow Mold Control Evaluation

Quali-Pro Auxiliary Trials: Sentryworld GC and Gateway GC

By Paul Koch, Department of Plant Pathology
and Jake Schneider, Department of Horticulture
University of Wisconsin / Madison

OBJECTIVE

To evaluate fungicides for the control of Typhula blight (caused by Typhula ishikariensis and Typhula incarnata) and pink snow mold (caused by Microdochium nivale).

MATERIALS AND METHODS

This evaluation was conducted at SentryWorld Golf Course in Stevens Point, Wis. on a Penneagle creeping bentgrass (Agrostis stolonifera) fairway nursery maintained at 0.5-inch cutting height, and Gateway Golf Club in Land O' Lakes, Wis. on a creeping bentgrass (Agrostis stolonifera) and annual bluegrass (Poa annua) fairway nursery managed at a height of 0.5 inch. Individual plots measured 3 ft x 10 ft (30 ft 2), and were arranged in a randomized complete block design with four replications. Individual treatments were applied at a nozzle pressure of 40 p.s.i using a CO^2 pressurized boom sprayer equipped with two XR Teejet 8005 VS nozzles. All fungicides were agitated by hand and applied in the equivalent of 2 gallons of water per 1000 ft 2 . Only one application was made at each site, on November 5, 2006 at Gateway GC and November 9, 2006 at Sentryworld GC. There was continuous snow cover on the plots at Gateway from November 30th, 2006 to late March 2007 (120 days) and at Sentryworld from mid-December

2006 to mid-March 2007 (90 days). Percent snow mold damage was recorded on March 28, 2007 at Sentryworld GC and Gateway GC. Data obtained were subjected to an analysis of variance to determine significant differences between treatment means.

RESULTS AND DISCUSSION

Disease pressure at the Sentryworld GC plot was fairly high this season (75% disease on untreated control) due to a combination of Typhula incarnata and Typhula ishikariensis, while pressure was even higher at Gateway GC (96% disease on untreated control) due primarily to T. ishikariensis. All treatments applied at both sites significantly reduced disease when compared to the untreated controls, but none of the treatments tested completely controlled disease symptoms at Gateway GC. Treatment 2 provided excellent control of T. incarnata, but failed to provide the same level of control of T. ishikariensis. No phytotoxicity was observed with any treatments applied. The mean percent snow mold per plot for each individual treatment is presented in the tables on Pages 9, 10 and 11.

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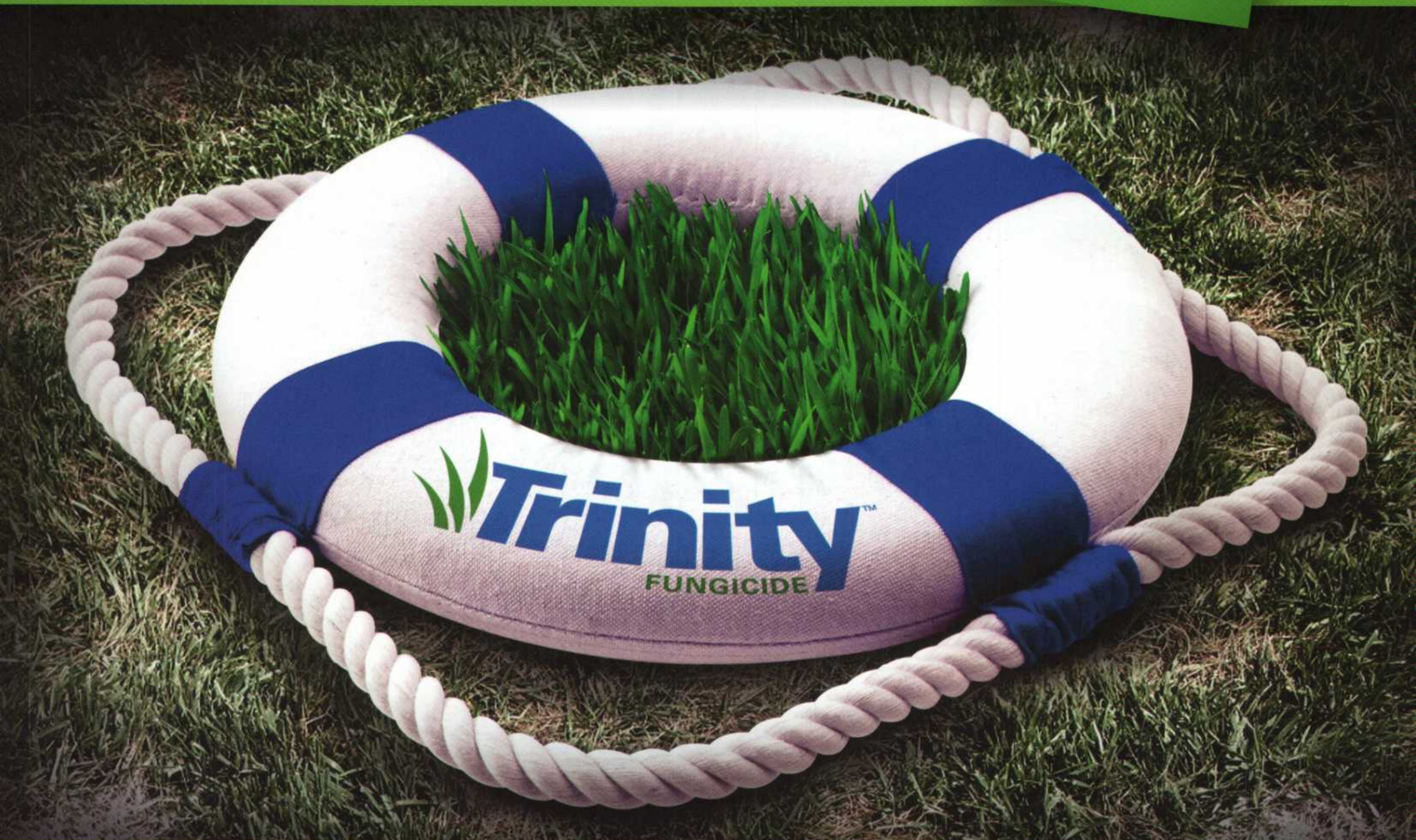

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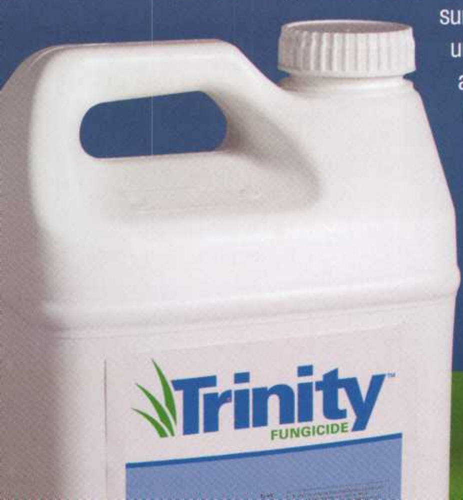

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Safety: *Keeping Awareness Alive*

How can you keep your people thinking about safety? Sit everyone down, hand out copies of a long list of recent safety infractions, and go through your long list of safety do's and don'ts. Make sure to include all of your pet peeves, and go through them one by one (at the top of my list is seeing two people in a truck moving in reverse without a spotter behind the truck).

- Or, rather than droning on and on
- And risking losing everyone's attention,
- Consider this:
- A study at the University of Minnesota
- Showed that with bullet point lists
- People retain the first and second points,
- And maybe the last one,
- But little or nothing else,
- And adult learners retain even less from long lists.

However, people do remember stories. Storytelling is as natural as breathing to most people. A story can be the vehicle that carries the safety message and commits it to memory. People do remember the point when it is told in the context of a story.

Get your people talking about safety. We set aside a portion of our regular company meetings for verbal near-miss reporting. Reports written by the shy and read aloud by others can work too. A near-miss is a safety related incident where things nearly went wrong; in some cases things nearly went terribly wrong - not an actual accident. Near-miss reporting is an opportunity to expand the experience of all employees and uncover weaknesses in safety training prior to an actual accident. It is a preventative measure, not an after-the-fact recognition.

Under no circumstances should this be a time to report actual accidents. Actual accidents need to be reported based on your company policy. Obviously management retribution for near-miss reporting in this context will have a cooling effect on this program. A safe environment for the employees to report their stories needs to be firmly outlined.

Employee interest in implementing this plan can be slow at first but stick with it, it really works. The format is simple (please focus really hard four short bullet points ahead):

- What happened?
- What did you do?
- What would you do different next time?
- Ask the rest of the group for comments.

Start with a general call for near miss stories from the past. Then shorten the time frame to the the last safety meeting. As part of a full safety program, near-miss reporting will create a portion of the program that lives and breathes on its own, rather than strictly using management driven safety initiatives.

A good example: "Last week we were removing a tree, and we had the entire area marked off with signs and cones, when a bike rider rode through the work zone and almost got hurt. So we moved the trucks to make it physically impossible for another bike rider to ride through. Next time we will make it impossible for a bike to get through and be more vigilant."

A poor example - "Yesterday we dented the back of the truck with the loader and did not get caught, so I'm telling you now because there is no retribution from management for stories told in this safe environment." Sounds like an unreported accident not a near-miss.

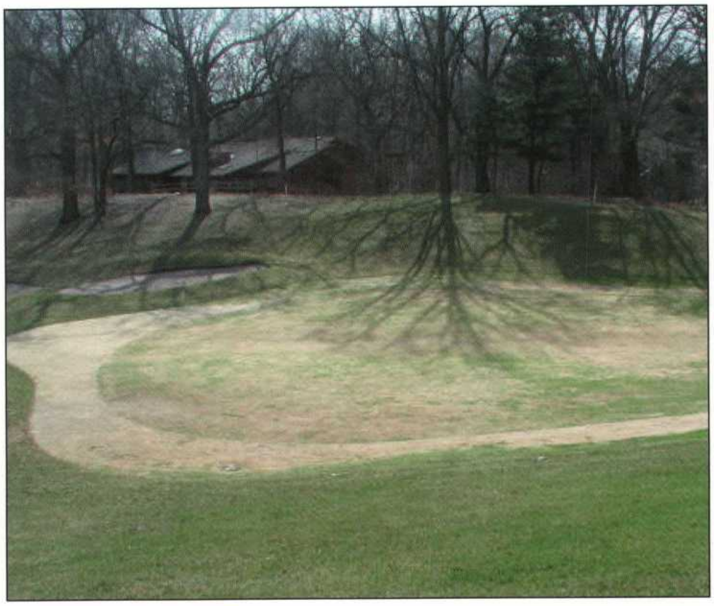
Once the stories are told or time runs out, employees then vote via applause for the best story. The company should offer a prize of some sort. My wife is no sports fan, but her favorite sports quote is from Randy Moss, the former Minnesota Vikings football player. After fake mooning the Green Bay fans and paying the resulting $10,000 fine, he said "I'm rich; I don't write checks for ten grand. I pay straight cash homey" How should the best story be rewarded? Straight cash homey.

(Editor's Note: Affiliate members are encouraged to supply articles from a question they have been asked by a Superintendent. Please send articles to scott@mgcsa.org.)

The Physiology of Low Temperature Stress (Winter Injuries!)

By Dr. W. A. (Bill) Torello
Professional Turf Consultant

Low temperature stresses are not solely the problem of freezing, snow-laden climates. Low temperature stresses greatly affect the southern climes as well, particularly at transition zones and elevated areas. However, the physiology or mechanisms of low temperature stesses are very different between cool and warm season grasses. The actual mechanisms of low temperature injuries and low temperature stress resistance are quite complex but if I had to use one word to encapsulate the entire picture.....it would be "carbohydrates." The natural fluctuations (and unnatural fluctuations caused by turf managers) of simple and complex carbohydrates directly reflects the level of low temperature resistance and possible damage. In my past incarnation as a turf professor, I would classify turf managers as "carbohydrate managers". This pertains not only to low temperature stresses but to most all environmental stresses including high temperature, drought, salinity and light (shade) stresses. In fact, it could very easily be shown that these stresses directly affect carbohydrate/sugar levels which then can directly affect the water status of the plant. Unnatural or wide fluctuations in carbohydrate levels can substantially contribute to drought stress or the movement of water from healthy, living cells which then result in drought injuries. Low and high temperature and salinity stresses are, in great part, drought stress induced injuries!



In this series of articles, I will cover, in detail, what low temperature stress injuries are, how they are induced, what determines the extent of injury and how turfgrasses deal with low temperature stresses. I will also talk about the cultural practices associated with reducing injuries as well as how turfgrasses deal with low temperatures stress naturally....through resistance mechanisms that you can enhance.

Before launching into the subject, we need to define a few things. Those of you that were my former students, this will be a good refresher! First, let's define environmental stress resistance. The term "resistance" is the primary term all of us should use when talking about the ability of a turfgrass to naturally deal with temperature, drought, salinity, light or other environmental stresses.

Resistance can include one or more of the following:

Tolerance: Physiological mechanisms a plant uses to deal with the stress once it enters into the tissues. What I mean here is simple. If the stress gets into the plant....i.e. salt taken up into tissues, low or high temperature changes inside the plant, water deficits in the tissues, the plant must have "internal" mechanisms to deal with the intrusion and minimize injury. In other words, the stress becomes internal but the plant can "tolerate" the intrusion by various physiological/biochemical mechanisms induced by the stress.

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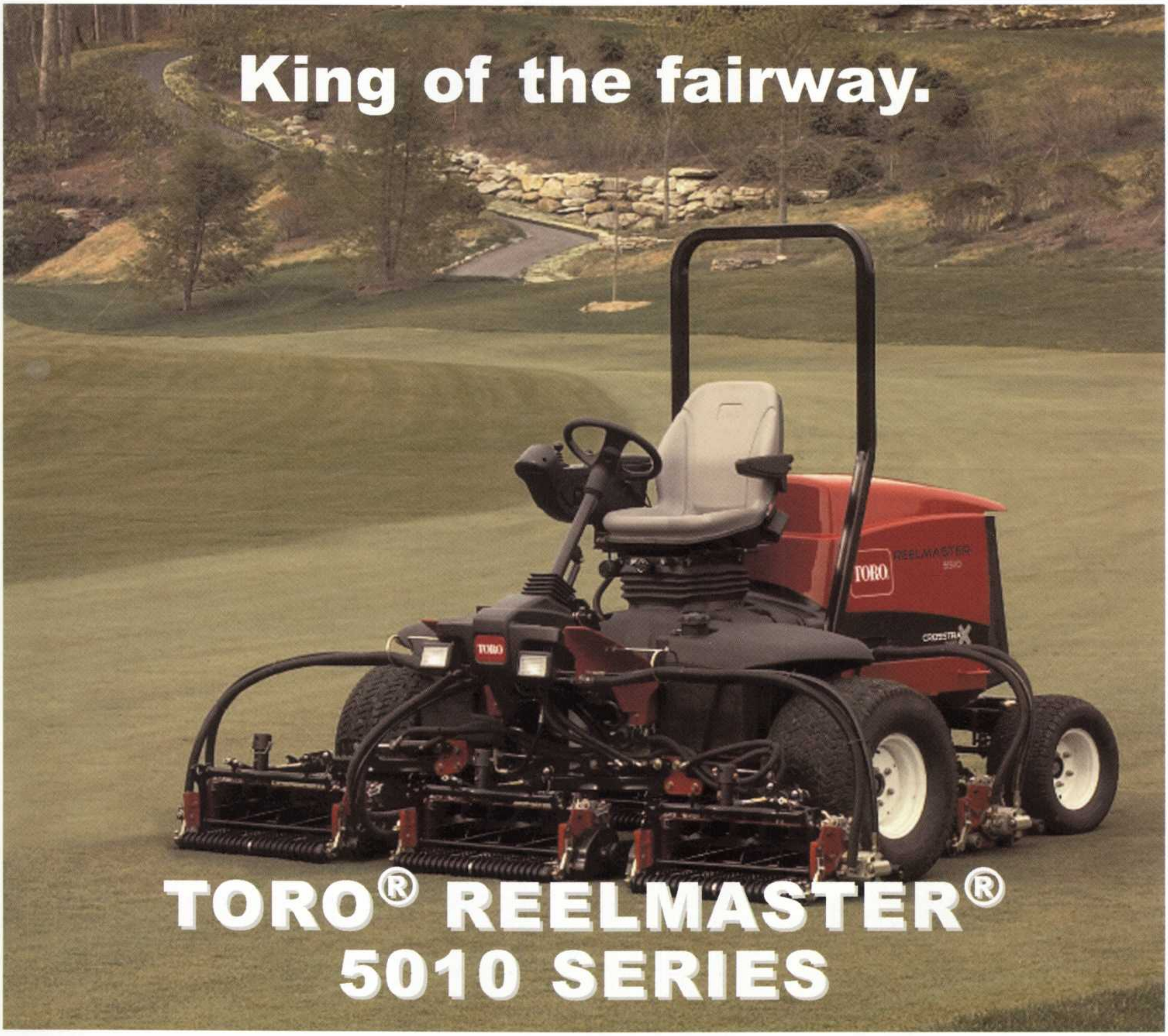

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TORO. Count on it.

Low Temperature Stress-

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Avoidance: The ability of a plant to actually keep the stress outside the tissues and not let it enter the inside of the plant. A classic example of this would be the ability of certain plants to limit or even exclude the uptake of sodium thereby "avoiding" sodium toxicity stress. When we talk about low or even high temperature stresses in turfgrasses, we do not include avoidance since internal temperatures closely reflect ambient temperatures.

Escape: A term used to define those plants which can employ mechanisms to actually completely escape the stress. Not to be confused with avoidance. Escape is the predominant mode of resistance in annual plants (such as Poa annua) that are prolific seed producers which, if severely injured or even killed by the stress, the dormant seed simply waits until good conditions prevail again to germinate. This is also true of those plants that can be induced into dormancy by such stresses as drought and high temperature. Drought induced dormancy of cool season or even warm season turfs is a mechanism of escape. Stems (rhizomes/stolons/tillers) remain living while foliage and roots are killed. When wetter and/or cooler conditions return, the stems vegetatively produce another root system as well as foliage. Carbohydrates are absolutely necessary for this to occur. If there is a limited amount of carbohydrate in the stem tissues, then dormant plants will have a very hard time "coming back" and regrowing. Extensive dead areas due to drought stresses are a direct reflection of the amount of carbohydrate that was stored in stems prior to the drought stress.

So, in a nutshell, resistance is defined by either tolerance, avoidance or escape or any combination of the three. This may seem like a minor thing to understand, but if you are to fully understand each stress and ways to cope with environmental stresses, you need to understand these distinctions.

What I intend to do is make this a series of "short" papers which will cover the following:

1. Defining low temperature stresses
2. Discussing the associated injuries: intensity, duration and extent.
3. Natural resistance mechanisms.
4. Culturally induced resistance.
5. New technology

Low Temperature Stress: Part II

We need to get a handle on how carbohydrates naturally fluctuate in turfgrasses to fully understand and get an appreciation of how they greatly affect whether a turf undergoes low temperature stress and, if so, the extent of injury. Understanding carbohydrate fluctuations is also very important as related to virtually all other environmental and even biotic stresses. For those of you that have higher degrees and understand these principles -- have patience. There will be many parts/articles to this subject.

Carbohydrates are a major biochemical group in all living systems. As a group, they can be very simple (such as glucose, sucrose and other simple soluble sugars) or very complex such as the turf storage

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Low Temperature Stress-

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carbohydrates starch and fructosans which are very, very large, insoluble molecules. To take it even further, cell wall material is primarily cellulose...which is a very large and much more complex carbohydrate compared to the storage forms. You get the point, it's a large group. We will be only focusing on simple, soluble sugars and the storage forms of starch and fructosan.

Simple sugars are water soluble for the most part. Easy example is sucrose (table sugar). Another example is glucose, the most important sugar in most biological systems, the first product of photosynthesis (and the stuff they pump into your veins when you are at the hospital to give you more energy). Notice that they both end in the suffix "ose." In fact, all simple soluble sugars end in "ose." Easy to identify. Glucose, fructose, sucrose, maltose, dextrose, lactose, xylose, arabinose, they are a large group but we are only interested in glucose, fructose and sucrose since they are the "saccharides" that commonly accumulate to significant levels in plants. Simple, one molecule sugars are called monosaccharides such as glucose, fructose and lactose. A common "disaccharide" is sucrose which is composed of a glucose and fructose molecule bonded together. Sugars having more than a few simple saccharides are called "polysaccharides" and if they get complex they no longer have the saccharide label such as starch, fructosan, cellulose, hemicellulose etc.

A simple analogy is to think of carbohydrates like "chains". Two links are disaccharides, a few links are polysaccharides, long - multi-branched chains are complex carbohydrates (storage carbohydrates) such as starch and fructosan. In fact, the storage carbohydrates have many, many thousands of "links". Each "link" is a simple glucose or fructose molecule. It is critical to understand this since the first product of photosynthesis....glucose is not only the plant's primary energy source, but also a material building block to make all other complex biomolecules....such as starch. All plants must have large amounts of glucose to grow and maintain healthy physiology. A plant can, and does, store glucose for later use by making large chains of it called starch or fructosan. These "storage" forms

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Low Temperature Stress-

(Continued from Page 19)

accumulate in tissues during times when photosynthesis is at its greatest rate and glucose is being "overproduced" beyond what the fast-growing plant needs. In fact, during these high growth periods, so much glucose is being produced that if it were allowed to stay in the glucose form, it would get to be toxic and act like a salt in the tissues. That's why overproduction of glucose is immediately shunted into making very large starch or fructosan molecules which can be accumulated safely and not have a "salt" effect.

When is the rate of photosynthesis and glucose production at its greatest? For cool season turfs it is in the spring and fall months when temperatures average around 65-70F. For warm season grasses it is in the middle of the summer when high temperatures ranging from 90-100F prevail. These are the periods when turfgrasses have the capacity to store large amount of glucose as starch and/or fructosan for later use when photosynthesis and production of glucose is low like in the summer months for cool season grasses and the winter months for warm season grasses. This is a critical point to understand. When environmental conditions do not favor photosynthesis, the plant cannot produce enough glucose to maintain growth and good physiological health and is prone to environmental stress and biotic stress injuries. If, however, the plant has "stored" vast amounts of glucose as starch, then it will have a "reserve" to draw on during these stressful periods. The starch or fructosan will be broken down back into glucose for the plant to use since photosynthesis cannot supply enough.

These periods of high photosynthesis and low photosynthesis along with high and low levels of glucose production are natural and occur each and every year. You can imagine that if the plant is inhibited in some way during the spring from producing large amounts of storage carbohydrate (starch), then it will literally be "starving" come the summer months (winter months for warm season grasses). Just as importantly, if the turf does not accumulate enough storage carbohydrate in the fall months, it will not have enough glucose to maintain proper metabolism through the winter "dormant" months and be very prone to winter-low temperature stresses. Remember, dormancy is not death. All cells and tissues, particularly in stems, remain alive and etabolizing...albeit very, very slowly. The turf needs glucose throughout the winter months and photosynthesis is either very, very slow or not functioning under snow/ice covers.

It is very important to understand these basic principles.

Low Temperature Stress III: Freezing Stress

Now we know that cool season grasses naturally accumulate large amounts of storage carbohydrate during the early to mid-fall when the rate of photosynthesis is at its greatest and overproduction of glucose is normal. This reserve carbohydrate is not only used as a "food" source for dormant tissues, it also acts as an "anti-freeze" as well as ensuring that cells and tissues maintain a high level of hydration.....keeps water in cells and avoids dehydration. Remember that winter months are very, very dry with low relative humidities which can, and do, dehydrate turfs, particularly when there is no snow or artificial cover. How the reserve carbohydrates, starch and fructosan, do this is really very clever. As you remember, reserve carbohydrates are very large molecules and, as such, do not act like salts in the tissues as pure glucose would. This is a protective mechanism that the turf uses so it will not

(Continued on Page 21)