

NEW DISEASES ARE DEVELOPING FACT OR FICTION?

Kate Entwistle delves into the murky world of fungi and reports the startling fact that there are more out there than you might believe.

I am quite sure that most people working in our industry could name up to ten common turfgrass diseases without too much trouble. However, naming all of the fungi that cause the diseases may be a little more of a challenge. Assume for a moment that you were asked to take a guess at the total number of fungi that could cause disease on turfgrasses. What would your answer be? Twenty, 50, 100, more than that? Actually, there is no correct answer to this because, quite simply, no one knows. However, I would not be surprised if the number were to be far in excess of any that you had thought and the reason for this is as follows:

'Most of the more than 100,000 fungus species known are strictly saprophytic, that is, they live on dead organic matter which they help to decompose. Some, about 50 species, cause diseases in humans and about as many cause diseases in animals, most of them superficial disease of the skin. More than 10,000 species of fungi, however, can cause diseases in plants. All plants are attacked by some kinds of fungi and each of the parasitic fungi can attack one or many kinds of plants.' Agrios, 1997.

It has been estimated that we have identified only around 10% of the total number of fungi on the planet. That being the case, it is fair to assume that there are likely to be about 1,000,000 different fungal species on the planet and although only a proportion will attack plants and a smaller number cause disease on turfgrasses, we are still talking about a large number of potential pathogens.

Although turfgrasses make up only a proportion of the total number of plants in the world, there are still a large number of potential pathogens that we know of. Changes in the pathogen populations and turfgrass cultivars, not to mention the introduction of new turfgrass species, means that the potential number of fungi capable of causing disease is increased still further.

So if any given fungus can cause disease on one particular grass, why can't it cause disease on all turfgrasses? The answer to this is genetics. The combination of genes within the turfgrass plant (the host) and the fungus (the pathogen) will determine firstly if disease is likely and secondly what type of disease it will be. The expression of any symptoms of disease will be modified by environmental conditions and this will ultimately determine the severity of the turfgrass disease.

Two of the mites recently identified as causing damage to Bermudagrass



The study of genetics is extremely complex but we can try to simplify it to see how this relationship between the host and the pathogen works in disease expression. Taking a step back for a moment, it may be worth considering what happens during the development and selection of a new grass cultivar. Turfgrass breeders select two parent grasses, each of which possess characteristics that the breeder wants to introduce in to the new grass. Through crossing these two parent grasses, sexual reproduction in the plants produces a number of offspring (the seed of the plant) each of which will be different from the other. Growing each of these plants to maturity will allow the breeder to select for some of the offspring that have the best characteristics from each parent. These few can then be studied further to see if they have new or enhanced characteristics over and above those that have been bred in to the plants. After many years of study, a new grass cultivar will be brought to the market which will be better suited to the requirements of the present conditions, whether that be tolerance to close mowing, colour, wear tolerance or whatever. The process is extremely time consuming and also very wasteful because many plants are produced by these crosses

that will never be considered for production of a new cultivar. However, one of the characteristics that will vary in the offspring of these plant crosses is their ability to resist fungal infection and therefore, disease development.

Plants can be regarded as being either highly resistant or not susceptible to attack by a pathogen or alternatively, highly susceptible and therefore not at all resistant. Resistance and susceptibility are two ways of expressing the same characteristic and so it is important to be aware of both. In selecting turfgrass cultivars that have inherited a characteristic that was intended from any two-parent crossing, it is inevitable that other characteristics, including disease resistance, will be affected too. The best outcome that any breeder can hope for is that any parental crossing will result in offspring (or new cultivars) that have inherited all of the best characteristics from each parent. I would guess that the chances of this happening are extremely low and even if it were possible, who decides on 'the best'? What you may require from a grass may differ markedly from what others think is important. Hence the range of grasses available today from which you can select those most suitable for your particular conditions.



Head and body of adult mite (actual size approx 1 mm)

But this story of genetics and the plants' ability to resist disease does not end there. This is a game in which there are two players, the grass and the fungus and the fungus has good reason to cause disease. If it is not able to do so, it will die out. Therefore it will (perhaps not consciously!) do all that it can to cause disease to plants even if their resistance has been enhanced through plant breeding. It does so in a very similar way to that which occurs in plants. Most fungi have the ability to reproduce in one of two ways, either asexually or sexually. Through asexual reproduction, the fungus can produce vast amounts of inoculum (or spores) over a very short period of time and this way it is able to take advantage of sudden changes in environmental conditions to cause disease epidemics. Each of the fungal spores produced by asexual reproduction is, however, roughly the same in its genetic make up and so if the environmental conditions change so as to prevent one of them from causing disease, it will prevent all of them in the same way. Through sexual reproduction, the fungus is able to alter the genetic make-up of the spores in much the same way that the turfgrass cultivars vary from the two parents. The change in the genetics of the spores produced by sexual reproduction means that new 'strains' or 'races' of the fungus are produced. Ultimately, the new fungal populations that result from sexual reproduction, may allow the fungus to cause disease under slightly different environmental conditions from the original population or on host plants that were previously more resistant to that fungus.

One of the greatest differences between the host and the pathogen is the speed at which variation can be introduced into the population. For the turfgrass cultivar, the introduction of new genes and thus new characteristics, can take about ten years from the initial parental crossing. For the pathogen, you could be talking about ten days, or a few weeks, for new strains or races to be produced. This massive advantage in the reproductive ability of the

pathogen means that disease is inevitable. Not only that, but it is occurring most of the time at levels which cause us no concern and that probably go unnoticed. It is only when there is an 'explosion' of activity from the pathogen that the symptoms of disease become all too apparent.

So, fungal disease is an inevitability. Diseases are likely to develop on the new grasses and cultivars that are being bred for use in amenity areas in much the same way that diseases affect all plants around the world. In turf, the environment and the way we manage the sward affect the severity of disease expression. The greater the pressure we put on the pathogen population to try and prevent it from causing disease, the greater the chance of selecting a strain or race of the pathogen that can overcome it.

New fungal diseases are being identified on turfgrasses around the world, due in part to our improved ability to identify the fungi responsible and also to our introduction of new turfgrass types and species. In addition, there are invertebrates (nematodes and insects) that are being identified on grasses that in the past were not known to cause damage to these plants. Apart from identifying that a root knot nematode, *Meloidogyne* sp., is causing extensive damage to creeping bentgrass swards throughout the UK and Ireland, I have recently found a mite that is causing damage to Bermudagrass turf. Although the latter may not affect you directly, it is another clear indication that there is still more to be learned regarding turfgrass pests, be they fungal or invertebrate and anyone that says differently, is, in my opinion, fooling themselves.

Dr Kate Entwistle runs The Turf Disease Centre, Waverley Cottage, Sherfield Rd, Bramley, Hampshire, RG26 5AG
Tel: 01256 880246 Fax: 01256 880178
email: Kate@theturf-disease-centre.co.uk
Website: www.theturf-disease-centre.co.uk