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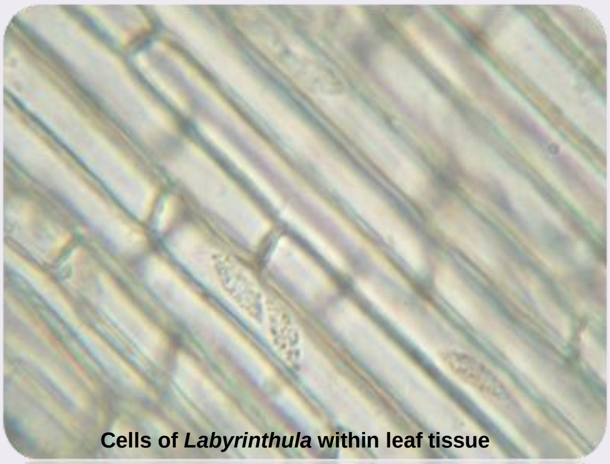
Information Sheet - Rapid Blight

Symptoms on *Festuca* / *Agrostis* / *Poa* sward, UK

- **Rapid Blight is caused by an organism called *Labyrinthula terrestris*. The organism is not a fungus and its exact classification remains uncertain**
- **The disease affects many cool season turfgrasses and may be present in non-symptomatic warm season turfgrasses**
- **The organism colonises and damages all pant parts**
- **The organism requires conditions of high salinity in order to be active and cause disease**
- **The disease can be managed by reducing salinity, the use of tolerant cultivars and timely application of certain fungicides.**

The symptoms of Rapid Blight were first described on *Poa annua* putting greens across southern California in 1995 (Martin et al, 2002) but it wasn't until 2003 that the causal organism was identified as a *Labyrinthula* species (Olsen et al, 2003) and named as *L. terrestris* sp.nov. in 2004 (Bigelow et al, 2005). The disease has subsequently been reported on *Lolium perenne*, *Poa trivialis*, *P. pratensis*, *Agrostis tenuis* and *A. canina* but it can be isolated from several other non-symptomatic turfgrasses (Bigelow & Olsen, 2004; Olsen & Kohout, 2006). To date, the disease has been identified in 11 states across the USA, the UK (Entwistle et al, 2005) & Spain and *Labyrinthula* has been isolated from non-symptomatic turfgrasses in Portugal. The disease is now thought to be caused by the deliberate and often mandated use of elevated salinity irrigation water, making it a unique example of an emergent plant disease potentially induced by human activity (Douhan et al, 2009).

The Pathogen, *Labyrinthula terrestris* This is the first known species of a Labyrinthulid organism to cause disease in terrestrial plants. Prior to its identification as the cause of Rapid Blight, the only other known pathogenic *Labyrinthula* species was *L. zostera* which is responsible for a wasting disease of the marine 'eelgrass' *Zostera marina*. During the 1930's, up to 90% of the eelgrass around the Atlantic coast of North America and Europe were lost to this disease (Muehlstein, 1989). Labyrinthula species produce spindle-shaped cells that move within ectoplasmic networks that are produced by surface organelles called bothrosomes. This network consists of branching and anastomosing (connecting) filaments that are capable of absorbing nutrients as well as attaching the organism to its substrate (Leander & Porter, 2001). This organism is often referred to as a 'marine or net slime mould' due to its production of an extracellular matrix through which the individual or aggregated cells move.



Cells of *Labyrinthula* within leaf tissue

However, this description incorrectly suggests that the organism is related to the true slime moulds and should therefore not be used and an up to date classification is given in Bigelow et al, 2005. The initial identification, by Olsen et al in 2003, to place this organism in the genus *Labyrinthula* was based on shared morphological characteristics. In 2005, Craven et al confirmed this identification using molecular techniques and went on to suggest a possible common ancestor for all turfgrass isolates of *Labyrinthula*. However, in 2009, Douhan et al completed a more extensive molecular study including isolates obtained from Europe and showed a clear difference in the genetics between the UK isolate and those collected across the USA and Spain, suggesting that the UK isolate may indeed be a unique species.

Host range Unfortunately all colonial and velvet bents (*Agrostis capillaris* and *A. canina*) perennial ryegrass (*Lolium* perenne) and meadowgrasses (*Poa* spp.) appear to be very susceptible to infection (Peterson et al, 2005). The pathogen can be isolated from non-symptomatic cool-season and warm-season turfgrass species and it is likely that it is present much more extensively in amenity situations than we are presently aware.


Symptoms on creeping bentgrass, Spain

Symptoms Initial symptoms of infection are watersoaked patches of turf that may appear slightly darker (brown/bronze) compared to the surrounding unaffected sward (see above). Turf may become yellow in colour, show rapidly expanding & coalescing areas of infection up to 1 meter or more in diameter and die within a few days of the initial symptoms being expressed (Olsen et al, 2008). The disease has been seen to develop on all areas of the golf course (greens, tees, fairways and roughs) and develops in the autumn and spring months where cool season turfgrasses are cultivated or on the cool season grasses used to overseeded bermudagrass turf prior to dormancy. The severity of the symptoms depends up on the severity of the salinity that is present or applied but symptoms begin to be expressed where salinity is around 2.0dS/m or above.

Epidemiology The disease develops on turf irrigated with water at moderate to high salinity (EC >2.0dS/m (Olsen et al, 2006)), i.e. sodium and/or bicarbonate levels, or on turf where the salinity level is high due to low moisture content in the profile. There is a significant variation in tolerance to rapid blight disease between different turfgrass species and also between cultivars of individual grass types. It has been possible to isolate the causal pathogen from non-symptomatic cool season turfgrasses that have been irrigated with water at <1.0dS/m and from non-symptomatic bermudagrass which appears to act as a reservoir of inoculum for infection of overseeded grasses. Symptoms can develop immediately following the first mow of the overseeded turf (Olsen et al, 2006) and in general, the severity of the disease appears to be correlated with an increase in salinity of applied irrigation.

Management Because this disease is so dependent on the level of salinity in the irrigation water or accumulating in the profile, any management aimed at reducing salinity will lessen the severity of the symptoms. Rainfall is perhaps the best and most effective control option.

Research is on-going to identify the relative tolerance of different grasses and cultivars and it would appear that the grasses with increased salt tolerance show increased tolerance to development of Rapid Blight. The grasses that currently show the greatest tolerance to infection are Seaside II bentgrass or creeping red or other fescues.

Regarding chemical control, research has indicated that the most effective approach is to apply high rates of pyraclostrobin and/or mancozeb (tank mix or alternate applications) where the disease is known to be a problem, especially where cool season grasses are used to overseed bermudagrass. Olsen et al, 2006 indicated excellent control using a high rate (0.9oz per 100sq.ft which is equivalent to 2.75 kg/ha) of pyraclostrobin applied either at the time of the first cut of the seedling turf or at the first signs of disease development. Moderate control was also achieved using early and repeat applications of pyraclostrobin and mancozeb (at 0.5oz and 6oz per 1000sq.ft respectively), either separately or tank mixed*.

Trifloxystrobin has also shown some efficacy but other fungicides used against other amenity fungal diseases, have shown little to no efficacy against this pathogen.

*** in the UK the current label rate for pyraclostrobin is 1.25 kg/ha and mancozeb is not approved for use on amenity turf**

Due to the low incidence of this disease in Europe, I have been working with Dr Mary Olsen, University of Arizona, USA and isolates of European *Labyrinthula* sp. have been sent to Dr Olsen for inclusion in her research.
Dr Olsen is one of the leading researchers on Rapid Blight and can be contacted at molsen@Ag.arizona.edu
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