Necrotic ring spot (NRS) (Ophiosphaerella korrae) and summer patch (SP) (Magnaporthe poae) infect cool-season grasses, particularly Kentucky bluegrass. The pathogens are damaging because they infect and kill turfgrass crowns and roots, resulting in slow recovery. The pathogens belong to a larger group that cause “patch” diseases in turfgrasses, although they are not taxonomically related. A patch is characterized by the blighted appearance of turf in a section of otherwise healthy turf. Patch diseases are difficult to diagnose in the field because some of the symptoms can also be caused by a variety of other stresses. For example, stress caused by poor or extreme soil moisture, or improper mowing will result in brown turfgrass in irregular shaped patches, as will repeated urination by dogs on the same spot. Diagnosis can also be difficult because of the similarity of individual symptoms between different pathogens.

**Symptoms**

Initial symptoms for NRS are small light green spots on the lawn that can be easily overlooked (Fig. 1). The first symptoms for SP consist of small areas of the lawn with poor growth, and wilted grass blades. As both diseases advance enlarging brown patches of turf will be visible that can range in size from 1-3 ft. in diameter. NRS usually occurs earlier in the season (late spring) when temperatures are still cool and soils are moist. Summer patch often becomes active later in the season (June-July) after temperatures have warmed up. In some areas of the lawn, NRS patches will disappear with higher temperatures, while in other areas infected patches may reappear under drought or heat stress. In the fall, NRS infection centers can reappear and may continue through winter and early spring. Symptoms of SP emerge during hot, wet weather (rainy days or heavy thunderstorms) in the summer. Symptoms from the previous summer of both diseases may still be visible in the fall and even the following spring. Recovery from both diseases is slow.
Even though grass blades do not become infected by either disease, they typically turn a reddish-brown to bronze, and finally straw color, after roots are infected and killed, depriving the plants of water and nutrients (Fig. 2). There are no leaf lesions on foliage infected with NRS or SP. Heat and drought exacerbate symptoms, making them more noticeable (Fig. 3). Frequently, a few turf patches will survive and re-colonize some of the infected areas, creating a ring-like pattern (Fig. 4). In other cases, all of the turf in a patch will die, leaving an indentation with dead grass in the lawn. As the diseases advance, roots, crowns, and lower stems will develop a black or brown discoloration. Deep fungal hyphae can be present on the surface of roots, stems and rhizomes (Fig. 6). These hyphae, however, are not unique to the NRS and SP pathogens. Other fungal turf pathogens produce them as well. Infected roots are usually brown to black. Pseudothecia (fruiting bodies containing spores) may be present on the infected crowns or roots, although these are rarely seen in the field (Fig. 7). Necrotic ring spot and SP are usually more severe in turf established from sod and in areas with compacted soil. When present, disease symptoms typically appear 2-3 years after turf establishment, but may appear as many as 10 years later. Older turfgrass varieties may also be more susceptible to both pathogens.

DISEASE CYCLE
Necrotic ring spot and SP pathogens survive harsh conditions in plant debris and are thought to colonize neighboring plants by growing on the surface of roots and rhizomes. The hyphae initially colonize the surface of roots eventually invading the root and rhizome tissues. Infection of roots leads to a decrease in water and nutrient uptake, exposing the plants to other environmental stresses or killing them. Infection of turf with NRS happens in spring or fall when soil temperatures reach 60-70°F at 3” depth. Infections with SP occur within a similar temperature range (65-70°F) in late spring.

DIAGNOSIS
To be certain that NRS or SP is causing the symptoms seen in turf, samples can be dug and checked for the presence of the pathogens (Fig. 7 and 8). Fruit bodies of NRS are rarely seen. For diagnostic purposes, roots are cleaned of soil and examined for growth cessation structures produced by SP (Fig. 8). If no growth cessation structures are found the disease is considered to be NRS. The Utah Plant Pest Diagnostic Lab (http://utahpests.usu.edu/ppardl/) provides sampling recommendations and accurate diagnosis.

MANAGEMENT
Management practices that reduce stress on turfgrasses will help to suppress NRS and SP. Raising mowing heights to 2.5 to 3 in. encourages deeper rooting of the turf, improving stress tolerance. Though not typically recommended, thicker thatch layers and more compacted soils may require vertical mowing or power raking. This is usually done to ensure good soil-to-seed contact for germination, if re-seeding an area is required. Lawns should also be watered deeply and as infrequently as possible without creating water stress. In the presence of NRS and SP, however, more frequent watering will cool the grass, reducing temperature stress and allowing infected plants to survive high afternoon temperatures. Avoid over-fertilization with nitrogen, particularly in the spring, and choose slow-release fertilizers as opposed to quick-release fertilizers that can exacerbate disease symptoms. Fertilizer formulations containing sulfur-coated urea and applications of elemental sulfur as soil amendments may help to suppress the diseases.

Resistant Turfgrass Cultivars*
There are no varieties of Kentucky bluegrass that offer complete resistance to NRS and SP, though some cultivars are more disease-tolerant than others. Midnight, NuDestiny, Everglade, Everest, Award, Granite Seed Company’s Const, Rugby II, Ginney II, Jump Start, America, Blue Velvet and Quantum Leap have shown some resistance to SP. Midnight, Award, Langara, NuDestiny, Blue Velvet, America, Jump Start, Everglade, Everest, and Ginney II have shown some resistance to NRS. Planting mixtures of two or more grass species and two or more disease-resistant cultivars will reduce the susceptibility of a lawn to NRS and SP. Overseeding infected areas with resistant cultivars can also reduce the incidence of the diseases. In severe cases, consider overseeding infected areas with perennial ryegrass, which is reported to be immune to NRS and SP. Overseeding with resistant varieties may also be less expensive, in the long term, than regular fungicide applications applied year after year. Be aware that different turfgrass species have different colors and textures that may be visible when seeded in mixtures. However, perennial ryegrass/Kentucky bluegrass are similar in color and texture and may impart resistance to NRS and SP.

Fungicides
There are some fungicides registered for use on SP and NRS. However, the timing of applications in the spring must be exact to assure effectiveness and the fungicide must come in contact with SP and NRS pathogens before they infect and colonize the plants. In addition, fungicide chemistries have to be rotated frequently to avoid fungicide resistance. Once the pathogens are resistant, they are much more difficult to control. Most fungicides labeled for SP and NRS have to be applied by a commercial applicator and cannot be purchased by home-owners. Products with the following active ingredients may be used: azoxystrobin, propiconazole, thiophanate-methyl, myclobutanil and pyraclostrobin**.

Prevention
Turfgrass plants that are severely infected with NRS and SP can easily be pulled from the soil because of widespread rotting of roots, rhizomes, and crowns. These plants may then spread the diseases if they are moved by mechanical equipment utilized on the site. Effectively cleaning equipment with a power steam washer before moving to another location will help prevent further spreading of the disease. Where NRS and SP infections are severe and recurrent, preventive fungicide application may help suppress the development of NRS and SP, but timing of application is essential (see information under Fungicides).

Fig. 3. A lawn showing more severe symptoms of NRS with coalescing rings.1

Fig. 4. Recolonization of infected area by turfgrass.1

Fig. 5. Blackening of Kentucky bluegrass roots and rhizomes infected with necrotic ring spot.2

Fig. 6. Dark hyphae of necrotic ring spot woven around a Kentucky bluegrass root.2

Fig. 7. A pseudothecium of necrotic ring spot embedded in a Kentucky bluegrass root.2
Fig. 8. Growth cessation structures produced by summer patch. 4

REFERENCES AND ADDITIONAL SOURCES


