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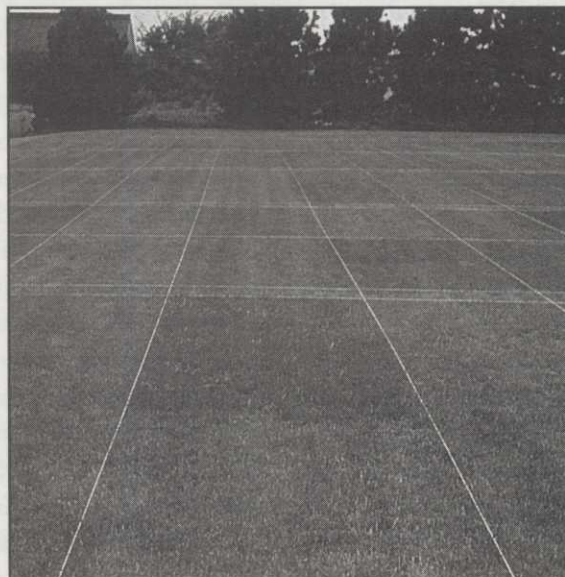
Nitrogen Fertilization's Effect On Turfgrass Disease Injury

by John E. Watkins
University of Nebraska, Lincoln

Fungi cause most of the diseases that occur on cool season turfgrasses in America. These fungal pathogens respond to both the environment and to cultural practices. Environment is the overriding factor that determines the seriousness of a disease outbreak. When environmental conditions favor the pathogen to the detriment of the host, disease occurs.

Turf managers must continually respond to changing environmental conditions in their battle against disease. Those who respond too slowly often watch in frustration as their turf dies despite their rescue efforts. However, managers that understand the relationship between the environment, cultural practices and disease development often can react quickly enough to prevent serious turf loss. Part of being able to modify cultural practices to discourage disease is knowing something about which, when, where and why diseases occur. One aspect of turf disease management is nitrogen fertilization.

It is impossible to generalize about the effects of fertilizers on all turfgrass diseases because of the differences in hosts and the variable characteristics of pathogens. Fertilizers do not directly affect the pathogen, but rather alter the host plant's metabolism or morphology to make it more or less susceptible or resistant to attack by pathogens.



Severe brown patch injury of perennial ryegrass plot (foreground) was caused by a low rate of N, 2 lbs./1,000 ft.² Photo: J. Watkins.

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A good fertility program stimulates a deep and extensive root system, controls shoot growth and provides recuperative potential from injury. During the active growth period, fertility programs should try to achieve a 3-1-2 balance of nitrogen-phosphorous-potassium. Many turfgrass diseases are more severe when this ratio becomes out of balance, particularly when the nitrogen component is excessively high or low. Excess nitrogen prolongs vegetative shoot growth. The plants are more succulent and have thinner cell walls, which are more easily penetrated by fungal hyphae. High nitrate levels ($\text{NO}_3\text{-N}$) cause leaves to exude greater amounts of nitrogen-rich glutamine and carbohydrates, both of which may enhance fungal spore germination.

Turfgrass managers are well aware of dangers of over or under fertilizing turf, and that the fine line between adequate and too much or too little fertilizer is not always clear. That range of fertility at which host susceptibility is minimized depends on the interaction of the environment with the host and the pathogen. High nitrogen may enhance disease injury on one host, but on a different grass host, it may promote recovery of the grass so the turf shows less injury in the presence of the same disease. Brown patch is a good example. Observations in our research at the University of Nebraska-Lincoln showed that 6 to 8 lb. of actual N/1000 ft²/season caused more severe brown patch development in tall fescue than 2 or 4 lb. of actual N/1000 ft². However, perennial ryegrass plots fertilized with 8 lb. N/1000 ft²/season showed less brown patch injury than when fertilized at lower N rates, and they recovered more quickly from infection. In general, excessive nitrogen encourages the development

of *Rhizoctonia* brown patch (on some hosts), *Bipolaris* and *Drechslera* leaf spots, summer patch, stripe smut, pink and gray snow molds and *Pythium* blight. Stem and crown rusts, anthracnose, dollar spot and red thread are often more severe when nitrogen is deficient. These nutrient-disease interactions are well documented in the scientific and technical literature. At the University of Nebraska-Lincoln, we have studied the effect of nitrogen rate on the development of brown patch on tall fescue and perennial ryegrass, dollar spot on bentgrass and crown rust on perennial ryegrass.

Nitrogen Effect On Brown Patch Injury

Brown patch, caused by the fungus *Rhizoctonia solani*, is a serious pathogen on all commonly cultivated turfgrasses. Symptoms vary depending on turfgrass species and mowing height. On home lawns, golf course fairways and similar turfs, two general types of symptoms can appear either simultaneously or separately. Field expression is the presence of patches or roughly circular rings of dead and dying grass surrounding areas of dead turf. These patches may be up to 2 feet in diameter. The individual lesions on leaf blades are distinctive and appear as long, irregularly shaped, ash-gray spots surrounded by dark brown borders. Symptoms on bentgrass greens appear as straw-colored patches that range in diameter from 3 inches to 3 feet. When the turf is wet, a narrow dark gray ring or arc composed of fungal mycelium and wilted grass blades mark the advancing edge of the patch. This halo is referred to as the "smoke ring" and is best observed in early morning.

Nitrogen is the key nutrient in plant nutrition and must be available to the grass at a constant uniform rate. The turfgrass manager should determine the turf's nutrient needs based on soil tests and the intensity of management desired. For example, golf course turf is an intensively managed grass community in which considerable stress is placed on its plants. Along with the intense management comes increased nitrogen use and a greater need for disease management. Golf course superintendents can use their nitrogen fertility program to advantage in reducing brown patch injury by promoting rapid recovery of infected turf.

In practice, plant growth can be controlled more precisely with several small nitrogen applications during the growing season than with an initial heavy application of a slow-release nitrogen fertilizer in the spring. In regions of North America where turf goes dormant in late fall, a fertilizer application in October, November or December is practical and often advantageous when the surface soil is frozen. There is little loss of soluble nutrients during winter, and they are in a position to aid in early recovery from winter injury and damage from snow molds. The concept of fall fertilization dates back to the 1930s. This approach works well for fertilizers such as urea, IBDU or ammonium nitrate which are not highly dependent on microbial activity or elevated temperature for nitrogen release. When applied at temperatures below 60 F., root growth is favored over shoot growth. This encourages the turf to photosynthesize long into the season, resulting in better fall and winter color, earlier spring green-up and higher levels of stored carbohydrates.

A study at the University of Nebraska-Lincoln showed that nitrogen levels of 6 and 8 lb. of actual N/1000 ft²/season caused significantly greater brown patch injury than 4 or less lb of nitrogen on a Rebel tall fescue turf (Table 1).

As expected, brown patch was more severe at higher nitrogen levels. The turf in those plots receiving no nitrogen was chlorotic, lacked vigor and was not aesthetically acceptable but contained little disease. However, at 2 lb. of nitrogen, the turf quality was acceptable while the disease injury remained low. At 4 lb. of applied nitrogen disease injury was intermediate. The 6 and 8 lb. nitrogen rates produced mod-

Table 1. Effects of nitrogen fertilizer rates on brown patch injury to Rebel tall fescue, 1990.

Lb. N/1000 ft ² /season	Visual brown patch ratings*		
	July 10	July 23	Sept. 4
0	2.0	1.3	1.0
2	3.0	3.3	1.3
4	5.0	4.8	2.8
6	5.5	6.5	4.8
8	6.3	6.5	5.3

*Brown patch injury was rated as 0 = no visible injury, 5 = 45-55% of the plot with injury, and 10 = 100% of the plot with injury. Research plots were inoculated annually in early June with *R. solani* cultured on sterilized oat seed to promote disease development. Liquid urea, 46-0-0 was applied monthly from April through October.

erately severe brown patch injury. At these higher nitrogen rates the turf was more vigorous, but the disease injury remained higher later into the season.

A similar study compared the effect of nitrogen rates on brown patch injury in perennial ryegrass. Preliminary results in Table 2 show that as nitrogen rate increased from 0 to 8 lb., brown patch injury decreased. This trend was consistent between the two cultivars as well as for the blend. The color and quality of the turf fertilized at 4 and 8 lb. N was visibly better than that fertilized at 0 and 2 lb. N.

We had expected just the opposite, i.e. higher N promoting/greater disease injury. Plots receiving little N become thin, recovered poorly from disease injury and developed a weed problem. Those receiving the higher N rates recovered quickly from disease injury and maintained good verdure, color and quality. The key to why there was less brown patch injury in the high N plots may have been that they were fertilized monthly for five months. Essentially, they were spoon fed and did not receive a large amount of fertilizer at any given time. This study is a long-term project scheduled for completion in 1996 or 1997.

Suppression of Dollar Spot Injury On Bentgrass With Nitrogen

In 1993 and 1994, dollar spot was one of the most active turf diseases in the Great Plains. Abundant rainfall created ideal conditions for infection by *Sclerotinia homeocarpa*, the causal fungus of dollar spot.

Table 2. Effect of nitrogen and cultivar blend on brown patch injury of perennial ryegrass, 1993.

Lb. N/1000 ft ² /season	<i>Brown patch injury</i> *					
	Manhattan		Manhattan II		Manhattan/Manhattan II Blend	
	Aug. 2	Aug. 24	Aug. 2	Aug. 24	Aug. 2	Aug. 24
0	6.1	7.2	4.9	6.0	4.4	6.3
2	6.2	7.2	4.5	5.9	5.6	6.1
4	5.7	6.4	3.5	4.8	5.0	5.7
8	5.4	5.4	4.2	3.2	3.9	3.7

*Brown patch injury was rated as 0 = no visible injury, 5 = 45-55% of the plot with injury, and 10 = 100% of the plot with injury. Research plots were inoculated annually in early June with *R. solani* cultured on sterilized oat seed to promote disease development. Fertilizer (urea 46-0-0 or sulfur-coated urea-32-0-0) was applied monthly from April through October.

Dollar spot symptoms vary depending on turfgrass species, mowing height and nutrition level. Under close mowing, symptoms appear as small, round, straw-colored spots roughly the size of a silver dollar. These can become so numerous on bentgrass that large areas will appear blighted. Turf maintained at taller heights shows a mottled, straw-colored pattern made up of 4- to 8-inch patches of blighted turf. Individual grass blades develop hourglass-shaped, bleached lesions surrounded by a reddish-tan border. Symptom development and turf injury are enhanced when nitrogen is deficient.

A study initiated in 1993 and continued through 1995 at the University of Nebraska-Lincoln showed that higher rates of fertilizer nitrogen suppressed dollar spot injury in bentgrass (Table 3). This study also compared quick-release urea (46-0-0) with slow-release, sulfur-coated urea (32-0-0) at 0, 2, 4, 6 and 8 lb. N/1000 ft²/year (results not shown). Fertilizer treatments were started in

May and continued monthly until the respective amounts of nitrogen had been applied.

Dollar spot injury was effectively suppressed at the 6 and 8 lb. nitrogen rates. There was no interaction between nitrogen rates and the slow- or fast-release carrier; however, by mid-season, the quick-release urea was giving greater disease suppression than the sulfur-coated urea. This was probably due to more nitrogen being available at a given time with the quick-release carrier than with the slow-release. Urea at the 6 and 8 lb. rate significantly burned the turf. Some fertilizer injury also occurred with the slow-release, sulfur-coated urea at 8 lb., but it in no way compared to the injury caused by urea at that equivalent high rate.

Based on these results, it appears the use of sulfur-coated urea at 4 to 6 lb. N/1000 ft² can suppress dollar spot development. At these rates, bentgrass is able to recover quickly from dollar spot injury.

Table 3. Effect of nitrogen rate on dollar spot injury to bentgrass turf, 1993 and 1994.

Lb. N/1000 ft ² per season	<i>Visual dollar spot ratings</i> *			
	1993		1994	
	June 30	Aug. 24	June 30	Aug. 24
0	1.8	7.4	3.0	7.5
2	1.3	5.3	2.6	6.3
4	1.3	4.3	1.1	4.1
6	1.5	2.6	0.95	1.2
8	1.0	1.0	0.85	0.85

*Dollar spot injury was rated as 0 = no visible injury, 5 = 45-55% of the plot with injury and 10 = 100% of the plot with injury.

Less Crown Rust at High Nitrogen Rates

As research plant pathologists we are opportunists, and when an unexpected disease outbreak occurs in a research plot, we take advantage of it. Such was the situation in 1994, when crown rust caused by *Puccinia coronata* became endemic in our brown patch-perennial ryegrass-N rate plots discussed previously in Table 2 of this article. Warm days and cool nights created long periods of dew which were ideal for crown rust development. Crown

Table 4. Effect of nitrogen rate and cultivar on crown rust development in perennial ryegrass, 1994.

Lb N/1000 ft ² /season rust*	Manhattan		rust	Manhattan II		rust	Manhattan/Manhattan II Blend	
	Crown quality**	Turf		Crown quality	Turf		Crown quality	Turf
0	7.6	4.3		5.1	6.4		4.7	5.5
2	7.1	4.6		4.1	7.0		3.3	6.9
4	6.7	5.1		2.6	7.4		4.5	6.8
8	3.6	6.6		1.4	8.4		2.1	8.0

*Crown rust was rated on a scale of 0-10 with 0 = no disease, 5 = moderate disease and 10 = severe disease.

**Turf quality was rated on a 1-10 scale with 10 being the highest quality.

rust was detected in late August, developed rapidly through September, becoming severe by early October. Table 4 shows that crown rust was effectively suppressed at the 8 lb. N rate on both Manhattan and Manhattan II and on the blend. Turf quality was also highest at the 8 lb. N rate. These results were not surprising since rust diseases respond to nitrogen fertilization. Slow growing, undernourished turf is prone to severe rust injury, while actively growing turf that is mowed regularly recovers rapidly from rust injury.

Quantity, Timing of Nitrogen Applications Impact Disease Injury

As shown by these examples (brown patch, dollar spot and crown rust), the quantity and timing of fertilizer-N applications have a major impact on turfgrass disease injury. Overstimulated turfs may divert plant defense chemicals to nitrogen metabolism and leaf production, thus opening the way for infection. Nutrient-deficient turfs lack vigor and

this allows the fungus to colonize new tissues. The recuperative potential of nitrogen-starved turf is poor. A balanced fertility program based on frequent light applications of nitrogen during the growing season should discourage disease infection and will provide the energy needed for recovery from any disease injury that occurs. Through prudent management practices, turfgrass managers should be able to integrate their fertilizer program with the use of fungicides and cultural practices to keep disease injury to a minimum.

Dr. John E. Watkins is a professor at the University of Nebraska-Lincoln in the Department of Plant Pathology. He has degrees in microbiology from the University of Wyoming and plant pathology from North Dakota State University. Dr. Watkins is active in extension and research on integrated management approaches for disease control in turfgrass and small grains. His current emphasis in turfgrass research is developing an integrated nitrogen fertilization-fungicide application program for control of dollar spot on creeping bentgrass.

Dr. Richard J. Hull, author of the following article, entitled *Nitrogen Usage by Turfgrass*, is the science advisor of *TurfGrass Trends*. Hull, a professor of Plant Science and Chairman of the Plant Sciences Department at the University of Rhode Island, was recently named chairman of the C-5 Division (Turfgrass Sciences) of the Crop Science Society of America. He has degrees in agronomy and botany from the University of Rhode Island and the University of California, Davis. His research has concentrated on nutrient use efficiency and photosynthate partitioning in turfgrasses and woody ornamental plants. He teaches applied plant physiology and plant nutrition. Dr. Hull is a frequent contributor to *TurfGrass Trends*.

Nitrogen Usage by Turfgrasses

What exactly do turfgrasses do with nitrogen and why do they use so much of it?

Richard J. Hull

University of Rhode Island

Nitrogen is the most abundant mineral nutrient in turfgrasses. The nitrogen content of turfgrass leaves ranges from 3 to 5% of dry tissue. It is the nutrient element normally applied in the greatest amount as fertilizer, and its deficiency symptoms are the first to appear when grass is not fertilized. The turf manager spends more on nitrogen than on all other plant nutrients combined. Most natural plant communities suffer from a chronic nitrogen deficiency.

Since providing nitrogen is an important part of turfgrass management, it might be helpful for the turf professional to understand why nitrogen is so important for turfgrass health and why it is needed in such large amounts. This article will consider the function of nitrogen in plants and suggest some management strategies which result from understanding these functions. Earlier articles in *TurfGrass Trends* (Hull 1994; 1995a; 1995b) have discussed the fate of nitrogen in the soil, conditions influencing the leaching of nitrate from turf and mechanisms by which grass roots absorb nitrate. None of those discussions considered what nitrogen does inside the plant or how efficiently it is utilized.

Nitrogen and Proteins

If asked what nitrogen does for plants and animals, most people would respond that nitrogen is part of proteins. Since everyone knows that proteins are important for living cells, then nitrogen must also be important. Although that is true, it tells you absolutely nothing about the role of nitrogen in living cells or even why it is present in proteins.

To understand the role of nitrogen, you need to know something about how living organisms work; what life is all about. Put simply, life is a series of controlled chemical reactions which enable an organism to grow and reproduce. These chemical reactions involve the burning (oxidation) of fuel molecules (food) to produce energy and chemical components by which cells grow and even make new cells. In animals, these fuel molecules are ingested as foods, while in plants, they are made from carbon dioxide (CO₂), water (H₂O) and sunlight through photosynthesis. Thus, life in its most elemental terms is a sequence of chemical reactions occurring in living cells by which food molecules are degraded to produce the energy and stuff to make more living cells.

The problem with this idea of life is that most of the chemical reactions which support the growth and reproduction of cells occur very slowly. This is so because chemical reactions among organic molecules only take place when the reactant molecules collide with each other in just the right way so the chemical reaction can occur. Only one random collision in several million will cause an appropriate reaction. If life depended on such random chemical events, growth would be a very slow process. We might live for centuries, but we would be relatively inert.

Life exists as we know it because the chemistry of life reactions is speeded up by the presence of catalysts. In living cells, these catalysts are proteins and are called enzymes. These catalytic proteins act by binding chemical reactants and bringing them together in exactly the proper orientation so they will react to produce new chemicals (products).

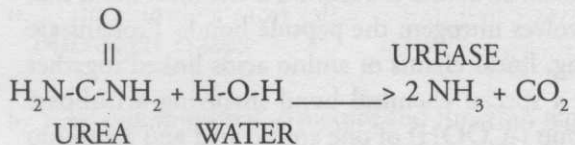
How enzymes work can be illustrated by the lock and key analogy. If you place a padlock and its key in a paper bag and shake the bag vigorously, how long would you have to shake that bag before the key entered the lock? The key obviously can fit into the lock, but would it do so by simply shaking the key and lock together? For this to happen, the key and lock would have to collide with each other in exactly the right way and with sufficient force for the key to enter the lock. To occur by random collisions, this might take a day, a month or years. It certainly would not happen quickly.

Now, if you took the key from the bag in your right hand and inserted it into the lock which you held in your left hand, you would be doing exactly what an enzyme catalyst does. An enzyme binds two chemical reactants and brings them together in exactly the right way so they will react with each other. You could fit thousands of keys in their locks during the time it would take just one key to enter its lock by shaking it in a bag. Thus, a chemical reaction occurs much more quickly in the presence of a catalyst than in its absence.

Two other important features of catalysts are illustrated by this story. The key must be able to fit the

lock for you to join them together. So too, a chemical reaction must be possible and energetically favored for a catalyst to make it go quickly. Also, in the process of fitting the key in its lock, you were in no way changed or consumed. Likewise, when a catalyst facilitates a chemical reaction, it is not permanently changed or used up. Therefore, one catalyst molecule can make a lot of chemical reactant molecules react to form chemical products. A little catalyst goes a long way.

As an example, the enzyme urease is a protein which binds the common nitrogen fertilizer urea and water bringing them together in just the right way so that a water molecule combines with a urea molecule forming an unstable urea-water hydride which degrades spontaneously to produce carbon dioxide and ammonia (NH₃).



This reaction occurs on grass leaves, in thatch and in the soil whenever urea fertilizer is applied to turf. Without urease present, urea would be relatively

Terms to Know

Acid - A chemical capable of releasing a hydrogen ion (proton) to solution. Organic acids are carboxyl groups and bound hydroxides capable of releasing a hydrogen. Metal cation that can form a covalent bonds with a base.

Base - A chemical species capable of accepting a hydrogen (proton). Organic bases contain a nitrogen which can bind hydrogen to form a hydrogen bond or become a cation. It can also form covalent bonds with metal cations.

Catalyst - A chemical species which alters the rate of a chemical reaction. Most catalysts increase reaction rates by increasing the probability of the reaction occurring.

Chlorosis - Yellow coloration of a leaf resulting from the destruction or loss of chlorophyll. Symptom of several mineral nutrient deficiencies, especially nitrogen and iron.

Enzyme - A protein capable of serving as a catalyst to a chemical reaction. Most biochemical reactions are catalyzed by a specific enzyme.

Nucleic Acid - A large linear molecule composed of ribose or deoxyribose sugars linked together with phosphoric acid units. Each sugar is bound to a purine or pyrimidine base and these in turn bind two such molecules together via hydrogen bonds forming a double helix. DNA and RNA are the most common nucleic acids in plants.

Protein - A linear chain of amino acids linked together by peptide bonds. A polypeptide.

inert and a poor source of nitrogen. Plant cells can utilize NH_3 but not urea. In fact, urea is a waste product in animal metabolism.

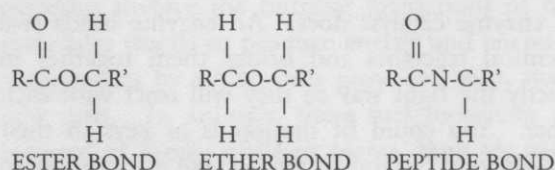
It is now generally recognized that most proteins in plants are enzymes and catalyze some biochemical reaction or facilitate some process, such as transport across a membrane. We understand the exact catalytic function of relatively few proteins present in plants but most probably carry out some catalytic role.

The Peptide Bond

At this point, you might be wondering what all this discussion of protein catalysts has to do with the function of nitrogen. The answer is simple; protein structure is based on a chemical bond that involves nitrogen: the peptide bond. Proteins are long, linear chains of amino acids linked together by a special chemical bond involving a carboxyl group ($-\text{COOH}$) of one amino acid and an amino group ($-\text{NH}_2$) of another. Such a peptide bond between the amino acids alanine and serine is shown in Fig. 1. A water molecule composed of the $-\text{OH}$ from the carboxyl group of alanine and a $-\text{H}$ from the amino group of serine is withdrawn,

forming a peptide bond between the terminal carboxyl carbon of alanine and the amino nitrogen on the number two carbon of serine. The actual process for making a peptide bond is much more involved than shown here, but this is the basic idea.

Peptide bonds link together the twenty different amino acids found in proteins, producing a long polypeptide chain which can consist of more than one thousand amino acids. The peptide bond linkage between carbon and nitrogen is more constrained in its rotation than an ester or ether bond between carbon and oxygen would be (Clarkson & Hanson 1980).



This produces a more predictable planar structure, causing the unbonded ends of each amino acid to project from the axis produced by the chain of peptide bonds and giving to the protein a distinct structure and chemical properties characteristic of the amino acids involved (Fig. 2).

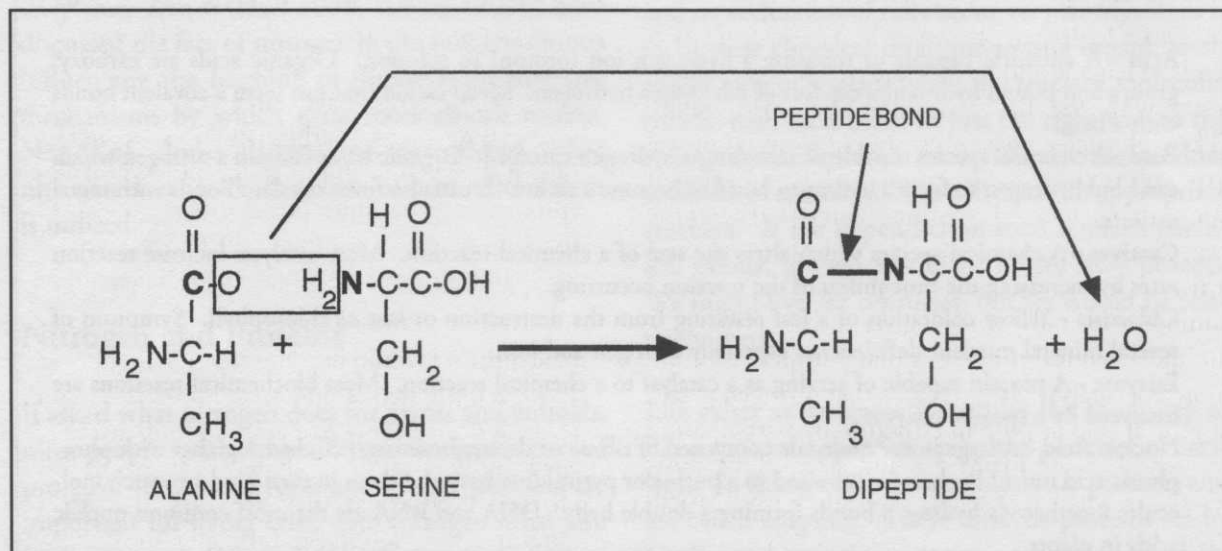


Figure 1. Formation of a peptide bond between two amino acids, alanine and serine, by removing a water molecule, thereby producing a dipeptide.

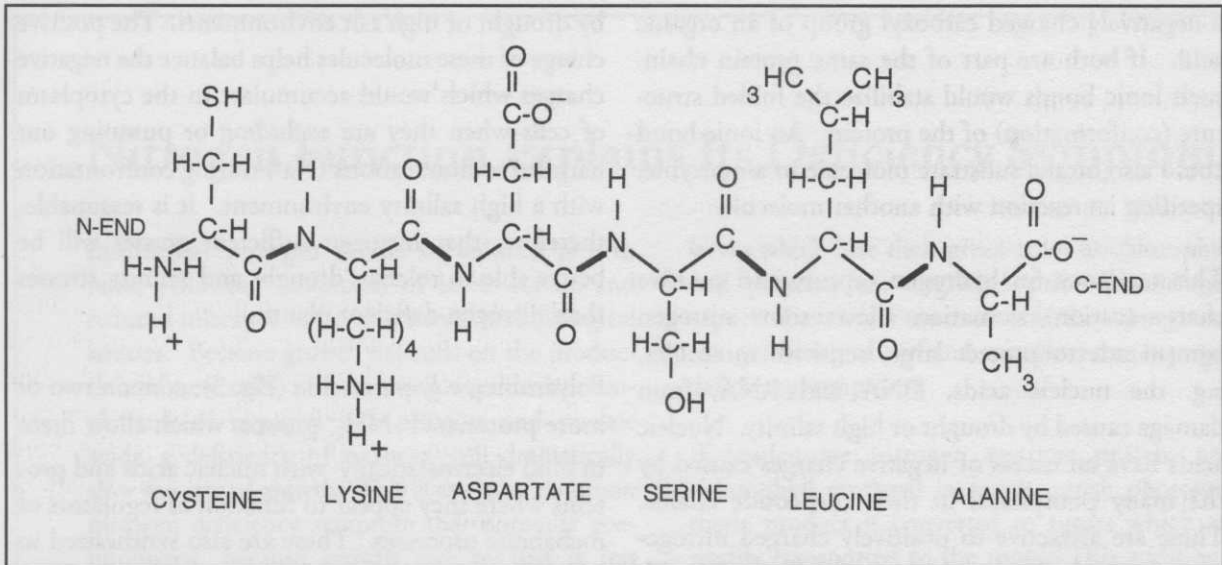
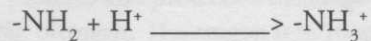


Figure 2. A peptide chain composed of six amino acids exhibiting the various chemical properties of proteins. The bold linkages are peptide bonds. The nitrogen and carbon ends are identified.

These amino acids provide the sites where chemical substrates can bind to the protein when the protein is acting as a catalyst. The -SH group of cysteine and -OH group of serine (Fig. 2) are polar and can share their hydrogens with a -C=O group of a substrate to form a hydrogen bond with that chemical and position it for reactivity. The carboxyl group of aspartate has a negative charge which can form an ionic bond with a chemical having a positive charge. Lysine is an organic base and has a positive charge when its terminal -NH₂ accepts a hydrogen (see below). The hydrocarbon ends of leucine and alanine are nonpolar and associate with the lipids of biological membranes. Through such associations, proteins can become a part of cellular membranes. The sequence of amino acids in a polypeptide chain determines the way the chain folds on itself (conformation), and this, in turn, dictates the position of reactive amino acid tails which gives each protein its unique chemical properties that permit it to function as a catalyst. It is obvious that nitrogen plays a central role in protein structure by participating in peptide bond formation and establishing positive sites when hydrogens associate with nitrogenous bases.

Nitrogen Bases

Its role in protein structure and function represents a major part of nitrogen's essentiality in all living cells. The presence of amino nitrogen groups (-NH₂) in a molecule allows it to function as a base. These bases have an unshared electron pair, which permits them to bind a free hydrogen and in so doing acquire a positive charge (electropositive group).



All amino acids have such an amino group attached to their #2 carbon, and at a normal physiological pH, this amino group binds a hydrogen (proton) and becomes positive (a cation). When forming a peptide bond as part of a protein, the -NH₂ group on the #2 C has its unshared electrons involved in a covalent bond so it is no longer free to acquire a H⁺ and become cationic (Figs. 1 & 2). However, the two amino acids lysine and arginine (Figs. 2 & 3) have a free amino group attached to their terminal ends, and these can be positively charged even when they are part of a protein. Such an -NH₃⁺ group can form an ionic bond with

a negatively charged carboxyl group of an organic acid. If both are part of the same protein chain, such ionic bonds would stabilize the folded structure (conformation) of the protein. An ionic bond could also bind a substrate molecule to an enzyme, speeding its reaction with another molecule.

This tendency for hydrogen capture and positive charge (cation) formation allows some nitrogen compounds to protect large, sensitive molecules, e.g. the nucleic acids, DNA and RNA, from damage caused by drought or high salinity. Nucleic acids have an excess of negative charges caused by the many phosphates in their nucleotide chains. These are attractive to positively charged nitrogenous bases and form electrostatic linkages with them, which shield the nucleic acid from dehydration injury or attack from high salt concentrations. Proline and glycine betaine (Fig. 3) are two nitrogen compounds which have been found to accumulate in plant cells when they are challenged

by drought or high salt environments. The positive charge of these molecules helps balance the negative charges which would accumulate in the cytoplasm of cells when they are excluding or pumping out harmful sodium cations (Na^+) during confrontation with a high salinity environment. It is reasonable, therefore, that nitrogen sufficient grasses will be better able to tolerate drought and salinity stresses than nitrogen deficient plants.

Polyamines, e.g. putrescine (Fig. 3), contain two or more protonated $-\text{NH}_3^+$ groups, which allow them to bind electrostatically with nucleic acids and proteins where they appear to function as regulators of metabolic processes. These are also synthesized in response to environmental stresses where they may serve to protect proteins and nucleic acids from extremes in temperature. Again, it is the ability of these nitrogen-containing molecules to become cations that endows them with protective properties.

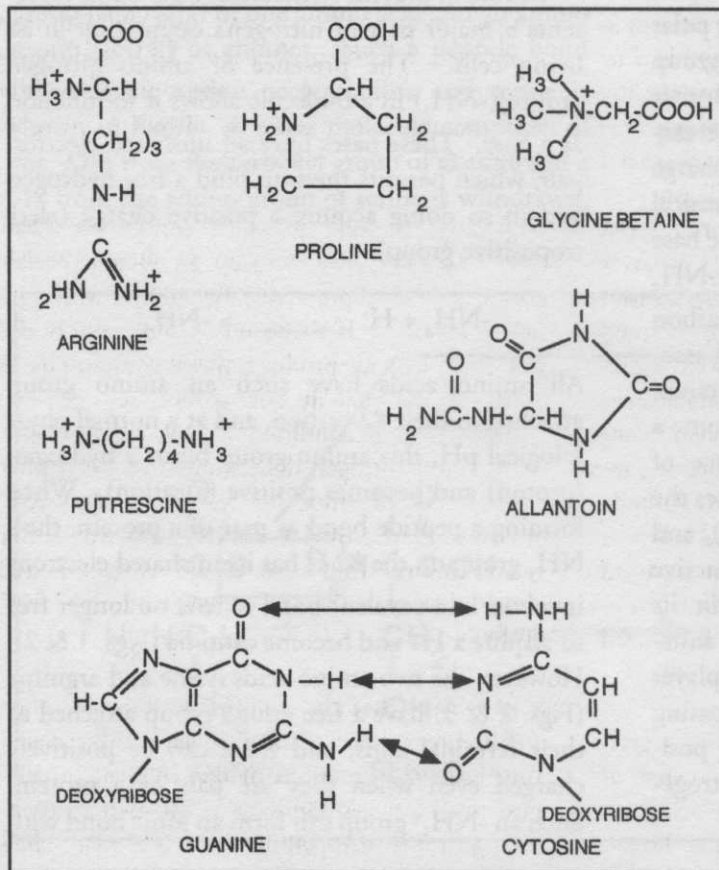


Figure 3. Examples of chemical structures in which N functions as a base attracting an H^+ and gaining a positive charge or forming a hydrogen bond.

Another example of an important function of nitrogenous bases is their ability to form hydrogen bonds with carbonyl groups ($-\text{C}=\text{O}$) and ring-bound nitrogens, thereby stabilizing the double stranded structure of nucleic acids. In Fig. 3, a nitrogenous purine base guanine is linked via three hydrogen bonds (double arrows) to a pyrimidine base cytosine. Such bonding not only stabilizes nucleic acid structure but also contributes to the transcription of DNA molecules by which complementary RNA molecules are formed. These RNA transcripts become the template on which the amino acid sequence of proteins is determined. Thus, nitrogen plays an essential role in the most basic life functions.

The unshared electron pair on nitrogen makes it sufficiently electronegative that it attracts a metal cation. This property

Nitrogen Function Explains Its Deficiency Symptoms

Insufficient nitrogen results in reduced growth rates, yellowing (chlorosis) of lower leaves, and reduced tolerance to environmental and biological stresses. Because growth depends on the production of new cells, and each cell requires its complement of nitrogen rich proteins and nucleic acids, a deficiency of nitrogen will dramatically slow the rate of growth. This is the most common nitrogen deficiency symptom that normally goes unnoticed, because growth rate in turf is less important than it is in most crop plants. One could argue that some growth reduction due to limiting nitrogen might be good because mowing frequency could be decreased. I am not convinced that argument is valid, due to the secondary symptoms caused by low nitrogen.

One of the most nitrogen demanding processes in leaf growth is the synthesis of chlorophyll and its associated proteins that make up photosynthetic units. A photosynthetic unit involves a single electron generating reaction center and several hundred chlorophyll/protein units (light capturing complexes) along with more than a dozen proteins required to generate the reductant necessary to fix carbon dioxide. Also, in cool-season turfgrasses, many rubisco molecules are required to bind the carbon dioxide prior to its reduction to carbohydrates. Photosynthetic machinery requires a lot of nitrogen, so under deficiency conditions, there is a reduction in the number of photosynthetic units produced. Fewer photosynthetic units translates into less chlorophyll and a loss of green color. Consequently a common nitrogen deficiency symptom is light green or yellow leaves. This leaf chlorosis is the most readily recognized characteristic of insufficient nitrogen supply.

Because nitrogen is highly mobile within a grass plant, its deficiency promotes a transfer of nitrogen containing compounds (amino acids and amides) from older leaves to emerging leaves. This transport is preceded by senescence of older

leaves which lose their green color as chlorophyll and proteins are degraded prior to nitrogen export. Lower leaf senescence further contributes to a yellowing of turf and contributes to nitrogen deficiency symptoms.

If inadequate nitrogen restricts protein and chlorophyll synthesis in leaves, much photosynthetic product is converted to sugars which are readily transported to the roots. This additional energy in the roots stimulates their growth and capacity to absorb and metabolize nitrate from the soil. Since the roots are the first plant organs to obtain whatever nitrogen is available, they will grow in response to the additional sugars obtained from the leaves at the expense of shoot growth. Thus, insufficient nitrogen will stimulate root growth over shoot growth and increase the root:shoot ratio of turfgrasses. This predisposes the turf to rapid nitrogen uptake and explains why turf responds so quickly to nitrogen fertilizers applied to nitrogen starved grass.

Turfgrasses respond to stress conditions (heat, drought, pathogen attack) by producing specialized proteins and protective metabolites which are rich in nitrogen. If nitrogen is in short supply, the plant's ability to produce these compounds is restricted and that in turn reduces its defensive response to stress conditions. Thus, a less obvious symptom of nitrogen deficiency in turf is greater vulnerability to stress injury. It is commonly observed that nitrogen starved turf will suffer greater injury from disease and environmental stresses than grass that is adequately but not excessively fertilized.

It is evident that the symptoms observed when turf becomes deficient in nitrogen can be related directly to the functions of this element in turfgrass nutrition. A corollary to this is that maintaining proper nitrogen nutrition is a major challenge to the turf manager.

bonds with proteins and other nitrogen containing groups. These metal atoms can function as coordination centers in complex pigment molecules such as chlorophyll a (Fig. 4). It is evident that nitrogen plays a pivotal role in binding the central Mg^{2+} atom in chlorophyll and stabilizing the complex ring structure of that molecule. Similar structures are stabilized by nitrogen-metal bonds in cytochromes, where Fe^{3+} functions as the coordination center and is involved in electron transport in photosynthesis and respiration itself becoming reduced and oxidized. Metals are critical to the structural organization of many enzymes, and their binding to protein normally involves nitrogen bases.

It should be clear by now that nitrogen, because of its uniquely basic properties, is absolutely essential to the structure and function of many metabolically important compounds including enzymes, nucleic acids, regulator molecules, some hormones and molecules involved in stress tolerance. No other element having these properties is as abundant in nature, and so the evolution of life had no choice but to rely heavily on the use of nitrogen.

Why So Much Nitrogen Is Required

If the major function of nitrogen is to serve as a catalyst, why is it required in amounts greater than any other mineral element? True, nitrogen is a major component of proteins and nucleic acids and is present in a vast array of primary and secondary metabolites, but even so, one might question why it is required at four times the amount of the next most abundant mineral element, potassium.

The answer to this question resides deep in the history of plant life on earth. When life began more than 3 billion years ago, and the first photosynthetic cells emerged in ancient lakes and oceans, the world was very different from what we know today. There was very little, if any, free oxygen, it being derived from photosynthesis, and much of the carbon was reduced and present as methane or other similar gases. Some CO_2 was

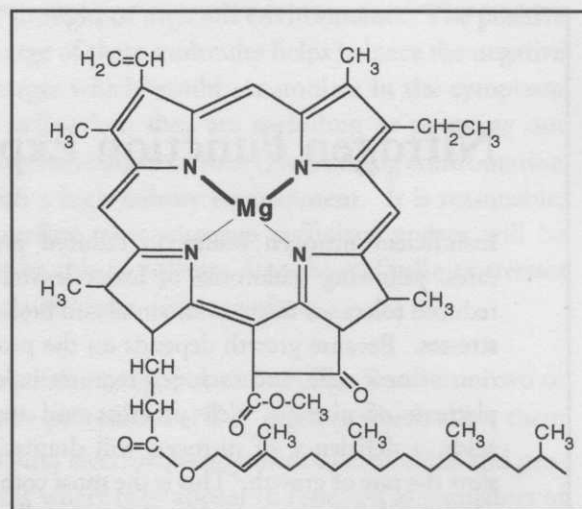


Figure 4. Structure of chlorophyll a showing the bonds formed with nitrogen through the sharing of an electron pair with magnesium.

present, and as photosynthesis evolved and released oxygen, the CO_2 concentration in the atmosphere and in the waters increased. The first photosynthetic bacteria and blue-green algae of the precambrian evolved an enzyme for fixing CO_2 which is very much like that present in plants today. Named ribulose-bisphosphate carboxylase, or Rubisco for short, this enzyme combines CO_2 with a 5-carbon sugar and produces two 3-carbon acids, the first products of photosynthesis.

Over the millions of years since the first photosynthetic cells, plants have invaded the land, and the atmosphere has become enriched with O_2 (21%) but largely depleted of CO_2 (0.03%). Rubisco, which evolved under high CO_2 and low O_2 , does not operate very efficiently under contemporary conditions. Under present levels of atmospheric CO_2 , rubisco functions at only half of its maximum rate and its ability to bind with CO_2 is inhibited by as much as 50% by O_2 .

To compensate for the poor CO_2 fixation ability of rubisco, most plants simply make large amounts of the enzyme. Adequate CO_2 fixation rates are maintained by producing so much rubisco, that it alone constitutes about 50% of all protein in

leaves. However, some plants have employed a prefixation enzyme, phosphoenol pyruvate carboxylase (PEPcase), that binds CO₂ to a 3-carbon acid to produce a 4-carbon compound, which is concentrated within special cells where it releases its CO₂ under conditions (high CO₂ and low O₂) where rubisco can function efficiently. Plants having this PEPcase CO₂ prefixation enzyme include warm-season turfgrasses and several summer weeds. These plants can carry on photosynthesis by devoting only 10% of their leaf protein to PEPcase and another 10% to rubisco.

This difference in nitrogen required for adequate photosynthesis is evident when the leaf nitrogen content of cool-season grasses (those dependent solely on rubisco) is compared with that of warm-season grasses (Table 1). While cool-season turfgrasses average 4.3% nitrogen, warm-season turfgrasses contain only 1.7% nitrogen. Based on this factor alone, warm-season grasses should be able to grow on about half the nitrogen required by cool-season grasses. As an additional indicator of how the low efficiency of rubisco contributes to the nitrogen requirements of leaf tissues, compare the nitrogen content of leaves and roots of cool-season grasses (Fig. 5). While leaves of nitrogen-sufficient grasses contained about 5% nitrogen, roots of the same grasses exhibited a 2% nitrogen content.

It appears that plant tissues can grow and maintain themselves adequately with a 2% nitrogen content (dry weight basis), but to compensate for metabolic inefficiencies, that level may increase to more than 5%. Rubisco is the major enzyme contributing to elevated nitrogen levels in cool-season grasses, but other inefficient enzymes may also promote an increase in tissue nitrogen. Breeding for metabolic efficiency in nitrogen use may reduce its requirement in turfgrass management.

When nitrogen is withheld from cool-season turfgrasses, the nitrogen content of leaves and roots declines by more than 50% (Fig. 5). When leaf nitrogen is reduced to less than 3%, deficiency symptoms become evident (see side-bar on N deficiency), indicating that the nitrogen supply is insufficient to support normal growth and devel-

opment. However, a range of leaf nitrogen levels can be identified (5+ to 3.5%) over which growth appears to be normal and no symptoms of deficiency are evident. This represents a condition of excess nitrogen where green color may be increased but no favorable growth responses are observed. Such excess nitrogen can cause unfavorable declines in the root:shoot ratio, predispose the turf to disease and render the grass less able to tolerate environmental stresses.

Nitrogen Mobility in Turf

Because nitrogen is required in relatively large amounts, most plants have evolved a number of characteristics which increase its efficiency of use. Paramount among these factors is nitrogen mobility. When nitrogen supplies become limiting or during normal leaf senescence, nitrogen in macromolecules is mobilized by conversion to simple compounds that are readily translocated to plant organs where the need for nitrogen is greatest. Allantoin (Fig. 3) is an efficient transport form of nitrogen, in that each molecule contains four nitrogen atoms. Various amides and other nitrogen-rich compounds are transport vehicles in different plants. This ready mobility of nitrogen within a plant permits the growth of new organs even when the supply is inadequate. In this way, a plant can produce flowers and seed, or generate vegetative storage organs using nitrogen mobilized from older leaves or other senescing structures.

Table 1. Nitrogen content of leaves from cool- and warm-season turfgrasses.

<i>Turfgrass</i>	<i>Leaf nitrogen*, % dry weight</i>
<i>Cool Season</i>	
Chewings fescue	4.8
Creeping bentgrass	4.9
Creeping red fescue	3.9
Kentucky bluegrass	4.3
Perennial ryegrass	4.7
Tall fescue	3.0

<i>Warm Season</i>	
Bahiagrass	1.4
Bermudagrass	2.5
Dallisgrass	1.3

* Based on Hull 1992

Total production is sacrificed when older leaves senesce and give up their nutrients to support new growth, but the plant is thereby provided with sufficient nutrients to complete its life cycle. Mobility within a plant is one way chronic nitrogen deficiency can be tolerated, and the plant is capable of self perpetuation.

Nitrogen mobility also permits plants to evolve a strategy to compensate for variable available soil nitrogen. When nitrate is absorbed in large amounts by roots, it is translocated to the shoots where it causes a shift in the allocation of photosynthetic products from sugars to amino acids. Sugars normally would be translocated to roots, but amino acids stimulate shoot growth which retains photosynthate in leaves and stems. Reducing resource allocation to roots slows their growth which, in turn, reduces the amount of nitrate absorbed. Thus excess nitrate uptake initiates a series of events that eventually shuts down the nitrogen supply to the plant.

Conversely, when nitrate levels in the soil are low, most nitrate absorbed is reduced and assimilated to amino acids within the roots and little is transported to shoots. Amino acids synthesized in the roots stimulate root growth, while the lack of nitrate in the leaves promotes photosynthate allocation to sugar synthesis and its translocation to roots. Thus, root growth is favored over shoot growth, and the plant's capacity to absorb more nitrate is increased. By differentially influencing shoot and root growth, nitrogen supply serves as a signal telling the plant how best to allocate its resources for maximum growth and health. Understanding these principles

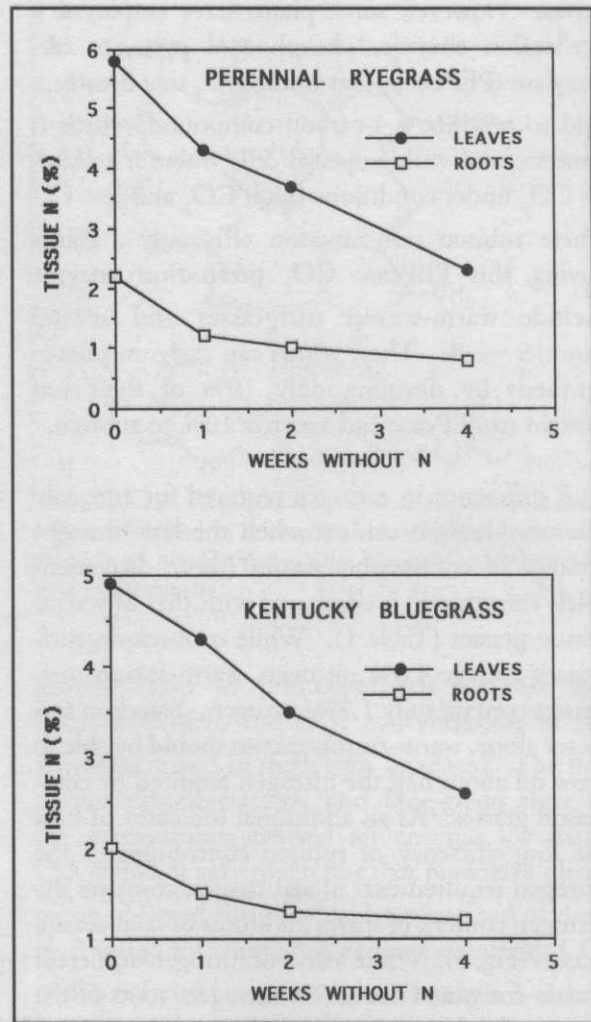


Figure 5. Change in nitrogen content of leaves and roots of perennial ryegrass and Kentucky bluegrass during a four-week withdrawal of nitrogen. Based on Bowman et al. 1989.

should allow the turf manager to utilize nitrogen more efficiently for sustained growth and maximum stress tolerance.

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Field tips: Nitrogen Function Explains Turfgrass Management

An appreciation of nitrogen function in turfgrasses can lead to a greater understanding of sound turf management. Several examples of this link between nitrogen use and turf management are presented.

- Recognizing that turf green-up and growth in the spring requires the biosynthesis of many nitrogen containing compounds reveals that turf will have its greatest need for nitrogen early in the growing season. Turf will exhibit nitrogen deficiency symptoms most readily in the spring. Because soil temperatures may remain cold long after grass growth resumes, the mineralization of soil nitrogen will be delayed and nitrogen supply may not meet plant needs. Monitor turf for nitrogen nutrition and apply small amounts of readily available nitrogen when soil supplies run low. A modest application of soluble nitrogen during spring green-up but before leaf growth resumes and again following the spring flush of leaf growth will help insure adequate nitrogen nutrition.

- During late summer, when cooler temperatures cause a resumption of turf growth, a much reduced root system may be hard pressed to absorb sufficient nitrogen to support good regrowth. Even though soil nitrate levels may be high at this time, the turf may not have sufficient roots to reach it effectively. A light application of soluble nitrogen during late summer will hasten turf recovery and promote more rapid re-establishment of an effective root system. Whenever growth is being initiated, the demand for nitrogen is high and fertilizer management should be adjusted to insure that those demands will be met.

- Leaves of cool-season turfgrasses are rich in nitrogen. Their removal as clippings would be a major loss of

nitrogen to the turf. Retaining clippings on turf is equivalent to applying 1.5 lbs N/1000 sq-ft as a slow release fertilizer. Since clippings filter down to the thatch and are decomposed when moisture conditions are favorable, they will release their nitrogen at a time and in a place most suitable for recovery by turfgrasses. Green clippings contain much more nitrogen and other nutrients than yellow senescent leaves that are naturally lost each season.

- Excess available nitrate in the soil will promote rapid uptake by roots and overpower their capacity to metabolize nitrate to amino acids and proteins. Under such conditions, rapid transport of nitrate and amide-N (glutamine) to the leaves will, under appropriate conditions, cause guttation fluid to collect at leaf tips. This fluid is rich in nitrogen compounds and other nutrients and can support the growth of disease organisms. When guttation fluid dries, the high salt content can cause tip burn. These conditions can be avoided by applying soluble nitrogen fertilizers at low rates only at times when turfgrasses utilize nitrogen effectively.

- Turfgrasses respond to stress conditions by inducing the synthesis of specialized proteins, some of which may serve as enzymes for the production of protective nitrogenous compounds. It is important, therefore, that sufficient nitrogen be available to the grass to support these nitrogen demanding processes. A low application in early summer of nitrogen, 35% of which is readily available, should insure that nitrogen supplies are adequate when stress demands are high. Turf can best tolerate adverse conditions when its nutritional status is favorable, and maintaining this status is a goal of the turf manager.



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