Summer Decline
Can Cool-Season Turfgrasses Take the Heat?

By Richard J. Hull, University of Rhode Island

This past summer has not been an easy time for many turf managers. The unrelenting drought and above normal temperatures took their toll on cool-season turfgrasses in lawns, athletic fields and golf courses. If conventional wisdom is to be believed, we have just seen the beginning of bad summers, as global warming sets in and summers generally become drier and hotter. On the other hand, is this really anything unusual? Cool-season turfgrasses always decline during the summer and greater problems should be expected as we try to push these grasses even further south.

In short, there is a fundamental problem inherent in attempting to maintain green, vigorous turf from cool-season grasses during the hottest months of the year. Some years are worse than others, but the problem is chronic. What is behind the summer decline of turfgrasses? Do we know enough about this problem to offer any hope of solving it? In this article, I will take a shot at answering these questions.

High Temperature Stress

What exactly is high temperature stress? This is not a simple question to answer because, in the field, high temperature is often accompanied by high light and insufficient water. Consequently, the resulting decline in turf quality is rarely caused by one stress alone, but by the interaction of several stresses. The principal factor of summer decline of turfgrasses is primarily heat, augmented by other stresses.

Optimum Temperatures for Shoot and Root Growth

Table 1. Optimum temperatures for shoot and root growth of cool- and warm-season turfgrasses.

<table>
<thead>
<tr>
<th>Grass type</th>
<th>Shoot growth Degrees F</th>
<th>Root growth Degrees F</th>
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<tbody>
<tr>
<td>Cool-season</td>
<td>59-75</td>
<td>50-64</td>
</tr>
<tr>
<td>Warm-season</td>
<td>81-95</td>
<td>75-84</td>
</tr>
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</table>

FROM DIPAOILA AND BEARD (1992)
This is the opinion of Dr. James B. Beard, who coined the term “summerkill” to describe summer decline of cool-season grasses. He includes opportunistic diseases in the mix of conditions contributing to summer turf decline and death, but heat augmented by drought is the primary cause. Dr. Bob Carrow, with the University of Georgia’s Experiment Station at Griffin, lists eight secondary factors that contribute to or result in summer turf decline. He too credits high temperature stress as being the primary culprit.

Turfgrasses vary in their resistance to high temperatures, which is not surprising because the optimum temperature for growth differs markedly among grasses. Warm-season grasses exhibit optimum shoot growth at temperatures some 20 degrees F higher than are optimum for cool-season grasses. Roots of cool-season grasses also grow best at temperatures 20 to 25 degrees F cooler than those best for warm-season grasses. The range in optimum temperatures for both grass types reflects the variation among different species within each type. Optimum temperatures for root growth are also about 10 degrees cooler than for shoot growth.

Given the shape of growth curves, temperatures either below and above the optimum range are by definition stressful. That is, they constitute conditions that in some way are limiting to plant growth. In the context of this discussion, we are concerned with supraoptimal temperatures and how they pose limits to the growth or performance of turfgrasses. There is a clear difference between cool-season and warm season grasses with regard to their tolerance of high temperatures (Table 1). The optimum temperatures for warm-season grasses are well within the supraoptimal range for cool-season grasses for both shoots and roots. This should tell us that a basic difference between these two grass types probably explains their differing high temperature tolerance. This will be addressed in the next section.

When temperatures become very high, plants experience direct thermal injury which leads to rapid death. For most turfgrasses, killing temperatures occur between 131 and 142 degrees F (DiPaola and Beard 1992). Warm-season grasses do require somewhat higher temperatures than cool-season grasses before death occurs, but the differences are not as great as for optimum growth temperatures.

Killing temperatures cause protein denaturation as thermal energy becomes so great that the structural integrity of the proteins can no longer be maintained. This results in a loss of enzyme activity and a collapse of biochemical pathways and their control. Lethal temperatures are not the cause of summer decline in turf quality because they are well above those at which decline is observed. Sublethal temperatures can kill turf, but normally indirectly through the effects of drought or pathogen caused injury. Thus, an explanation of heat injury in cool-season grasses will likely be found in that which distinguishes them from warm-season grasses.

C-3 Photosynthesis and Consequences

The basic difference between cool- and warm-season grasses is in their method of capturing carbon dioxide (CO₂) for photosynthesis. All plants fix CO₂ by means of the same enzyme, ribulose bisphosphate carboxylase/oxygenase (RubisCO). This enzyme catalyzes the following reaction.

\[ \text{RuBP} + \text{CO}_2 \rightarrow 2 \text{PGA} \]

Here ribulose bisphosphate (RuBP), a five-carbon sugar, binds with CO₂ to form an unstable 6-carbon molecule that spontaneously decomposes into two three-carbon molecules of 3-phosphoglyceric acid (PGA). This reaction is energetically favored, but only occurs in the presence of the RubisCO enzyme. Because the product of this reaction that contains the carbon of fixed CO₂ is a three-carbon acid (PGA), photosynthesis that fixes CO₂ by this reaction is called C-3 photosynthesis. From PGA, the carbon passes through several
reactions until it is reduced to the level of carbohydrates, sugars and starch, or regenerates the RuBP necessary to keep the photosynthetic carbon fixation cycle going.

Rubisco is an ancient enzyme, almost as old as life itself. It evolved in a world significantly different from what we have today. When photosynthesis first emerged, the atmosphere was enriched in CO$_2$ and very low in oxygen (O$_2$). Over the years, atmospheric CO$_2$ levels have decreased to about 0.035 percent of atmospheric gases, while O$_2$ has increased to 21 percent of the atmosphere. Under these modern conditions, Rubisco finds itself much less efficient in fixing CO$_2$. First of all, there is much less CO$_2$ available, so the enzyme can only operate at about half of its maximum rate. In addition, O$_2$ competes for the CO$_2$ binding site on the enzyme where the following oxygenase reaction is catalyzed.

$$\text{RuBP} + \text{O}_2 \rightarrow \text{PGA} + \text{P-Glycolate}$$

Under high temperatures, photosynthetic rates decline as photorespiration increases.

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$$\text{RuBP} + \text{O}_2 \rightarrow \text{PGA} + \text{P-Glycolate}$$

Here RuBP binds with O$_2$ but the five-carbon product that is formed degrades to one PGA and one two-carbon phosphoglycolic acid molecule. This oxygenase reaction fixes no CO$_2$ and the P-Glycolate formed is metabolized via a pathway that releases CO$_2$ and consumes energy. Because this overall reaction occurs only in the light, consumes O$_2$ and releases CO$_2$, the process is called photorespiration.

When temperatures are high, the solubility of gases in water decreases, which makes both CO$_2$ and O$_2$ less available for fixation by Rubisco. However, the ratio of CO$_2$:O$_2$ in solution decreases from 0.051 to 0.038 as the temperature increases from 41 degrees to 95 degrees F, which means that as the temperature rises, less CO$_2$ compared to O$_2$ remains in solution. Therefore, photorespiration is favored while photosynthesis is hindered (Taiz and Zeiger 1998). As a result, under high temperatures, photosynthetic rates decline as photorespiration increases.

**C-4 to the Rescue**

The decrease in photosynthetic efficiency caused by low CO$_2$ and high O$_2$ has been addressed in some semitropical plants that normally grow under high temperatures. These plants have evolved a separate CO$_2$ trapping and transport mechanism by which they can concentrate CO$_2$ in special cells to levels more than ten times that normally available to Rubisco. They do this by initially fixing CO$_2$ into a four-carbon acid, according to the following reaction catalyzed by the enzyme phosphoenolpyruvate carboxylase (PEPcase):

$$\text{PEP} + \text{HCO}_3^- \rightarrow \text{OAA}$$

Here a three-carbon acid, phosphoenolpyruvic acid (PEP), reacts with a bicarbonate ion and produces a four-carbon acid, oxaloacetic acid (OAA). In water, bicarbonate ion is in equilibrium with atmospheric CO$_2$ according to the reaction:

$$\text{CO}_2 + \text{H}_2\text{O} \leftrightarrow \text{H}_2\text{CO}_3 \leftrightarrow \text{HCO}_3^- + \text{H}^+$$

The equilibrium between CO$_2$ and HCO$_3^-$ is such that, in the sap of leaf cells during photosynthesis, HCO$_3^-$ is 50 times more concentrated than CO$_2$. Since PEP-case fixes the more abundant HCO$_3^-$ rather than CO$_2$, its reaction rate is much greater than that of Rubisco which can only react with CO$_2$, not HCO$_3^-$. In addition, PEP-case does not react with O$_2$ so there is no photorespiration to decrease carbon fixation.

Once carbon is incorporated into a C-4 organic acid, that acid is transported into bundle sheath cells which surround the vascular bundles of the leaf (Fig. 1). In these bundle sheath cells, the C-4 acid is decarboxylated to form a three-carbon acid and a CO$_2$. The CO$_2$ accumulates in these specialized cells to levels 10 times higher than that normally available for photosynthesis. The elevated CO$_2$ levels in the bundle sheath cells allow Rubisco to fix carbon efficiently; operating at near maximum rates with much less competition from O$_2$ so little or no photorespiration occurs. The preliminary trap and transport of CO$_2$ in C-4 plants allows them to maintain high photosynthetic rates at temperatures that
would inhibit photosynthesis in C-3 plants.

You probably are familiar with many C-4 plants. These include the weeds that grow vigorously during the heat of summer when cool-season turfgrasses are barely hanging on. Crabgrass, nutseed, fall panicum, purslan, and spotted spurge are all C-4 plants. They are highly competitive with turfgrasses during the summer because of the advantage C-4 photosynthesis gives them over C-3 plants. Warm-season turfgrasses are also C-4 plants and they do not experience summer decline because of more efficient photosynthesis.

**Root Starvation**

The initial impact of high temperatures on cool-season turfgrasses is less photosynthesis due to increased rates of photorespiration. With less photosynthetic product, there are fewer sugars available for translocation to the roots. The relationship between photosynthetic efficiency and root activity was described in an earlier *TurfGrass Trends* article (Hull 1996) on managing turf for optimum growth of roots. When roots are deprived of sugars, their growth rate slows and this limits their ability to seek water and nutrients. This, however, is just the beginning of root decline.

Turfgrass shoots do not experience high temperatures without the soil also becoming warm. Elevated soil temperatures cause root respiration to increase and this speeds the rate of sugar consumption in roots. This respiratory increase is not photorespiration, but normal oxidative respiration that burns sugars to produce the energy needed by roots to support their growth and function.

However, by the time root respiration is stimulated by soil heating, high shoot temperatures have already stimulated photorespiration in leaves and reduced the flow of sugars to the roots. Thus, roots find themselves consuming sugars more rapidly, while their supply is curtailed. If this condition persists for more than a day or two, root starvation will occur and death soon follows.

Even in humid climates, the death of shallow roots causes a release of organic mucilage into the soil that binds with water and fills pore spaces retarding water percolation through the soil (Carrow 1996). The retention of water in the soil reduces the O$_2$ levels on which roots depend for their respiration and metabolism. Anaerobic soil conditions further stress roots. Soon, the entire root system is sick or dying, and water transport to shoots is seriously impeded.

During hot weather, reduced transpiration caused by a failing root system allows leaves to overheat and direct thermal injury can occur. If this happens rapidly, grasses will not have the time or energy necessary to go into summer dormancy, and turf death will result.

Unlike drought stress, which occurs progressively as the soil column dries, heat stress can come on suddenly and create a lethal condition before the grass can respond to it.
them more vulnerable to heat injury during the hottest times of the year, which would not be the case under natural conditions.

**Oxygen Radicals and Membrane Peroxidation**

The actual destruction of turfgrass shoots by high temperatures is complex, but, except in rare circumstances, is probably not due to thermal denaturation of proteins that occurs when temperatures exceed 120 degrees F. Such temperatures can occur when transpirational cooling is inhibited and kill exposed leaves. But they are not likely to affect entire shoots in a turf sod. Toxic O$_2$ radicals are a more likely cause of high temperature induced death.

During photosynthesis, light energy causes the formation of numerous short-lived electron transport intermediates.

When CO$_2$ is being reduced to carbohydrates, it constitutes a natural sink for these electrons, and their carriers cycle back to pick up additional electrons to keep photosynthesis going. However, if high temperatures cause a shortage of CO$_2$ or promote stomates to close and prevent gas exchange between leaf and atmosphere, these electron carriers will accumulate and reduce other substances such as O$_2$.

$$O_2 + e^- \text{ Carrier} \rightarrow O_2^- + \text{ Carrier}$$

The first $O_2$ radical formed is superoxide ($O_2^-$), which can readily reduce metal ions present in the cell such as ferric iron (Fe$^{3+}$) or cupric copper (Cu$^{2+}$).

$$O_2^- + Fe^{3+} \rightarrow O_2 + Fe^{2+}$$

These reduced metal ions along with superoxide can damage sensitive enzymes and membranes, so most plants make an enzyme specifically designed to destroy superoxide: superoxide dismutase (SOD). Here, two superoxide ions interact and redistribute electrons:

$$2O_2^- + 2H^+ \rightarrow O_2 + H_2O_2$$

One is oxidized back to $O_2$ while the other is reduced to a peroxide ion ($O_2^2$) which combines with two H$^+$ to form a hydrogen peroxide (H$_2$O$_2$) molecule. Normally, the H$_2$O$_2$ is degraded to two water molecules by a peroxidase enzyme that is closely associated with the photosynthetic machinery. However, if the normal flow of reducing power (electrons) to CO$_2$ is impaired,
H$_2$O$_2$ is produced in greater amounts and will react with reduced metal ions to form hydroxide radicals (•OH). These are among the most destructive substances known to biology.

\[ \text{H}_2\text{O}_2 + \text{Fe}^{2+} \rightarrow \text{OH}^- + \cdot\text{OH} + \text{Fe}^{3+} \]

Not only is •OH highly destructive, but the cell has no effective means of detoxifying it. These •OH radicals can oxidize enzymes and membrane lipids, inactivating them and, in the case of membranes, making them leaky.

These reactions occur most readily in the chloroplasts of green leaf cells because that is where photosynthetic reactions are concentrated and where light energy is converted to chemical energy. However, the sites of respiratory oxidation (mitochondria) are also places where O$_2$ radicals will form and can be destructive, especially under conditions promoted by high temperatures. The plasma membrane, which encloses the protoplast of every cell, contains many lipids and these can be oxidized by O$_2$ radicals changing their properties, disrupting uptake processes and even the integrity of the membranes.

The significance of these reactions to heat injury in turfgrasses has been supported by recent research from Kansas State University (Liu and Huang 1998). Dr. Jun Huang's group has demonstrated a nice correlation between heat injury to creeping bentgrass and a decline in the O$_2$•- degrading enzyme (SOD) and an increase in lipid peroxidation accompanied by greater cellular leakage.

**What Can a Turf Manager Do?**

All this chemistry and recent research does not paint a very encouraging picture for the turf manager. The basic vulnerability of cool-season grasses to heat stress is inherent to their photosynthetic physiology. High temperatures will cause a decline in photosynthesis and the follow-up destructive events will occur with very little the turf manager can do to prevent them. The prospects for improvement on the genetic front are not much better. While cultivars of several turfgrasses have been identified that exhibit greater resistance to heat stress, the differences are not great and the mechanisms behind their heat resistance have not been well defined, although they are being studied. In short, there is little likelihood that any cool-season turfgrass will be developed that can tolerate heat as well as warm-season grasses.

So is summer decline of cool-season grasses inevitable? Yes, but the process can be avoided or at least delayed by proper turf management. Some suggestions proposed over the years follows:

**Prevent Heating** — Since the primary stress involves leaf heating and the resulting decline in photosynthetic output, managing turf to prevent heating would be the most direct strategy. Transpiration is the natural way plants prevent overheating, so managing grass to encourage transpiration would seem reasonable.

Maintaining adequate, but not excessive, moisture levels would be a good start. Managing for maximum root growth will also help turf to obtain water and tolerate some root injury near the soil surface. Encourage good air flow over the turf. That will reduce humidity and increase transpirational cooling.

Thinning thick vegetation and installing fans may be necessary. Syringing just before the hottest time of the day will help reduce leaf temperatures in dry climates, but may have little benefit when humidity is high. Increasing the mowing height will give the turf more leaf surface and increase photosynthetic production. It will also provide more shading and insulation of the soil surface and delay root zone heating.

**Reduce Nitrogen Fertilization** — Fertilization can be adjusted to maximize heat tolerance. Reduce nitrogen to maintenance levels so shoot growth will not be stimulated at the expense of root growth. This needs to be initiated before hot weather arrives and will probably require the use of slow-release materials. This nitrogen effect has been clearly demonstrated under controlled environments...
(greenhouses and growth chambers), but frankly, field results have been less consistent. However, the depressing effect of high nitrogen on root growth is likely and should be avoided when any summer stress is anticipated.

Beard (1997) favors higher potassium availability during the hot season. This makes sense because potassium controls stomate function and helps stabilize plant water status. Essentially, turf should enter the hottest time of the year under balanced nutrition that does not stimulate excess shoot growth.

**Plant Right Species and Cultivars —**
Species and cultivar selection for heat resistance should be considered when new construction or renovation is planned. Beard (1997) has ranked turfgrasses according to their heat resistance (Table 2). Naturally, the warm-season grasses are best, but among the cool-season options, tall fescue, hard fescue and Kentucky bluegrass are generally most heat resistant. Annual ryegrass, annual bluegrass and rough bluegrass are among the least heat tolerant turfgrasses. Unfortunately, all the bentgrasses exhibit little heat resistance which gives you few options for greens and tees. However, cultivar differences in heat resistance have been identified so even here, some choices are available.

**Delay Aerification —** Cultivation can be scheduled to minimize summer decline. On sand greens, Carrow (1996) emphasizes the importance of maintaining proper infiltration of water and air. The plugging of soil pores during the death of shallow roots can seriously aggravate a heat stress problem. Aerification with light top-dressing prior to the onset of summer heat will likely be helpful. Continued cultivation during the summer is also encouraged (Carrow 1996), but reduce top-dressing and minimize abrasion, since leaves are more subject to mechanical injury during heat stress.

The new technology for forcing air through putting greens by utilizing drainage tiles will probably have value for reducing heat associated stresses (Dodd et al. 1999). Although this approach to managing heat stress on greens has not yet been critically tested, it offers promise for alleviating several adverse conditions that are known to contribute toward heat injury.

While the prospects for dramatic advances in managing summer decline in cool-season turf are not encouraging, the situation is anything but hopeless. Thoughtful and conscientious turf management clearly is the answer. I hope this overview of the physiological causes of summer heat injury will contribute to your understanding of the problem and will be useful in helping you refine and improve your summer turf management strategy.

**TABLE 2. COMPARATIVE HEAT RESISTANCE OF TURFGRASSES**

<table>
<thead>
<tr>
<th>Heat Resistance Rating</th>
<th>Turfgrasses</th>
</tr>
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<tbody>
<tr>
<td>Excellent</td>
<td>Bermudagrass, buffalograss</td>
</tr>
<tr>
<td></td>
<td>Seashore paspalum, Zoysiagrass</td>
</tr>
<tr>
<td>Good</td>
<td>St. Augustine grass, centipedegrass</td>
</tr>
<tr>
<td></td>
<td>Medium Tall fescue, hard fescue, Kentucky bluegrass</td>
</tr>
<tr>
<td>Fair</td>
<td>Chewings fescue, red fescue, sheep fescue, perennial ryegrass</td>
</tr>
<tr>
<td>Poor</td>
<td>Creeping bentgrass, colonial bentgrass, redtop, Canada bluegrass</td>
</tr>
<tr>
<td>Very poor</td>
<td>Annual bluegrass, annual ryegrass, rough bluegrass</td>
</tr>
</tbody>
</table>

FROM BEARD 1997

Dr. Richard J. Hull is professor of plant science and chairman of the Plant Sciences Department at the University of Rhode Island. His research is focused on nutrient use efficiency and photosynthetic partitioning in turfgrasses and woody ornamentals.
The Impact of Climate On Turfgrass Pest Activity

By Christopher Sann, Pest Forecasting Group Inc.

Over the years, turfgrass experts like Dr. Eric B. Nelson at Cornell University and Dr. Dave Shetlar at The Ohio State University have more than once emphasized to me various aspects of the general relationship between climate and pest activity. However, it wasn't until I became actively involved in the production and running of a series of computer-based models to produce turfgrass pest activity favorability forecasts, using recent, current, and forecast weather conditions, that this direct linkage has become clear.

To the existing breadth of these experts' understanding, born of their strong understanding of pest biology, I would add the following observation based on two years of working with these models. Even in the presence of a relatively large insect population or a dense mass of disease inoculum, significant visual host damage is not likely to occur when conditions of temperature and moisture are substantially outside the minimums required for pest activity to occur.

Restated, pest activity, whether that activity leads to significant damage or not, is a direct function of the interaction between the pest, the host and the recent and/or current weather conditions that fall within definable ranges. If the conditions fall substantially outside (either below or above) these ranges, then significant pest activity is not likely to occur.

Pest Activity and Climate

All of the common pests of turfgrasses are exothermic — meaning they do not produce the majority of their own internal heat. Insect, disease, and weed pests absorb most of the energy required in their life processes from their surrounding environment. Within some limits, these life processes are greatly dependent on this inflow of outside energy to function properly. Also, the speed of progression to maturity is directly related to the quantity and pace of the energy inflows.

From a pest management point of view, understanding this biological concept can lead to a significant new set of tools. If you can measure the energy inflows (usually as temperature) in some meaningful way and relate that measurement to a pest's observed life processes, then it should be possible to estimate the current life-cycle stage, called phenology, of a pest at any point in time. Once the pest's phenology (in relation to turf damage) and the progress toward maturity is understood, then measuring potential energy absorption should provide a means of estimating the likelihood of pest damage.

In addition to temperature (energy), all three types of the turfgrass pests mentioned above are also dependent on moisture availability in at least one portion of their life cycle. Like temperature, measuring moisture availability can also be used to estimate the likelihood of pest activity, if it is understood what role moisture plays in the life cycle.

Temperature and Moisture

Although it is difficult to generalize about the roles that temperature and moisture play in common turfgrass pest biology, for the most part it can be said that moisture plays an 'enabling role' and temperature plays a 'driving role'.

Moisture enables the life processes to occur or continue because it often plays a
crucial role in the pest's life cycle stages, such as when a newly laid chinchbug egg absorbs moisture from the soil or thatch prior to hatching, or when prolonged leaf wetness periods induce fungal spore germination or enhance mycelial growth. Furthermore, temperature provides the energy required to drive the life cycle toward maturity, such as the progress of a scarab beetle through three instar grub stages, pupation, and adult emergence and activity.

In other words, as long as the minimum moisture requirements of a phenology are met, then progress of that species toward maturity is a function of the amount of energy absorbed.

Moisture

How moisture enables pest activity will vary significantly by species. For instance: Once a chinch bug egg has been laid in the soil or thatch at or near the soil's surface, the egg must absorb several times its own weight in water within first 48 to 72 hours or egg mortality rises significantly. Research has shown that grubs will migrate down into the soil profile as soils begin to dry. Several scarab beetle species may have the apparent ability to delay adult emergence from their pupal stage during dry soil periods. Spore germination for certain foliar diseases like Dreschlera Leaf Spot can only occur during prolonged leaf wetness periods and Brown Patch mycelial growth is enhanced by a dense turf canopy with a moist microenvironment.

The nature of the role that moisture plays for each species will vary. In the case of the chinch bug egg during the period after ovaposition and prior to hatching, the need for enough moisture to be absorbed is critical, but short-lived. In the case of many soilborne diseases like Pythium, Summer Patch, etc., the pathogen's moisture needs can be met by a broader range of values and over longer periods of time.

Temperature

How temperature drives the progress toward maturity is less species specific than moisture, but just as important.

Temperature or energy availability over a geographic area is often more consistent than moisture, since it generally follows large-scale trends such as rising temperatures during the transition between spring and summer or falling temperatures in the fall. Temperature is always there from one day to the next with incoming solar radiation providing the relatively consistent energy source that drives all life. Moisture, on the other hand, is far more a function of seemingly random events.

Energy — The Driving Force

An insect's life is predetermined, meaning that one life cycle stage must follow another and the insect's growth can only progress through this defined number of stages, often within a finite period of time.

Whether a species survives or not at a given location is ultimately a function of how much heat the insect can absorb during its various phenologies and how fast it can absorb that energy. If the total available accumulated annual heat (energy) at a location is not enough to complete the species' life cycle, then the chances are very high that the species will not survive in that location. If, however there is more then sufficient available energy to complete the insect's life cycle, than the likelihood that the species will survive at that location is greatly enhanced. If there is sufficient available energy to complete more than one generation per year, then the likelihood that an insect species will thrive is substantially increased.

If the total available energy of a geographic area is marginal or insufficient for the establishment of a viable population of a scarab beetle species with an annual life cycle (meaning all life cycle stages are completed in 365 days), then that species will not likely survive over the long-term. Even though there is not enough available energy for an annual scarab to survive, there may be enough available energy for a two-
or three-year life cycle scarab species to survive and thrive in the same area. This is an example of how the climatic conditions at a location can limit the number and types of pests species found in that area.

In a slightly different way, the long-term survivability of diseases is also determined by temperature. Some population of almost all of the common disease pathogens can be found at virtually any location at any time of the year. This 'survival' or 'background' population can survive various and prolonged adverse climatic conditions in resting bodies for several years. It is only when these survival populations are subjected to optimum growth conditions for a long enough period of time that host disease symptoms actually develop.

**Pest Population Dynamics**

Because of the seemingly rapid onset of many turfgrass diseases and some insects, turf managers often fail to realize that pathogens or insects have often been active for extended periods prior to the first appearance of damage.

This period of population building is actually very logical from a theoretical point of view. If, for instance, the total population of all of the plant disease pathogens that grew in area during one year were to survive the extended periods of adverse conditions, both summer and winter, that normally occur and germinate and produce an ever increasing population the following year, then the pathogen populations would have built up to the point that most vegetation in that area would most likely have been killed off. With the loss of most of the vegetation, all the animals that directly or indirectly depended on plants as the ultimate food source would also die.

This same principle applies to plant damaging insect pest populations. If all of the insects that were alive prior to the onset of prolonged periods of adverse climatic conditions survived and reproduced the following year, then all vegetation that those insects feed on would most likely cease to exist also.

Insects are subject to the same population buildup as diseases. It is common knowledge in cool season areas that only a small fraction of the previous year's chinch bug population survives over most winters. Once the spring temperatures rise above a minimum level, early overwintering chinch bug adult activity begins with a subsequent rapid buildup of actively feeding juveniles and adults often leading to visual damage at a later date.

The understanding of the mechanisms of this buildup in actively feeding chinch bugs has led to a management strategy that targets these overwintering adults just as they begin to become active. This early intervention strategy targeted at reducing the population of fertile adults is said to be so effective, that depending on the location (available energy), the species targeted, and the control product used, it can take one to three years for the population of chinch bugs to return to the precontrol application levels.

**How Climate & Pest Biology Interact**

The rebuilding of pathogen populations, following prolonged adverse climatic periods, from low background levels to those required to cause the onset of symptoms, is most likely to occur during a series of multiple short and long favorable climatic periods.

During any 365-day period, there are likely to be many short periods when suboptimal to optimal pathogen growth conditions are met. During these periods, sometimes as short as 72 hours, pathogens can become active and progress through a complete generation ending in an intermediate or long-term resting or survival stage.

Through a number of these short favorability periods, the pathogen populations can either stay stable or slowly build. If, however, as often happens, these short favorability periods are followed by extend-
ed favorability periods, then the disease’s ability to cycle through a complete generation in a relatively short period of time can lead to multiple generations and a building population or rise in so-called disease inoculum levels. Once a threshold of inoculum (which varies by pathogen species, host species, host susceptibility, and climatic conditions) has been reached, any additional periods of favorable weather, even if only marginally favorable, can rapidly lead to an exponential increase in internal disease damage and the onset of external visual symptoms on the host.

Following this scenario, it becomes evident that there can be prolonged periods of pathogen activity without the onset of significant internal effects or external visual symptoms on the host species. But once the pathogen population reaches a minimum threshold, any additional pathogen activity can lead to significant damage.

A direct example of this apparent delay between the onset of pathogen activity and the appearance of external visual symptoms can be seen in the results of aerial remote sensing of agricultural crops fields or golf course turf. When multispectrum infrared scanners are used to examine fields or areas of crops or turf, the resulting output will often show normally appearing plants with unseen internal damage or stress that only develops into external visual symptoms at a later date.

The same scenario applies to insects. Insects are often active before a so-called threshold population can produce significant visual symptoms. As with diseases, the threshold population that produces identifiable visual symptoms will depend on the pest species, the host species, the host condition and the climatic conditions.

**Measuring Activity Ranges**

Since temperature and moisture are the principle elements that determine climatic favorability for pest activity, it is necessary to establish a range of values for each.

There are two approaches to determining these ranges. The first method is use the most basic concept in applied climatology and take systematic measurements of these weather elements and correlate the resulting data to field observations of pest activity. The second is to use the same observation and correlation techniques, but done under controlled laboratory conditions.

In the case of insect activity, since moisture plays a secondary role to temperature, comparing the direct observations of insect activity and a quantitative measurement of temperature collected in the field works fairly well.

The most common measured method of relating insect activity to temperature is to use an accumulation measure called 'degree-days'. Degree-days are a means of measuring daily or hourly average temperature, which are then summed over a period of time to create a measure of accumulated heat (energy).

Degree-days are calculated by summing the daily high and low temperature (F or C), dividing that sum by two, and then subtracting a predetermined species-specific threshold number (usually 42 or 50 degrees F). The result, if it is a positive number, is added to the sum of all of the previous positive numbers calculated during the observation period to create ‘accumulated degree-days’. If the result of the daily DD calculation is a negative number, then it is ignored.

This accumulated degree-day number is then correlated to observed insect activity or phenology change by beginning the DD accumulation at a repeatable point, such as egg hatch. In the case of the Hairy Chinch bug, early overwintering adult activity (the point each spring when overwintering adult activity starts) is expected to begin when the accumulated degree-day YTD total reaches ~ 145 DD (base 50 degrees) at a turf location. (Note: The phrase base 50 is used to denote the accumulating threshold of 50 degrees F.)

The nice thing about using YTD degree-day accumulations to estimate the current phenological stage and likely early activity period of Hairy Chinch bugs, or most other turfgrass damaging insects for that matter, is that the likelihood of damage from early insect activity is very small. It's small, because the amount of accumulated heat/early activity is low and the initial insect population is also usually low. Additionally, the first estimate of insect activity
of the season allows for sufficient time to scout suspected or historic activity locations to monitor whether there is actually any activity.

The negative part about using DD accumulations for estimating insect activity is that the numbers are fairly squishy approximations, usually based on correlations done at only a few locations. And they do not take localized microclimate conditions, like a southern exposure or thin turf cover, that would increase local YTD DD numbers into account and they ignore any accumulated heat below the observation threshold temperature level.

The setting of observational or accumulating thresholds is based on the assumption that the majority of an insect's progress toward maturity occurs simultaneously with observable activity. Unfortunately, work done on an insect's metabolic mechanisms has shown that a substantial amount of internal metabolic activity may be taking place at temperatures between freezing and the threshold value in the absence of observable activity, enough metabolic activity that this below threshold energy may be the cause of the substantial variances seen between estimated activity and actual observed activity.

Because of this assumption of no significant progress toward maturity at temperatures below threshold and that observations were done in the field setting subject to several uncontrollable variables as well as the observation multiple insect phenotypes, estimations of specific insect activity using DD accumulations will always be just approximations.

The timing or implementation of insect control activities should always be subject to change from those created using DD calculations when any local or special conditions related to potential insect populations occur. Conditions, like milder than normal winter conditions that allow a larger than normal percentage of the previous year's insect population to survive, should always prompt earlier than normal scouting, often considerably before the accumulated DD estimate of initial activity.

In the case of diseases, most of the correlations between climate and pathogen activity are based on a combination of laboratory created data for temperature and both laboratory and field level observations for moisture.

A considerable amount of temperature work has been done on pathogen growth on auger plates. In these cases, pathogen growth on a growing media is checked for the temperature at which it is occurring and often the speed at which the colony growth progresses.

For instance, laboratory growth plate tests have shown that the fastest growing common turfgrass pathogen is Pythium at something over 3 cm per day and that Brown Patch probably has one of the shorter intergenerational times of ~72 hours, beginning to end.

Moisture studies for diseases are usually done in two ways - either as growth chamber studies on inoculated hosts or through direct observation field studies. These techniques are often used to determine optimum leaf wetness for pathogen germination and/or mycelial growth.

As effective as they are at measuring the ranges of both temperature and moisture in pathogen activity, there are several shortcomings in the use of these techniques.

First, because many of these studies are done in the controlled environment of a growth chamber, the resulting data may overestimate the speed and intensity of field level symptom onset because of the studies uniformity and optimization of conditions — something that is rare in the field. Second, in many cases the studies involve the practice of inoculating hosts with an unnaturally high concentration of pathogens to precipitate symptom onset thereby saving time.

Finally, many of these laboratory growth and plant greenhouse tests are done in the absence of any naturally occurring competitive or antagonistic pathogens that could change the intensity and speed of symptom onset in the field.

Within the obvious limitations of the work done to date, the establishment of
workable ranges for minimum and optimum pest activity can provide a foundation for the beginning of a substantial look at how changes in each parameter are liable to effect pest activity.

How Precipitation Changes Affect Pest Activity?

Direct quantitative field measurements of the effects of climate parameter changes on pest activity are rare and may not actually exist for turfgrasses. However, there is a substantial amount of raw observational data that could be translated to describe these cause-and-effect relationships, if enough time and resources were available, but lacking both, what follows are several model predictions that can serve as a means to illustrate these relationships.

An analysis of the effects of the addition of 1.25 inches of supplemental irrigation per week during the summer months on the estimated annual Anthracnose favorability at five U.S. locations is very instructive. Table 1 shows how the use of weekly supplemental irrigation during summer months added to the average climate over the last 30 years causes a change in the average annual Anthracnose favorability.

In the case of Tucson, the addition of supplemental irrigation raised the average estimated favorability to a numerical value of 18 from 0, in Atlanta the average rose from 38 to 39, in Chicago from 19 to 26, in Dallas from 21 to 51 and in San Francisco from 6 to 28.

(Note: In the above example, the average annual favorability means that in the case of Atlanta, for 38 or 39 percent of the year, climatic conditions are favorable to Anthracnose pathogen activity.)

This information illustrates two concepts. First, the addition of added moisture to turfgrass can dramatically increase pathogen activity favorability in areas that normally have low rainfall (see Tucson and Dallas).

Second, added moisture in areas of moderate to high rainfall may not increase favorability as much.

In fact, added moisture during periods of suboptimum temperatures may have no additive effect on pathogen activity at all. So added moisture requires the proper range of temperatures for long periods to have a substantial effect on the average favorability over the course of a year.

For added moisture to actually increase favorability over what might be expected under normal conditions, it must come at a time of optimal temperatures but suboptimal moisture. Added moisture to a wet environment will usually not increase the level of pathogen activity: Optimum moisture conditions means just that.

What added moisture can do in this circumstance is to prolong a favorability period, and that can lead to increased disease inoculum, which can lead to increased symptom expression at a later time.

Adding moisture over and above normal rainfall during insect activity periods may have the effect of reducing insect activity. First, air temperatures during rainfall/irrigation events are usually lower causing less available heat. Second, added rainfall/moisture at the wrong phenology stage can actually increase insect mortality, such as soil inhabiting insects suffering from lack of

<table>
<thead>
<tr>
<th>Location</th>
<th>without irrigation</th>
<th>with irrigation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tucson AZ</td>
<td>0</td>
<td>18</td>
</tr>
<tr>
<td>Atlanta GA</td>
<td>38</td>
<td>39</td>
</tr>
<tr>
<td>Chicago IL</td>
<td>19</td>
<td>26</td>
</tr>
<tr>
<td>Dallas TX</td>
<td>21</td>
<td>51</td>
</tr>
<tr>
<td>San Francisco CA</td>
<td>6</td>
<td>28</td>
</tr>
</tbody>
</table>
available oxygen in saturated soil or causing an increased population of naturally occurring insect predacious microbes.

Temperature Fluctuations

Table 2 shows a comparative analysis of the occurrence dates of both the 450 and 1500 DD totals for the eight climate districts of North Carolina for 1997 versus the 1961 - 1990 expected normal average.

Early in 1997 was one of the warmest springtimes in this century, and the occurrence dates of the 450 DD and 1500 DD thresholds showed a substantial change from the expected. The 450 DD threshold is the expected date for hatching of the first new generation of Chinch bugs in North Carolina.

The average date for the eight districts was 14 days sooner than would normally be expected, with two of the districts reaching the threshold more than 30 days early.

The average for the 1500DD threshold or the expected hatch date of the second generation of Chinch bugs, was five days sooner than expected, with two districts more than ten days early as temperatures in the early summer were actually below normal.

Whether this early probable chinchbug activity actually led to visible damage is a function of the size of the overwintering adult population, the strategy used to determine control product application timing and the overall condition of the hosts.

But it is safe to assume that the use of early intervention control strategies, based on the historic average first expected occurrence calendar dates, particularly when using short duration control materials, were not applied at the optimal times.

Summary

Many of the elements of climatic conditions and plant pest activity have been shown in a very indirect way in turfgrass research. However, since little actual research has been done to describe these connections, computer modeling can produce a significant amount of information. Modeling can provide managers and researchers with good, practical information about how climatic conditions can affect both pest species and host plants.

Until the exact nature of connection between climatic conditions and pest activity can be more thoroughly researched, turfgrass managers will have to continue to rely on their observational skills to judge when, how, and with what actions or materials to control the many species of insect, disease, and weed pest that damage or disrupt their managed sites.

### Table 2. The effects of 1997 warm spring temperatures on the likely timing of 1st and 2nd generation egg hatch of Chinch bugs in North Carolina.

<table>
<thead>
<tr>
<th>Location</th>
<th>1st Hatch Normal DD</th>
<th>1st Hatch 1997 DD</th>
<th>2nd Hatch Normal DD</th>
<th>2nd Hatch 1997 DD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal DD</td>
<td>450 DD</td>
<td>May 20</td>
<td>1500 DD</td>
<td>Jul 16</td>
</tr>
<tr>
<td>CD # 1</td>
<td>May 20</td>
<td>May 17</td>
<td>Jul 20</td>
<td></td>
</tr>
<tr>
<td>CD # 2</td>
<td>Jun 7</td>
<td>Jun 2</td>
<td>Aug 3</td>
<td>Jul 28</td>
</tr>
<tr>
<td>CD # 3</td>
<td>May 17</td>
<td>May 2</td>
<td>Jul 5</td>
<td>Jun 30</td>
</tr>
<tr>
<td>CD # 4</td>
<td>Apr 12</td>
<td>May 2</td>
<td>Jul 1</td>
<td>Jun 25</td>
</tr>
<tr>
<td>CD # 5</td>
<td>Apr 5</td>
<td>Apr 5</td>
<td>Jun 26</td>
<td>Jun 11</td>
</tr>
<tr>
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<td>Apr 17</td>
<td>Jun 21</td>
<td>Jun 10</td>
</tr>
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<td>CD # 7</td>
<td>May 4</td>
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<td>Jun 22</td>
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</tr>
<tr>
<td>CD # 8</td>
<td>May 12</td>
<td>Apr 27</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Sorry Folks, I Just Had to...You Know

We can control some things in life and other we can’t. One of those we can’t is age. Sue Gibson let me write this column to explain that TurfGRASS Trends is a human undertaking and this particular human ran into more roadblocks than he could overcome. I apologize and hope you will forgive me and Advanstar for being late.

I got into this business largely because my parents derived tremendous joy from their landscape. Their pleasure touched me, and I eventually became a horticultural journalist. Now that they are facing the final lap of their lives and I have benefited from 30 years of a career by following their lead, I can only say, they come first. Every few hours I talk with my mother, who is in a hospital bed 2,000 miles from my office. I’m no momma’s boy, but I will excuse myself from any call to talk to her. There isn’t much I can do, but I have only one priority for the time being — returning the favor of their devotion and love.

We all run this course one day, some sooner than others. Even so, none of us seem prepared. My guide is doing what I hope my two children will do for me when I am in the same situation.

In a few weeks, my son will start his first job as an assistant superintendent at a golf course on the amazing central coast of California after graduating from Cal Poly, San Luis Obispo. That would not have happened if my dad had not taken me to Shaw’s Garden in St. Louis year after year, season after season. My daughter is working on becoming a professional photographer and graphic artist, primarily from her joy of landscape photography. She and her brother are as close as fraternal twins because of their shared admiration for nature. That came through me, not from me, and it wasn’t one sided at all. Their mom is the artist, the one who taught them to believe in themselves, pick a career in which they make a difference and to prove it through education. I hope you understand.

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