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DISCLAIMER

DAFFODIL DISEASES AND PESTS

DEDICATION

For his many years of service to the American Daffodil Society, including a stint as Chairman of the Health and Culture Committee, I dedicate this booklet to Mr. Willis H. Wheeler.

DAFFODIL DISEASES AND PESTS

by

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PREFACE

The compilation of my seven articles on daffodil diseases and pests into one booklet is the idea of my long-time friends, Dick and Kitty Frank. To them, I express gratitude for taking on this project.

My original intention for writing these seven articles was for the purpose of facilitating the transfer of information from the limited scientific literature on daffodil diseases and pests to the inquisitive, lay daffodil grower. If I have been successful in this attempt, it is because of my graduate training as a plant pathologist at Purdue University, sixteen years college teaching experience, and an undying desire to share what I have learned with all those who wish to know more about daffodil diseases and pests. For any errors which may have crept into these articles, I apologize and accept responsibility.

Lastly, I would like to thank the following people for permission to use the illustrative material which appeared in the original seven articles: Dr. Gary Chastagner, Mr. Willis H. Wheeler, Dr. A. A. Brunt, Dr. Richard M. Lister, and Mr. C. C. Doncaster. Additionally, I would like to thank the American Phytopathological Society, the Bulb Research Centre at Lisse (The Netherlands), the Glasshouse Crops Research Institute (England), and the Ministry of Agriculture, Fisheries and Food (England) for permission to use illustrative material.

Theodore E. Snazelle, Ph.D.
Clinton, Mississippi
February 1986

Chapter 1

SOME BASIC CONCEPTS

"We see . . . our plants wither without being able to render them assistance, lacking as we do understanding of their condition." Fabricius (1774)

Perhaps the quotation from Fabricius is the only reason that I need to begin this first article of a series on daffodil diseases and pests. Or, perhaps, it was the comment of a daffodil enthusiast who said to me, "Don't take too long in giving the program (on basal rot) as it is too depressing!" that makes me feel compelled to write. However, in the final analysis, it is the quotation of Fabricius that strikes to the core of my very existence. It would be unforgivable of me as a plant pathologist not to try to understand what causes a daffodil to suffer, and it would also be unpardonable of me not to share my understanding with those of you who want to understand fully why daffodils suffer. For if we know the why or cause of their suffering, we hope in time to either prevent or to control that suffering. Such is the way of a plant pathologist.

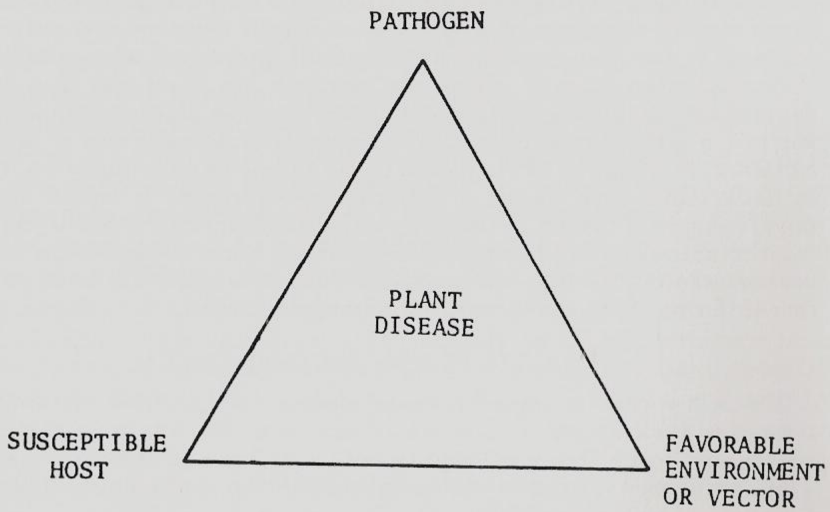
WHAT IS PLANT PATHOLOGY?

The best way to arrive at an initial understanding of the meaning of a term is to look up the root words of that term. For instance, the Greek root words for pathology are *pathos* — to suffer and *logos* — to study. Thus, pathology is literally the study of suffering, and plant pathology is the study of suffering plants.

WHAT IS PLANT DISEASE?

As you might suspect, there are many definitions for disease; however, a simple and workable definition is that disease is a departure from a state of health. Thus, plant disease would be the departure of a plant from a state of health. There are at least two basic types of plant diseases: 1) infectious plant disease, and 2) noninfectious plant disease. Infectious plant disease is the departure of a plant from a state of health as a consequence of that plant becoming infected by a microbe, e.g. bacterium, fungus, virus, nematode, etc. Microbe is actually a term reserved for etiological (disease-causing) agents which are cellular or multicellular in nature, e.g. bacteria, fungi, nematodes. As a virus is sub-cellular in nature, it is called a microbe only for the sake of convenience. Noninfectious plant disease is that disease which is not caused by microbes but instead is caused by non-biological agents such as air pollutants, mineral nutrient deficiencies, freeze or frost damage, and damage due to wind, hail, and excess of rain. Drought is also a cause of noninfectious plant disease. If the etiological agent which causes the plant disease is a microbe, it is called a pathogen. Significantly, the pathogen or etiological agent causing an infectious plant disease can be transmitted from a diseased plant to healthy plant causing it to become sick. Despite the popular belief, it is important to realize that disease does not spread; however, the pathogen causing the disease does spread, i.e. the pathogen can move from a diseased plant to a healthy plant causing the previously healthy plant to become diseased. Thus, pathogens spread; diseases don't spread. Of course, the same is not true for noninfectious plant disease, i.e. air pollutants can't be transmitted from plant to plant as can pathogens.

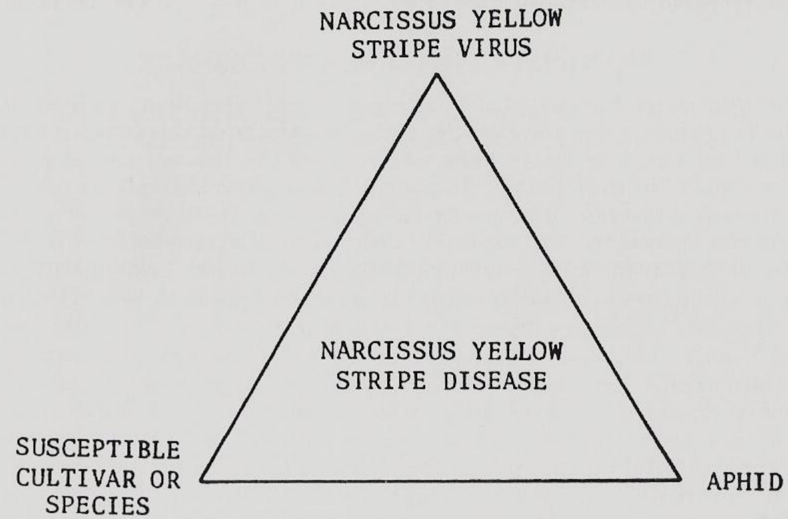
FIGURE 1 - INFECTIOUS PLANT DISEASE TRIANGLE



INFECTIOUS PLANT DISEASE TRIANGLE

Infectious plant disease is usually considered to be the product of at least three factors: 1) a susceptible plant, 2) a pathogen, and 3) a favorable environment and/or vector. A vector is a biological agent which carries the pathogen from a diseased plant to a healthy one. For example, the vector of the virus causing narcissus yellow stripe disease is an aphid. In Figure 1, the infectious plant disease triangle illustrates that infectious plant disease is the consequence of the interaction between a pathogen, susceptible host plant, and favorable environment or vector. In Figure 2, an infectious plant disease triangle has been constructed for the narcissus yellow stripe disease. Here you can see that this disease is the consequence of the interaction between susceptible narcissus cultivars, narcissus yellow stripe virus (NYSV), and an aphid vector. In some instances, infectious plant disease is considered to be the product of four equally-important factors (susceptible host plant, pathogen, vector, and favorable environment), not just three factors, and may be thought of as a pyramid rather than a triangle with each of the four apices of the pyramid being occupied by one of the four factors.

FIGURE 2 - NARCISSUS YELLOW STRIPE DISEASE



SPECIES CONCEPT

A species is a specific kind of plant, animal, fungus, or bacterium. With the exception of the viruses, all the pathogens causing disease in daffodils are designated by species names which are either written in italics or underlined, e. g. *Fusarium oxysporum* is the fungal pathogen causing the narcissus basal rot disease. The application of species names to virus pathogens is rarely done. Thus, a virus is often named by considering the plant species infected and the symptoms caused on that species. Hence, the name narcissus yellow stripe virus (NYSV) for the plant virus which infects narcissus causing the yellow stripe symptom. Often, as you have already observed, plant viruses are referred to by an abbreviation, e.g. NYSV, which was derived from the first letter of each word of the common virus name, e.g. *n*arcissus *y*ellow *s*tripe *v*irus.

SIGNS AND SYMPTOMS

The sign of an infectious plant disease is the actual presence of the pathogen as seen with the unaided eye or with a microscope. For instance, in bulbs which are badly infected with *Fusarium oxysporum,* you can often see with the unaided eye the actual fungus as a pinkish-white growth on the outer bulb scales and around the basal plate. Or, in the case of the bulb and stem nematode, *Ditylenchus dipsaci,* the nematode can often be seen under low power magnification with the light microscope when a fleshy bulb scale is dissected in a drop of water on a glass slide. For the most part, the viruses are all too small to be seen with even the best light microscope.

A symptom of an infectious plant disease is the visible effect of the pathogen on the host, e.g. the yellow stripes which appear on the foliage of daffodils infected with NYSV is a symptom of the narcissus yellow stripe disease. Also, the blackened or necrotic (dead) fleshy bulb scales near the basal plate of a daffodil bulb infected with *Fusarium oxysporum* are a symptom of narcissus basal rot.

NONINFECTIOUS PLANT DISEASE

Noninfectious diseases of daffodils are usually less than obvious to the daffodil grower; however, notable exceptions include the tattered flowers after a hail storm or heavy rain which beats the flowers to the ground. Mineral nutrient deficiencies do not usually express themselves too easily in daffodils although they are easily recognized in other plants through discolored leaves, etc. For example, a shortage of magnesium or iron may result in impaired chlorophyll synthesis by the plant. Consequently, the foliage will appear yellow or chlorotic instead of green as would normally be expected. Lightning damage may be recognized by a circular area of dead plants following an electrical storm. Although you might not ordinarily think too much about it, light is very important for daffodils to continue flowering. For instance, daffodils planted on the north side of a house may receive almost constant shade and will often flower poorly or not at all in subsequent years if they are left in that location. Although I am not aware of air pollutants being very important in causing damage to daffodils, they are particularly damaging to a number of broad-leaved plants. For instance, the main phytotoxic (plant toxic) component of smog is peroxyacetyl nitrate (PAN) which causes bronzing or silvering of the lower leaf surface. Also important as pollutants in causing foliar damage are sulfur dioxide which causes interveinal chlorosis in leaf veins and ozone which causes a bleached stippling of the upper leaf surface.

PLANT PESTS

Certainly you might consider the fungi, nematodes, and viruses as plant pests; however, as we have already designated them pathogens, then the question arises as to what is a plant pest. Although the definition may be somewhat arbitrary, a plant pest may be exemplified by arthropods, e.g. insects, mites, etc., and animals like squirrels. Of course, the most infamous of the arthropod pests of the daffodil is the narcissus bulb fly. *Lampetia equestris.* Also, the bulb scale mite, *Steneotarsonemus laticeps,* is an occasional pest, particularly with bulbs which are forced into bloom. As for squirrels, they seem to have an affinity for the shallow-planted miniature cultivars and small species forms. Other two-legged and four-legged animals, e.g. boys and dogs, respectively, seem to have an uncanny ability to smash daffodils with their feet!

CONCLUSION

It is my hope that this initial article might serve to whet your appetite for subsequent articles on fungal diseases, virus diseases, nematode diseases, and control of these diseases. Also, attention will be given to noninfectious diseases of daffodils and daffodil pests in future articles.

Chapter 2

FUNGI AND FUNGAL DISEASES — BASAL ROT

"Clean and round, heavy and sound, in every bulb a flower."
Ye Narcissus or Daffodyl Flowre, and hys Roots (1884)

Fungi have been, and will continue to be, both a friend and foe of mankind. In the role of friendship, we only have to recall that the antibiotic penicillin is produced by fungus called *Penicillium notatum* or *P. chrysogenum.* Furthermore, there are few of you who haven't enjoyed the delectable qualities of *Agaricus campestris bisporus,* the common cultivated mushroom, which is available in the produce section of your neighborhood supermarket. To my way of thinking, there is little in this world more appetizing than a steak smothered in mushrooms! Fungi have also been, and will continue to be, the bane of mankind, his animals, and his plants. For instance, the wheat rust fungus, *Puccinia graminis,* ravaged Roman wheat so often that the Romans created two gods, Robigo and Robigus, to whom to pray in order that their wheat might be spared (1). Needless to say Robigo and Robigus were not very good plant pathologists. Perhaps the most devasting plant disease which impacted so heavily on human history was the late blight of potato in 1845 and 1846 which caused a massive famine in Ireland and the immigration of more than one and a half million Irish to the United States. Finally in 1861, DeBary proved that the fungus *Phytophthora infestans* was the cause of the late blight of potato (2). More recently, narcissophiles have been concerned with a most destructive fungal disease of narcissus bulbs, basal rot. This disease is particularly damaging in warm, moist climates such as in the Southeastern United States.

WHAT IS A FUNGUS?

Under older classification schemes, fungi were traditionally placed in the plant kingdom even though they had few characteristics in common with plants, e.g. no chlorophyll to convert radiant energy of the sun into chemical energy which ultimately was found in the bonds (covalent) which bind the carbon, hydrogen, and oxygen atoms together in the glucose molecule. Since fungi are not able to synthesize their own simple organic molecules like glucose as do green plants, they must instead acquire many of their organic compounds from an external source by either being a saprophyte or a parasite. A saprophyte is a microbe such as a fungus which requires a nonliving source of organic carbon to grow, i.e. it might be found growing on decaying vegetation. In contrast, a parasite is a microbe which requires a living organic source of carbon to grow, e.g. a daffodil bulb. In actuality, most of the fungi which parasitize plants are facultative parasites in that they have the faculty or capability to grow as either a parasite utilizing a living source of carbon or as a saprophyte utilizing a nonliving source of carbon. The basal rot fungus, *Fusarium oxysporum* f. *narcissi,* is a facultative parasite. Thus, when the basal rot fungus is infecting a narcissus bulb it is existing as a parasite; however, when it is growing in the laboratory in a petri plate containing potato dextrose (glucose) agar, it is existing as a saprophyte. Today, the fungi are not placed in the plant kingdom. Instead, they are placed in a kingdom all their own, the kingdom Fungi (3).

In morphology, the fungi can be divided arbitrarily into two categories: 1) the unicellular fungi, e.g. the yeasts and 2) the filamentous fungi like the basal rot fungus. Our attention will be directed to the filamentous fungi. The basic vegetative (non-reproductive) structure of the filamentous fungi is the hypha (hyphae, plural). A hypha is a tubular filament which, depending upon the fungal species, may or may not have crosswalls called septa (septum, sing.). If a hypha possesses crosswalls or septa, it is said to be a septate hypha; however, if the hypha lacks septa, it is said to be an aseptate hypha. A mass of hyphae as might be seen growing in a petri plate culture of a fungus is called a mycelium.

Typically, fungi can reproduce either asexually or sexually. Sexual reproduction in most fungi takes place less frequently than does asexual reproduction. Sexual reproduction involves ultimately the union of nuclei which contain the genetic material (DNA) to form a zygotic nucleus. Thus, this process superficially resembles that of mammals where a sperm unites with an egg to form a zygote. The similarity between sexual reproduction in mammals and in fungi really ends with the formation of a zygote. As already mentioned, asexual reproduction occurs more frequently in most fungi than does sexual reproduction. As a matter of fact, a large number of plant parasitic fungi either do not reproduce sexually; or if they do reproduce sexually, they do it so rarely that they

FIGURE 1 - VEGETATIVE MORPHOLOGY OF FILAMENTOUS FUNGI

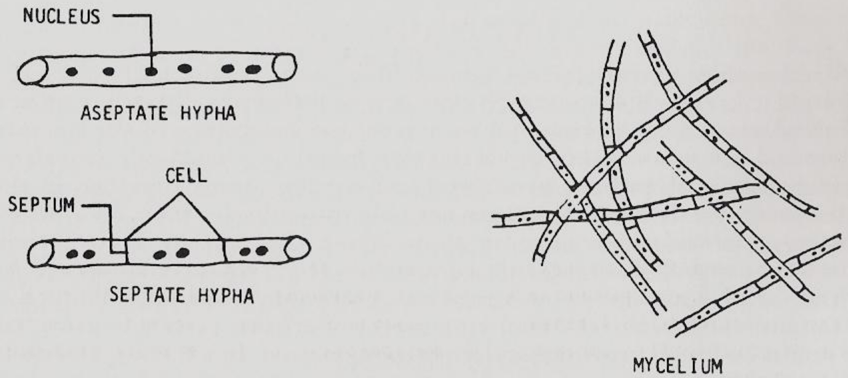
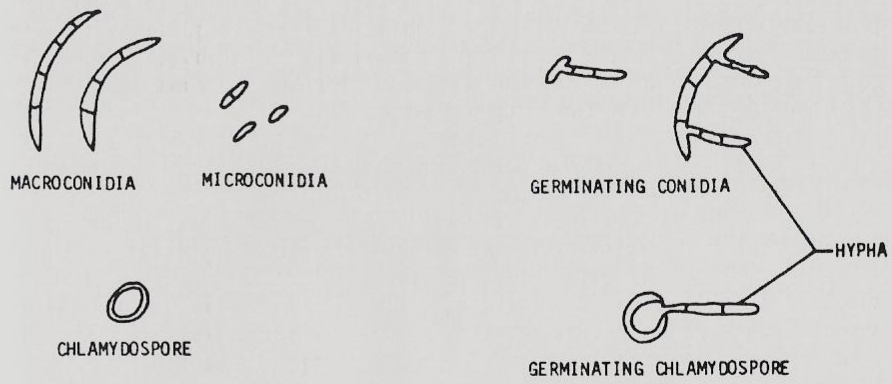


FIGURE 2 - CONIDIA OF FUSARIUM OXYSPORUM



are commonly placed in an order of fungi called the Fungi Imperfecti. "Imperfecti" means that a fungus is imperfect if it lacks a sexual cycle whereas it would be perfect if it possesses a sexual cycle. Asexual reproduction of fungi typically involves the production of conidia (conidium, sing.) or spores. The conidium or spore is a specialized propagative or reproductive body consisting of one or a few cells (Figure 1). When the spore or conidium comes into contact with a proper substrate, e.g. a daffodil bulb, and the conditions are right, e.g. temperature, moisture, etc., the conidium or spore will germinate to produce a hypha which will penetrate the substrate, e.g. narcissus bulb (Figure 2). Thus, the bulb would then be said to be infected by the fungus.

BASAL ROT

In the quote, "Clean and round, heavy and sound, in every bulb a flower," lies the expectation of every daffodil grower. Sometimes this quote does not hold true because of diseases of the narcissus bulb. There are two major diseases of narcissus bulbs. Firstly, there is bulb rot caused by the bulb and stem nematode *Ditylenchus dipsaci;* secondly, there is basal rot which is caused by the fungus *Fusarium oxysporum* f. *narcissi.* In the species name *F. oxysporum* f. *narcissi,* the "f" stands for form. Thus, the form of *F. oxysporum* which infects narcissus bulbs is *F. oxysporum* f. *narcissi.* Some other forms of *F. oxysporum* are *F. oxysporum* f. *lycopersici* which causes wilt of tomato and *F. oxysporum* f. *tulipae* which causes bulb rot in tulips.

Basal rot of narcissus as caused by *F. oxysporum* f. *narcissi* was first described in England in 1887 by M. C. Cooke and in 1911 in the Netherlands by J. Westerdijk (4). It is generally, but not universally, accepted (4) that this fungus is rather specific for its host and is not found in soils not previously cropped with narcissus. Thus, it would follow that this problematical fungus is introduced into the soil by growing narcissus bulbs in it. Thus, the statement by Price (4) rings ominously in my ears: " . . . the bulb itself must carry the seeds of its own destruction." So, it would seem that the basal rot fungus is introduced primarily into garden soil by planting infected narcissus bulbs on the site or by planting bulbs which bear the fungus spores externally on the dry, papery, outer scales. However, Price (4) has also presented some evidence which suggests that the fungus can be introduced into the soil by planting it with tulips. In apparent disagreement with Price, Melville (5) reports that the basal rot fungus can also be found in soils where narcissus bulbs have never been grown.

New infection of narcissus bulbs in the soil primarily occurs in the fall by the germinating spores penetrating wounds in the basal plate which are made by emerging roots (6). Previously, infection of bulbs in the soil was believed to take place primarily late in the growing season when the roots became infected, and the fungus moved into the basal plate and on into the bulb (5,7); however, new work by Langerak (6) of the University of Wageningen, The Netherlands, has shown that new infections only occur during the first few weeks after planting when the roots are emerging from the basal plate. This infection can only occur when the soil temperature is above 12°C or 53.6°F (6). Even though the fungus can thrive in the soil at temperatures below 12°C (53.6°F), it can't infect narcissus bulbs at this temperature. Thus, one aspect of basal rot control would be late fall planting when the soil temperature at 4 - 6 inches deep is 12°C (53.6°F) or below. In the United States, Gould (7) has reported that post-harvest infection is important and begins at the base of the bulb and also at sites of wounding caused during lifting of the bulbs. Post-harvest infection of bulbs in the U. S. has traditionally been controlled through dips in fungicide shortly after lifting. In the Netherlands, no particular benefit in the control of basal rot has been accomplished by immediate post-harvest fungicide dips (6). Instead, the Dutch practice quick drying of bulbs after lifting and storage under temperatures as cool as possible.

Once the bulb has become infected in the soil, the fungus kills the basal plate (Figure 3) and spreads upward through the scales, causing them to become necrotic. This death is easily observed by stripping away the dry, papery, outer scales to expose the inner fleshy scales (Figure 4). These inner scales are typically dark brown as they emerge from the infected basal plate and white nearer the nose of the bulb. In the garden, basal rot is indicated when foliage fails to appear in the spring or by stunted plants having smaller or deformed blooms and early maturing foliage. With bulbs in storage which either were infected the preceding fall when the roots were emerging from the basal plate or which were infected during lifting by the fungus entering wounds, the fungus will spread throughout the bulb causing it to rot and ultimately become only a dust-filled paper shell. Obviously, a hand full of dust in your hand indicates basal rot; however, basal rot is also indicated when the bulb feels soft when it is

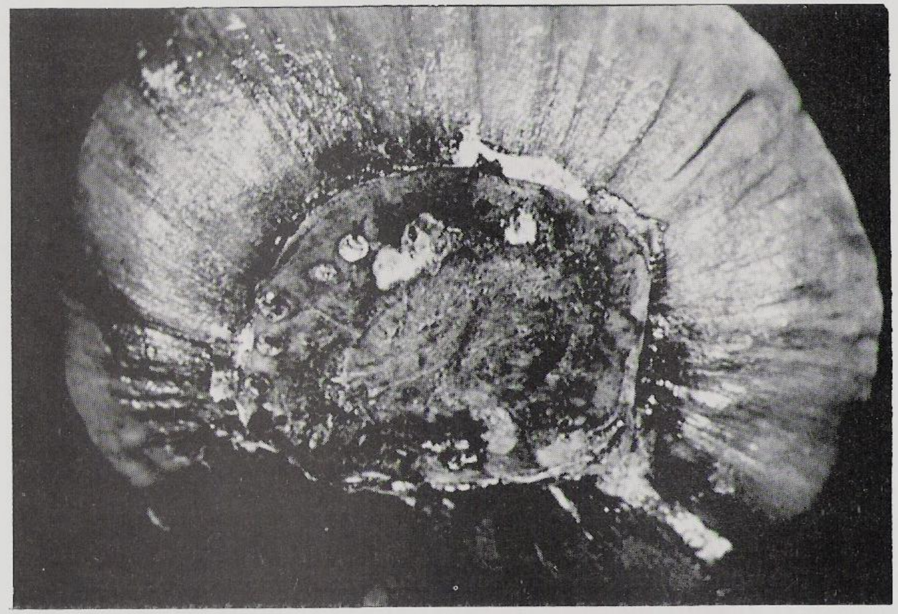


Figure 3 — Basal Plate of Narcissus Bulb Infected by *Fusarium oxysporum* f. *narcissi*. (Print made from slide furnished by Dr. Gary Chastagner, Washington State University.)

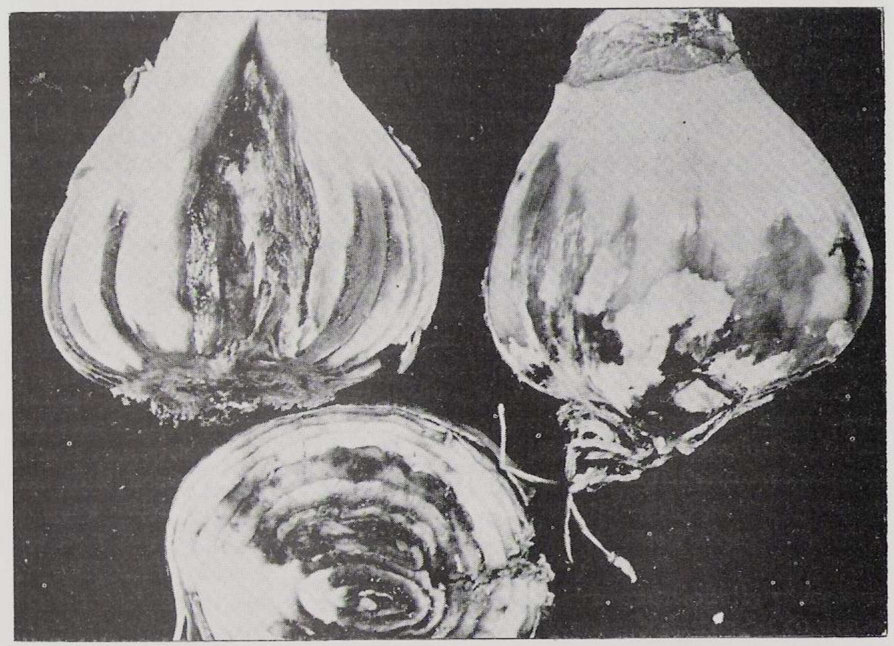


Figure 4 — Fleshy Scales of Narcissus Bulb Infected by *Fusarium oxysporum* f. *narcissi*. (Print made from slide furnished by Dr. Gary Chastagner, Washington State University.)

squeezed. Sometimes the fungus is clearly present on the surface of infected bulbs as a pinkish dust (Figure 5). Although amateur daffodil growers seldom hot water treat their bulbs, commercial growers who routinely practice hot water treatment have observed that infected bulbs have a tendency to float whereas healthy bulbs will sink (5).

Factors which contribute to losses of bulbs to basal rot include: 1) early planting of bulbs when the soil temperature is above 12°C (53.6°F), 2) planting of susceptible cultivars, e.g. "bulb-rotting whites," 3) planting of bulbs in areas which are not well-drained, 4) use of high nitrogen fertilizers when fertilizing, and 5) failure to practice crop rotation to reduce the amount of fungus in the soil.

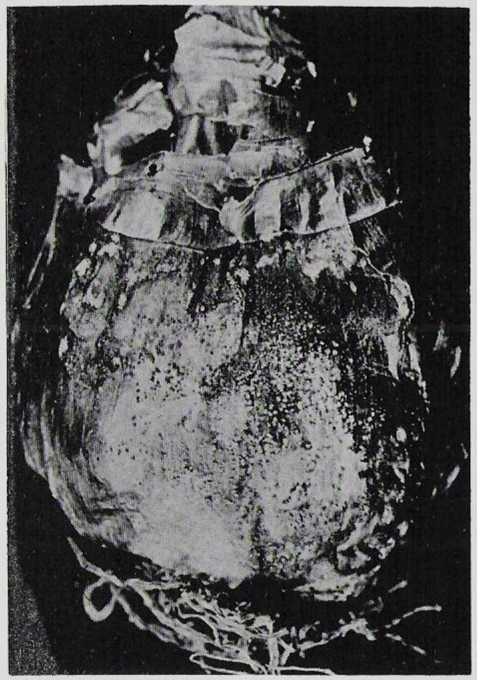


Figure 5 — Basal Rot Fungus on Outer Scales of an Infected Narcissus Bulb. (Print made from slide furnished by Mr. Willis Wheeler.)

BASAL ROT CONTROL

Factors which can minimize the losses of bulbs to basal rot include the following: 1) avoidance of high nitrogen fertilizers; 2) planting of bulbs only in raised beds or in otherwise well-drained sites, e.g. slopes and hillsides; 3) practice of crop rotation, e.g. not growing bulbs in a previously cropped area for a period of 5 - 6 years will markedly reduce fungus buildup in the soil (5), or fumigate the soil with methyl bromide to kill the basal rot fungus which is there so that crop rotation is not necessary; 4) mulching of beds to reduce soil temperatures; 5) buy bulbs only from a source known to supply basal-rot free bulbs, i.e. bulbs are not

soft when you receive them; 6) avoid planting known susceptible cultivars such as cultivars with white perianths or all white cultivars which have Madame de Graff (1 W-W) in their pedigrees (5); 7) plant cultivars which are known to have a high level of resistance to the basal rot fungus, e.g. St. Keverne (2 Y-Y); and 8) use fungicidal dips, e.g. benomyl, to minimize losses due to basal rot.

In addition to the preceding commentary, I would like to propose a specific annual procedure to be followed by the amateur daffodil grower to minimize losses due to the basal rot fungus: 1) Delay planting of bulbs until soil temperature is below 12°C (53.6°F). 2) Give bulbs a 0.5% benomyl dip, e.g. Benlate, (3 tablespoons of benomyl/gallon of water or 19.0 g benomyl/gallon of water) for 30 minutes and plant while still wet. 3) Lift bulbs as soon as the foliage has completely died down and dip bulbs in 0.5% formalin (19 ml 40% formaldehyde/gallon of water or 4 teaspoons 40% formaldehyde/gallon of water) for 15 minutes or give a benomyl dip as before. This dip, whether it is 0.5% formalin or 0.5% benomyl, should be applied preferably as soon as the bulbs are lifted but definitely within 48 hours after digging (8). 4) Hang bulbs to dry in a cool, dry, and airy place until time to plant. 5) Repeat step #1.

I believe that application of the preceding procedure for basal rot control will substantially reduce losses due to the basal rot fungus. However, it should be noted that no procedure for basal rot control can ever be expected to be 100% effective. Even though the procedure that I have outlined is potentially effective in reducing losses of bulbs due to basal rot, it is not without potential problems. Benomyl to the plant pathologist is what penicillin G is to the physician: benomyl is the most effective fungicide available for control of many fungal diseases of plants, e.g. black spot in roses which is caused by the fungus *Diplocarpon rosae,* narcissus basal rot, etc., whereas penicillin G is the antibiotic of choice for treating numerous bacterial infections. Today, many bacteria which were previously susceptible to penicillin G are now resistant to it. Unfortunately, many fungi are acquiring resistance to the systemic benzimidazole fungicides, e.g. benomyl, thiabendazole, carbendazin, furidazol, and thiophanate (9). Thus, intensive use of the benzimidazole fungicides is not recommended as it might render them ineffective in basal rot control (8). Therefore, to avoid this problem in my procedure, I would recommend a three-year alternative procedure as follows: In the first year, benomyl would be used on bulbs both at the time of lifting and planting. In the second year, formalin would be used on the bulbs at the time of lifting, and benomyl would be used on them at the time of planting. Lastly, in the third year, formalin would be used to treat the bulbs at lifting. As for the third year planting, perhaps it is time to suggest giving the bulbs a formalin dip at the time of planting. As an alternative to the formalin dip at planting in the third year, no dip of any kind would be given. Instead, the only control measure to be used would be late planting when the soil temperature is below 12°C (53.6°F). Formalin has been used for a number of years in the hot water treatment of bulbs. One of the purposes of the formalin in hot water treatment is to kill any spores of the basal rot fungus which might be present and which would conceivably contaminate all the bulbs receiving the treatment. Of course, the reason for a formalin dip at planting time would be for the same purpose. However, I am not aware of formalin ever having been used as a bulb dip at planting time. Theoretically, it would destroy any inoculum, e.g. spores, present on the bulbs at the time of planting and thereby reduce the chance of infection when the roots emerge from the basal plate of the bulb. This three-year alternative cycle is obviously time consuming and laborious for the amateur grower; however, the reward for such effort very well might be the effective management of basal rot in susceptible cultivars or in new, very expensive cultivars with which you don't wish to take any chances. No matter whether the annual or three-year alternative basal rot control procedure is followed, delay in planting until the soil temperature is below 12°C (53.6°F) is imperative if maximum basal rot control is to be achieved.

With regard to crop rotation to reduce the amount of basal rot fungus in the soil, soil fumigation with methyl bromide (2 lbs/100 square feet) is said to be effective in the control of *Fusarium* (10). However, Apt and Gould (11) reported that the use of methyl bromide (2 lbs/100 square feet) to control the root-lesion nematode of narcissus, *Pratylenchus penetrans,* unexpectedly resulted in an increased incidence of basal rot in the cultivar King Alfred. They attributed this effect to the elimination of fungi which were antagonistic to the basal rot fungus but which allowed the basal rot fungus to survive and thrive. Despite Apt and Gould's observation, fumigants like methyl bromide are generally used in control of nematodes, soil insects, and weed seeds as well as soil fungi (2). A major problem which limits the use of a soil fumigant like methyl bromide is that it is not generally available to the amateur daffodil grower. In Tennessee, it can only be obtained if you possess a pesticide applicator permit which is issued by the Tennessee Department of Agriculture after you have attended a pesticide training session.

CONCLUSION

Within this article, some basic information was presented regarding fungi in general and *Fusarium oxysporum* f. *narcissi* specifically. Much discussion was given regarding basal rot control including two proposed procedures for minimizing basal rot losses which differ substantially from most of the currently recommended procedures.

A subsequent article on fungal diseases of narcissus will cover the following diseases: fire, smoulder, scorch and others.

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ADDENDUM

Since the preceding text was written, several points have been raised which do require comment. Firstly, in addition to avoiding high nitrogen fertilizers to control basal rot, organic nitrogen sources, e.g. fresh manures, etc., and undecomposed organic matter, e.g. leaves, grass clippings, etc., should be avoided. Secondly, crop rotation of only two to three years in the cool areas of the Pacific Northwest is all that is required to significantly reduce the basal rot inoculum in the soil. Perhaps this might also be true elsewhere. Thirdly, the reference to 0.5% benomyl actually means 0.5% Benlate, Dupont's brand name for benomyl. By formulation, Benlate is 50% benomyl and 50% inert ingredients. Thus, 0.5% Benlate would actually be 0.25% benomyl. Nonetheless, the recommended concentration for a 0.5% Benlate dip is 3 tablespoons Benlate/gallon of water as previously mentioned. Lastly, the suggested use of 0.5% formalin as a pre-planting dip should be used with caution as it could possibly damage the bulb if the basal plate has already begun to swell. Thus, some research on formalin as a pre-planting dip is necessary before it can be recommended for use on a broad scale; however, 0.5% formalin can be safely used as a post-lifting dip.

Chapter 3

FUNGI AND FUNGAL DISEASES - SCORCH. SMOULDER, FIRE, AND SOME OTHERS

"Ours is a military campaign against agents that destroy our plants. We cannot wage this campaign successfully without knowing the measure of the enemy's ability to destroy." K. Starr Chester (1959)

The enemies with which we will concern ourselves in this article are the fungi, with the exception of the basal rot fungus, ***Fusarium oxysporum*** f. sp. ***narcissi,*** which was discussed in a previous article. You are fully aware of the measure of the ability of the basal rot fungus to destroy narcissus bulbs. Perhaps what you are not familiar with is the measure of the respective abilities to destroy of eight additional fungal species which also infect narcissi (1,2,3). None of these fungi are nearly so damaging to narcissus as is the basal rot fungus. Consequently, few daffodil growers other than commercial growers ever seem to pay much attention to these other fungal diseases and their control.

SCORCH

Scorch is primarily a leaf disease of narcissus which is caused by the fungus ***Stagonospora curtisii.*** This disease can be most destructive, particularly in warm, moist regions such as the Southeast (2). The fungus survives the winter on neck scales of daffodil bulbs (1,5). Thus, as the leaves emerge from the bulb, they become infected (3). This type of infection is called a primary infection as it is the initial infection caused by inoculum (fungus) already present on the bulb, i.e. primary inoculum. As primary infection involves the leaf tips (Figure 1) and causes them to turn brown, scorch is commonly confused with frost damage (3,5). Sometimes as much as one-third of the leaf beginning at the tip and extending downward will be blighted in primary infection. Secondary infections of previously healthy foliage are a consequence of spores being splashed by rain from pycnidia, sack-like structures containing spores which are embedded in the blighted leaf tips of narcissus showing primary infection. These spores (secondary inoculum) infect the leaves causing elliptical, reddish-brown lesions with dark peripheries (Figure 2). Further spread of the fungus from the secondarily infected plants to other healthy plants can occur by the splashing of spores from the lesions. If infections are particularly severe, premature dying down of foliage will occur. There is some evidence that ***Stagonospora curtisii*** and perhaps ***Botryotinia narcissicola*** (formerly ***Sclerotinia narcissicola),*** the smoulder fungus, cause a neck rot of narcissus bulbs (5). Control of primary infections may be accomplished by eliminating primary inoculum from neck bulb scales by the hot water treatment as given for nematodes, i.e.

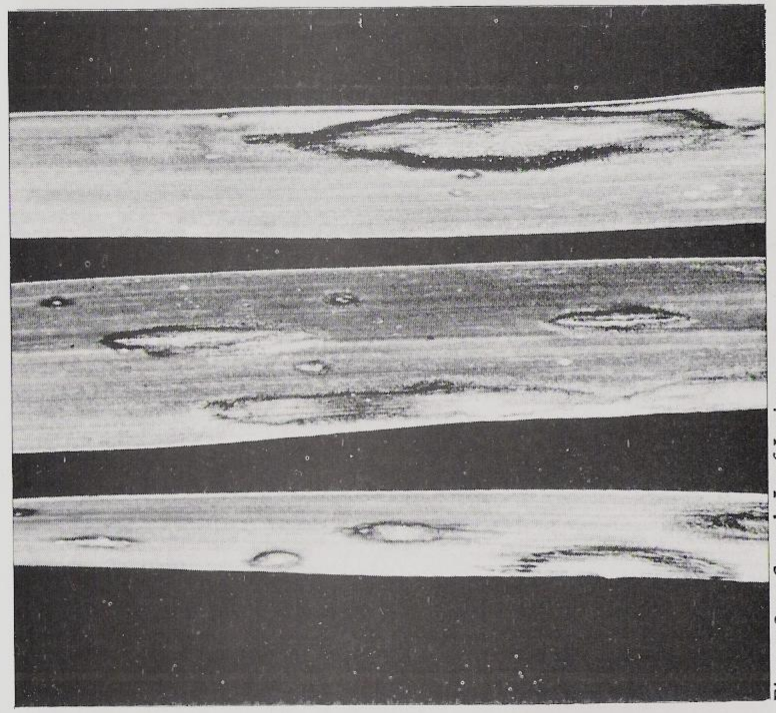

Figure 2 — Scorch Leaf Lesions
Print made from slide furnished by Dr. Gary Chastagner.
Washington State University.

Figure 1 — Scorch
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Washington State University.

0.5% formalin (½ gallon 37% formaldehyde/100 gallons of water) at 110 - 111°F (43.3 - 43.9°C) for 4 hours (4). Secondary infections may be controlled by spraying the foliage with mancozeb, zineb, or Benlate approximately three times. The first spray should be applied when the foliage is three inches above ground, secondly before bud break, and lastly after flowering (1). The neck rot stage of the scorch disease can be minimized by hot water treatment or a Benlate dip (5).

SMOULDER

Smoulder is a disease of narcissus which involves both leaves and bulbs (1). This disease is caused by the fungus *Botryotinia narcissicola*. *Botryotinia narcissicola,* formerly *Sclerotinia narcissicola,* is the perfect form of an imperfect fungus, *Botrytis narcissicola.* Recall that perfect refers to sexual reproduction of a fungus whereas imperfect refers to a fungus which reproduces asexually (6). The life cycle of the smoulder fungus is not clear. The fungus is found as sclerotia on the scales of the bulbs and near the base of flower stems (Figure 3). A sclerotium is a hardened mass of hyphae which may serve as an overwintering structure. When environmental conditions are right, it may germinate to produce conidia (spores) and/or hyphae. The conidia and/or hyphae produced from germinating sclerotia on the neck scales of a bulb do not seem to account for infection of leaves as they emerge from the bulb and break through the soil. Rather the inoculum seems to be hyphae in an infected bulb which invades the leaves before they emerge from the bulb. As the first foliage leaf emerges through the soil from an infected bulb, it curls and along the inner edge of the curled leaf will be found an elongated brown lesion (Figure 4) which often bears masses of conidia (7). Production of smoulder lesions on leaves is favored by moist conditions in poorly drained soils (7). There is little evidence to suggest that these conidia produced on the leaf lesions account for secondary spread in the growing crop (8).

Since there seems to be little or no evidence of secondary spread of the fungus from leaves of an infected plant to leaves of an uninfected plant, then the question arises as to how does the smoulder fungus move from plant to plant? What does seem to be involved is that the smoulder fungus is commonly present on bulb scales in some areas and "grows on senescent narcissus tissue and can persist from year to year on older scales of bulbs without producing symptoms above ground (on the foliage). Living, white scale tissue is invaded only where bulb scale mites, *Steneotarsonemus laticeps,* have fed (9)." Thus, the spread of the smoulder fungus in a bulb population would be due to the presence of both the smoulder fungus and the bulb scale mite on a bulb. In Scotland, hot-water treated bulbs are typically planted and lifted after two years. During the first year, the incidence of the smoulder fungus on foliage leaves is minimal; however, in the second year, the incidence is significantly increased (8). Presumably what is happening is that the bulb scale mite population has increased during the first year in bulbs which carry the smoulder fungus on their dry, outer scales. Thus, as the bulb scale mite feeds on the white, fleshy scales, tiny wounds are created which serve as portals of entry for the opportunistic smoulder fungus. Bulbs thus infected would be indicated in the second year down by the formation of lesions near the tips of leaves which have just emerged from the bulb and

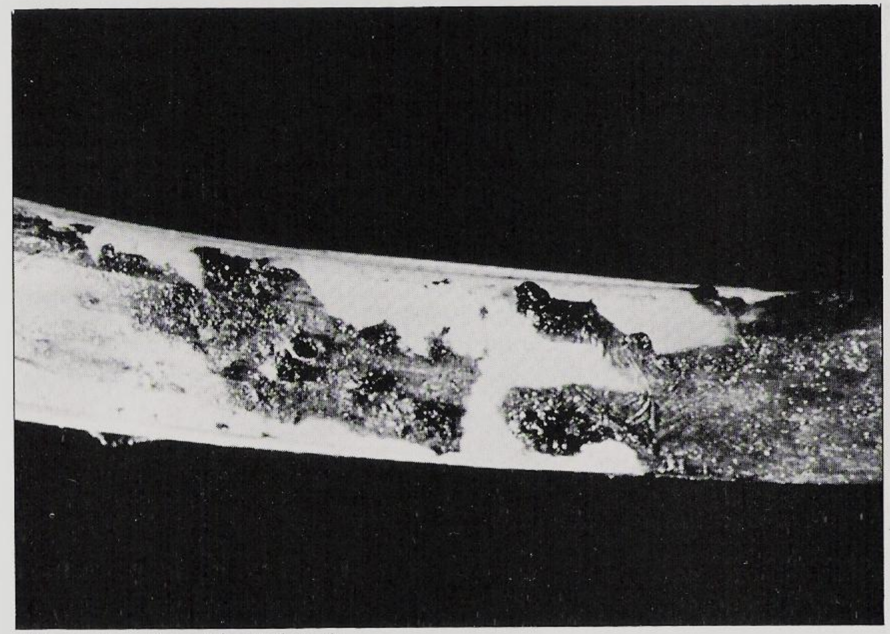

Figure 3 — Smoulder Sclerotia
Print made from slide furnished by Dr. Gary Chastagner,
Washington State University

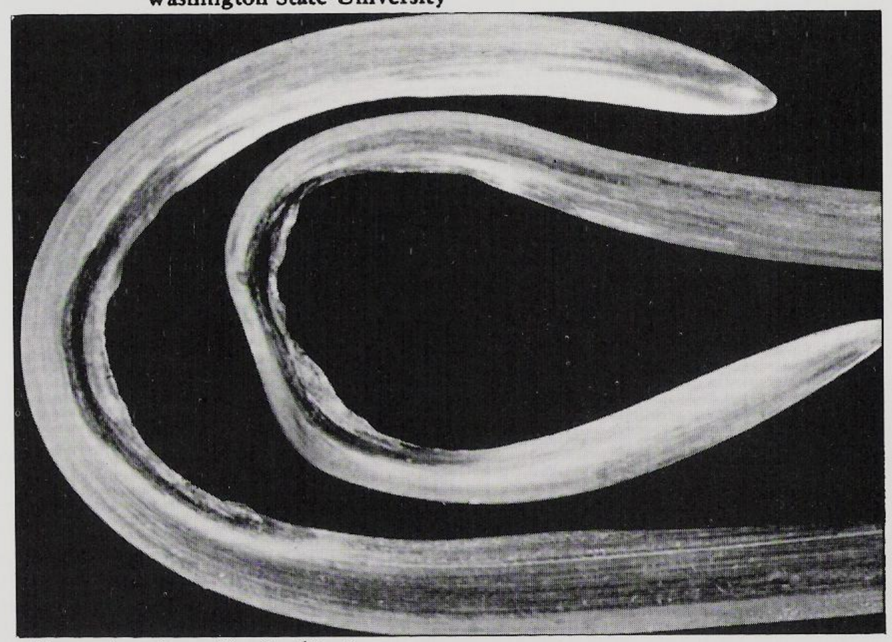

Figure 4 — Smoulder Leaf Lesions
Print made from slide furnished by Dr. Gary Chastagner,
Washington State University.

have broken through the soil. Continued feeding by the bulb scale mites on white, fleshy bulb scales and subsequent spread of the smoulder fungus through portals of entry created by the bulb scale mites would presumably account for the bulb rot which sometimes occurs. It also seems obvious that both the smoulder fungus and the bulb scale mite might spread from a mother bulb to the offsets or bulb chips. No evidence is available to suggest that the bulb mite, *Rhizoglyphus echinopus*, has any role in the spread of the smoulder fungus (8).

Control of the smoulder fungus when it is a problem might best be accomplished by annual lifting of the bulbs and dipping them for 30 minutes in 0.5% Benlate (3 tablespoons Benlate/gallon of water) or 0.5% formalin (4 teaspoons 40% formaldehyde/gallon of water) within 48 hours after lifting to reduce the smoulder fungus inoculum present on the outer bulb scales. After the bulbs are thoroughly dried out, they would be given the same hot water treatment as mentioned earlier for control of the scorch fungus to eliminate the bulb scale mite from the bulbs. Thus, the post-lifting fungicidal dip and subsequent hot water treatment might be effective in control of the smoulder fungus should it ever become a significant problem. In the Pacific Northwest, a recommended control of the disease on the foliage is spraying with mancozeb or zineb every two weeks from the time the leaves emerge from the soil (4).

FIRE

Fire is a disease of daffodils caused by *Botryotinia (Sclerotinia) polyblastis*. The disease affects the flowers, stems, and leaves but not the bulbs. Disease development is favored by warm, moist weather (3). Sclerotia in the soil debris from the preceding growing season germinate in the spring to produce many ascospores (sexually produced spores) which serve as inoculum to infect flowers. The first symptom on the flower is small, watery, brown spots. Ultimately, the entire flower may be destroyed. Conidia produced on infected flowers infect the stems below the flowers and the leaves causing elongated, brownish lesions (Figures 5,6) to develop which may coalesce causing the premature death of the foliage and flower stems. Sclerotia are then produced on the fallen leaves and stems to furnish inoculum for the next season (1). Control is difficult and involves crop rotation (3), removal of all flowers as soon as the disease is detected, and spraying with mancozeb or Benlate every 7-14 days from the time the disease is first observed until it stops spreading (4). Tests this past year have shown that mancozeb gave only marginal control while Benlate gave excellent control (11).

WHITE MOLD

White mold is a fungal disease caused by *Ramularia vallisumbrosae* (1,2,3) which affects the leaves and sometimes the flower stems. Symptoms on leaves and flower stems are greenish-white spots and streaks which are usually covered by white fungal growth (1) and later by sclerotia (3). The disease does not appear to be too common in the United States. Inoculum for infection comes from conidia or spores produced by germinating

Figure 5 — Fire Leaf Lesions
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Washington State University.

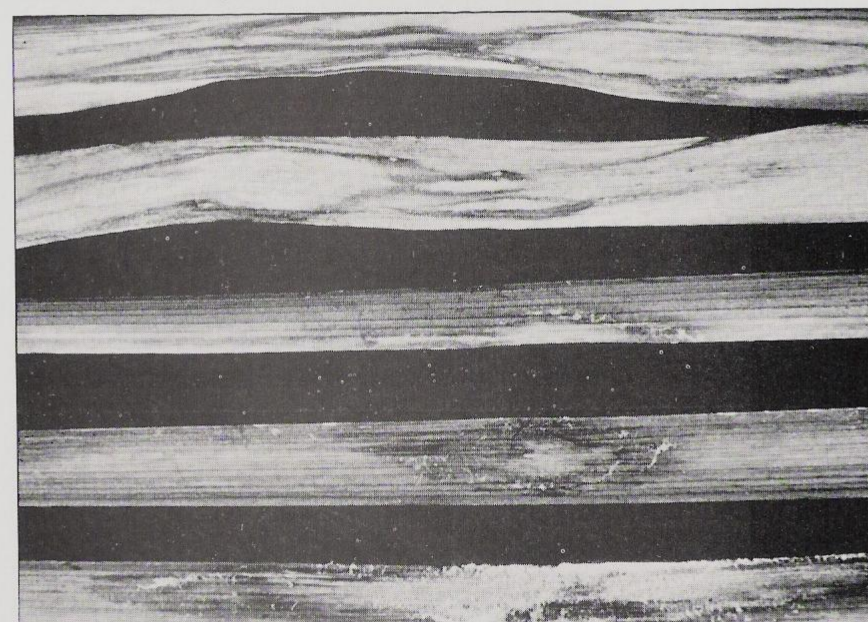

Figure 6 — Fire Leaf Lesions
Print made from slide furnished by Dr. Gary Chastagner,
Washington State University

sclerotia which have persisted on decayed leaves, etc. (1). The disease can be controlled by foliar sprays of mancozeb, zineb, or Benlate which are given in the same prescribed manner as for control of scorch. Other control measures are crop rotation and cleansing away of debris which might contain sclerotia from the neck of the bulb (3).

WHITE ROOT ROT

White root rot is an important disease in the Isles of Scilly (1) but either does not occur or only occurs rarely in the United States. It is caused by the fungus *Rosellinia necatrix.* The fungus causes "a black rot of outer (bulb) scales and white strands of fungus may be seen on or near the basal plate (1)." A wide range of herbaceous and woody plants serve as a reservoir of the fungus (1). Thus, when the soil temperatures are high, bulbs may become infected by the fungus if it is present in a plant reservoir (1). There does not seem to be any economic control measure available at this time.

CROWN ROT

Crown rot or wet scale rot (3) is a disease caused by *Sclerotium rolfsii* which can rot bulbs in the soil (2). The fungus appears as a whitish growth on or between the bulb scales. Also present are reddish-brown sclerotia on or between the scales (2). This fungus may persist in the soil for long periods of time (3), probably as sclerotia. Thus, germinating sclerotia in close proximity of an uninfected daffodil bulb would lead to its infection. Control involves hot-water containing 0.5% formalin treatment (2) of the bulbs and crop rotation. Also, treating of the soil with terraclor is said to be effective in reducing the fungal inoculum in the soil (3).

SOFT ROT

Soft rot of narcissus bulbs is a storage disease caused by the ubiquitous common black bread mold, *Rhizopus stolonifer* (2,3). Rot of bulbs as a consequence of infection by the ever-present spores of *Rhizopus stolonifer* is favored by dense packing of bulbs, high temperatures, and lack of ventilation (3). Also, rot is favored by mechanical injury or sunburn injury of the bulbs (2). Control of this disease is by avoiding injury of the bulbs, keeping the bulbs cool and well-ventilated during storage and shipment, and by drying and cooling the bulbs quickly after hot water treatment (2).

BLUE MOLD

Blue mold is a storage or transit disease of bulbs which is caused by various *Penicillium* species (2). Infection by spores or conidia of this ever-present fungus is favored under cool, moist conditions when the bulbs have not been dried rapidly enough after fungicidal dips or hot-water treatment (2). The author has noted this fungus on the outer bulb scales of bulbs which had been received in parcels from both England and Northern Ireland. The bulbs did not seem adversely affected and no losses were noted the next spring. Avoidance of blue mold is achieved by keeping the relative humidity below 70% during storage and by avoiding injury of the bulbs (2).

CONCLUSION

Your author hopes that this article and the preceding one on basal rot have served their intended purpose which is to educate, but not unduly alarm, the enthusiast so that he (she) might make the necessary decisions regarding disease control and prevention when the relatively rare phenomenon of disease presents its ugly head. Perhaps the statement made by R. K. S. Wood in 1973 (10) is what is needed for reassurance: "We cannot remind ourselves too often that disease is a relatively rare phenomenon and that particular pathogens are able to parasitize (infect) only a very small proportion of the plants available to them."

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VIRUSES AND VIRUS DISEASES

"The infection is not caused by microbes, but by a *contagium vivum fluidum.*" M. W. Beijerinck (1898)

During the latter years of the past century, considerable attention was given to a disease in tobacco which was called "mosaic." In 1886, Adolph Mayer demonstrated the infectious nature of the etiological agent which causes tobacco mosaic by taking sap from diseased tobacco plants and inoculating it into healthy plants which subsequently developed the mosaic symptom (1). Mayer erroneously concluded that the cause of tobacco mosaic was a bacterium despite his not being able to isolate such a microbe. In 1890, Dimitrii Ivanowski demonstrated that the etiological agent of tobacco mosaic could pass through a bacteria-proof filter (Chamberland filter candle) and still induce the mosaic disease of tobacco. Despite this observation of the filterability of the tobacco mosaic agent, Ivanowski, like Mayer before him, concluded incorrectly that tobaco mosaic was caused by a bacterium. Finally, in 1898, M.W. Beijerinck (Figure 1) demonstrated again the filterability of the tobacco mosaic etiological agent through a bacteria-proof filter and concluded that tobacco mosaic was caused by a *contagium vivum fluidum,* i.e. a contagious living fluid. This conclusion was significant because it suggested that tobacco mosaic was not caused by a bacterium but by something more novel. Interestingly, in the same year of Beijerinck's work with tobacco mosaic, Loeffler and Frosch also demonstrated that the etiological agent of foot and mouth disease of cattle could be passed through a bacteria-proof filter (2). Thus, the field of virology was begun.

WHAT IS A VIRUS?

A simple definition for a virus is that it is an infectious macromolecule which replicates (reproduces) itself only inside a living cell. Although viruses are sometimes thought of as obligate, intracellular parasites, the term parasite is usually reserved for etiological agents which are cellular or multicellular in nature, not subcellular as are the viruses, and which are thought of as living organisms. However, the viruses are usually not considered to be living unless the only criterion for living is the ability to reproduce. Nonetheless, the reference to a virus as an obligate, intracellular parasite is convenient if one remembers that this usage simply means that the virus is obligated to an intracellular (within the cell) existence and can not be cultured outside of the living cell.

Virion is the term used for a single virus particle. A virion consists of two parts: 1) a nucleic acid, usually ribonucleic acid (RNA), which serves as the genome or genetic material of the virion, and 2) the capsid which is a protein coat which encloses the nucleic acid. The part of the virion which initiates infection in a healthy plant is the nucleic acid whereas the capsid merely serves to protect the nucleic acid from destruction when the virion is in the extracellular (outside of the cell) state.

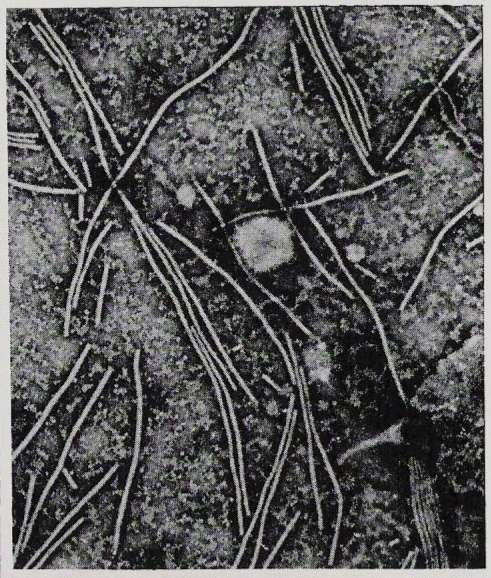
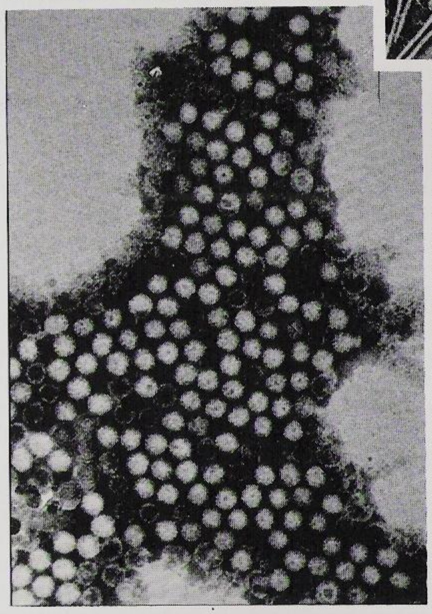





Figure 1, top left: — M. W. Beijerinck (1). Photograph used by permission of the American Phytopathological Society.)

Figure 2, top right: — Electronmicrograph of Narcissus Mosaic Virus. (Used by permission of Dr. A. A. Brunt, Glasshouse Crops Research Institute, England.)

Figure 3, bottom left: — Electronmicrograph of Narcissus Tip Necrosis Virus. (Used by permission of Dr. A. A. Brunt, Glasshouse Crops Research Institute, England.)

Figure 4, bottom right:Electronmicrograph of Tobacco Rattle Virus. (Furnished by Dr. R. M. Lister, Purdue University.)

Plant virus virions which infect narcissus have several capsid shapes or forms: 1) the isometric or spherical form, and 2) the helical form. Two types of helical forms are the filamentous where the virion is flexuous, and the rod-shaped where the virion is rigid. Exemplary of the isometric form is narcissus tip necrosis virus (Figure 3) whereas narcissus mosaic virus (Figure 2) is representative of the filamentous form. Tobacco rattle virus is an example of a virion which is rod-shaped (Figure 4). The capsid of all virions is comprised of protein subunits.

Recall that plant viruses are named according to the plant infected and the symptoms caused, e.g. narcissus yellow stripe virus, and that an abbreviation for the virus is commonly used which is derived from the first letter of each word in the virus name, e.g. NYSV for *n*arcissus *y*ellow *s*tripe *v*irus (3).

NARCISSUS VIRUSES

Throughout the past sixteen years, viruses infecting narcissus have received considerable attention in daffodil literature (4,5,6). It is interesting to note that the first edition of *The Daffodil Journal* contained an article by Harold S. King on daffodil viruses (4). At the time of this writing, seventeen different viruses have been described which naturally infect narcissus (2,5,6,7,8,9,10). Of these seventeen viruses, nine have isometric particles or virions and eight have filamentous or rod-shaped particles or virions (Table 1). Five of these viruses (jonquil mild mosaic, narcissus degeneration virus, narcissus yellow stripe virus, narcissus white streak virus, and narcissus tip necrosis) are known to occur only in narcissus (7). Narcissus mosaic virus is known to naturally occur in both narcissus and nerine whereas narcissus latent virus infects narcissus, nerine, and bulbous iris (7). Carnation latent virus has been found only in the cultivar Grand Soleil d'Or (10) and has only a limited host range (2). Tobacco rattle virus, which was first isolated from narcissus in Holland, has also been found infecting a number of other plant species (5,8). The remaining eight narcissus virus (arabis mosaic virus, broad bean wilt virus, cucumber mosaic virus, raspberry ringspot virus, strawberry latent ringspot virus, tobacco ringspot virus, tomato black ring virus, and tomato ringspot virus) have extensive natural host ranges (7).

VECTORS

Recall that a vector is a biological agent which carries the pathogen, e.g. virus, from a diseased plant to a healthy plant (3). Principally, aphids and nematodes are the vectors of most of the narcissus viruses; however, some narcissus viruses have no known vector and are said to be transmitted only by mechanical means, e.g. handling the foliage, cutting, cultivation, etc. (Table 2).

Since daffodils are rarely colonized by wingless aphids (apterae), many growers find it difficult to accept that a number of the viruses which are found infecting narcissus are transmitted by aphids (5,8,9). It is the winged forms of aphids (alatae) which are believed to be largely responsible for the spread of aphid-transmitted viruses of narcissus. Evidence for this comes from trapping experiments in England where large numbers of alatae were found to visit daffodils (5,8,9). In a specific case, nine different species of aphids have been shown to be able to

TABLE 1 - NARCISSUS VIRION MORPHOLOGY (2, 5, 6, 7, 8, 9, 10)

Isometric or spherical viruses[1]	Rod-Shaped and filamentous viruses
arabis mosaic virus (AMV)	carnation latent virus (CLV)[4]
broad bean wilt virus (BBMV)	jonquil mild mosaic virus (JMMV)[5]
cucumber mosaic virus (CMV)	narcissus degeneration virus (NDV)[3]
narcissus tip necrosis virus (NTNV)	narcissus latent virus (NLV)[4]
raspberry ringspot virus (RRSV)	narcissus mosaic virus (NMV)[3]
strawberry latent ringspot virus (SLRSV)	narcissus white streak virus (NWSV)[2]
tobacco ringspot virus (TRSV)	narcissus yellow stripe virus (NYSV)[3]
tomato black ring virus (TBRV)	tobacco rattle virus (TRV)[6]
tomato ringspot virus (TRSV)	

[1]All these virions have a diameter of approximately 30 nm. A nanometer (nm) is one-billionth of a meter (m), i.e. 0.000000001 m.
[2]Filamentous particle is approximately 750 nm in length.
[3]Filamentous particles range from approximately 548 nm to 568 nm in length.
[4]Filamentous particle is approximately 650 nm in length.
[5]Filamentous particle of unreported length.
[6]Rod-shaped particles of two lengths: 190 nm and 110 nm.

TABLE 2 - VECTORS OF NARCISSUS VIRUSES (2, 5, 6, 7, 8, 9, 10)

Aphid vectors	Nematode vectors	Mechanical means
broad bean wilt virus (BBWV)	arabis mosaic virus (AMV)	narcissus mosaic virus (NMV)
carnation latent virus (CLV)	raspberry ringspot virus (RRSV)	narcissus tip necrosis virus (NTNV)
cucumber mosaic virus (CMV)	strawberry latent ringspot virus (SLRSV)	
jonquil mild mosaic virus (JMMV)	tobacco rattle virus (TRV)	
narcissus degeneration virus (NDV)	tobacco ringspot virus (TRSV)	
narcissus latent virus (NLV)	tomato black ring virus (TBRV)	
narcissus yellow stripe virus (NYSV)	tomato ringspot virus (TRSV)	
narcissus white streak virus (NWSV)		

transmit NYSV from diseased to healthy narcissus plants (5). All the aphid-transmitted viruses of narcissus are probably non-persistent viruses. Persistence is the term for the time a vector remains infective, i.e. capable of transmitting the virus after it is acquired from a diseased plant. A non-persistent virus is one in which the vector remains infective for only a few hours, usually less than four (2). Basically what is meant by saying that a virus is non-persistent is that the virus is acquired quickly by the aphid while it is feeding on a diseased plant and is also transmitted quickly when that same aphid subsequently feeds on a healthy plant. As a matter of fact, non-persistent viruses can be acquired by a feeding aphid in as little as 10 to 30 seconds and subsequently transmitted to a healthy plant in the same short period of time (2). Aphid-transmitted narcissus viruses, in addition to being non-persistent, are often called stylet-borne. The stylet is one of the aphid's mouth parts which is used in feeding on a plant. A stylet-borne virus is one which is carried on the stylet of the aphid. Thus, as an aphid feeds, it inserts its stylet into the diseased plant, acquires the virus externally, and subsequently inoculates a healthy plant when it again inserts its stylet to feed. There is some controversy over the way non-persistent viruses are transmitted by their aphid vectors; therefore, the use of the term stylet-borne should perhaps be avoided for the meantime (2). There is no evidence to suggest that the aphid-transmitted narcissus viruses are also seed or pollen-borne (8). Thus, the seed produced from crosses between plants which were infected by aphid-transmitted viruses will be free of virus. Although all the aphid-transmitted narcissus viruses are mechanically-transmissible, neither handling of plants nor the cutting knife probably account for much transmission of these viruses. Instead, it is only the aphid which carries these viruses from plant to plant. Of course, the offsets or bulblets of a virus-infected bulb will also be virus-infected. However, NMV, which has no known vector, is easily transmitted by mechanical means.

All the nematode-transmitted narcissus viruses seem to be transmitted by only three genera of nematodes (5,8). It is important to note that neither the bulb and stem nematode, ***Ditylenchus dipsaci,*** nor the root lesion nematode, ***Pratylenchus penetrans,*** serve as vectors of any of the narcissus viruses. Tobacco rattle virus (TRV) seems to be only transmitted by *Trichodorus* species and the other six viruses by *Longidorus* or *Xiphinema* species (5,8). As with aphids, the nematode vectors have mouth parts which include a stylet which is inserted into plant cells as the nematode feeds. The mechanism of acquisition and transmission of the virus by the nematode is quite involved; however, the insertion of the stylet into plant cells is involved with both virus acquisition and transmission. The nematode vectors of narcissus viruses often feed on young tissue near root tips. With regard to acquisition and transmission of viruses, the viruses can be acquired in 15 minutes to 1 hour of feeding and transmitted in a similar period (2). Once a virus has been acquired by a nematode vector, it can be retained for weeks (2). Of the seven narcissus viruses having nematode vectors, only the four ringspot viruses are known to be seed-borne in narcissus (5).

SYMPTOMS

Recall that a symptom is the visible effect of the pathogen on the host, e.g. yellow stripes on the foliage of a NYSV-infected daffodil (3). At best, the identification of a plant virus disease by symptom expression is an inexact science. Perhaps plant disease diagnosis by the reading of symptoms is an art rather than a science. The preceding statements seem to be particularly well-suited for diagnosis of virus diseases in narcissus. The reason for this is that symptom expression in a virus-infected plant can be modified from the expected by several factors: 1) Symptom expression in a particular virus disease may deviate from the expected due to differences in genotype (genetic constitution of a particular cultivar) of the infected cultivars; 2) environmental conditions, e.g. temperature, soil

pH, soil fertility, etc., may also modify symptom expression in an infected cultivar; and 3) cultivars infected with a virus-complex, i.e. several different viruses infecting the same plant, may have masking of symptoms of one or more of the viruses, or the virus-complex may cause a synergistic effect where the symptom expression of the several viruses acting together is greater or more severe than the sum total of the individual viruses acting independently. In spite of the foregoing, having a "learned eye" is particularly useful in recognizing what may indeed be a virus problem in a daffodil planting.

Carnation latent virus (CLV)

The author could find nothing in the literature available to him regarding symptom production of CLV in narcissus. As the virus name implies, the virus probably produces no readily apparent symptoms in carnation; perhaps the same is also true in narcissus. As mentioned earlier, this virus has only been reported to be found in the cultivar Grand Soleil d'Or which had come from Israel (10).

Broad bean wilt virus (BBWV)

In work done in Japan, BBWV was isolated from narcissus (7); however, the author could find nothing in the literature available to him regarding symptom expression in narcissus.

Cucumber mosaic virus (CMV)

Symptomatology of CMV alone in narcissus is not known as it usually occurs in a complex with several other viruses. It commonly occurs in Grand Soleil d'Or but rarely in trumpet daffodils (5,8).

Jonquil mild mosaic virus (JMMV)

Very little is known about JMMV except that it is prevalent in *N. jonquilla* and causes a mosaic (5,8). In mosaic diseases, the leaves show a "patchwork of discrete, and usually unchanging, dark and light green areas (2)."

Narcissus degeneration virus (NDV)

In work with virus-free plants of the cultivar Grand Soleil d'Or, NDV causes conspicuous chlorotic (yellow) leaf streaking and color breaking in the flowers (10). It would appear that NDV infects only cultivars derived from *N. tazetta* (10).

Narcissus latent virus (NLV)

This virus produces inconspicuous symptoms in narcissus (8). It is common in many cultivars of narcissus (14).

Narcissus yellow stripe virus (NYSV)

Narcissus yellow stripe virus causes conspicuous yellow stripes on the foliage (Figure 5). Symptoms of NYSV typically appear early in the growing season and fade as the season progresses. These symptoms could easily be confused with those caused by tobacco rattle virus (Figure 6). Color breaking of perianth segments is also caused by NYSV (Figure 7). Color breaking usually shows up as white stripes or splotches in the perianth segments. In some cultivars, e.g. King Alfred, tolerance to NYSV is seen and the foliage symptoms are masked (6).

Narcissus white streak virus (NWSV)

Foliage symptoms of NWSV (Figure 8) begin first as purple stripes at the tips of leaves some 3-6 weeks after flowering and finally become white stripes (5,8). The lesions often coalesce and become necrotic, causing the foliage to mature or die down prematurely (5,8).

Figure 5, top left: Symptoms of Narcissus Yellow Stripe Virus. (Photograph made from slide furnished by Dr. Gary Chastagner, Washington State University, and used by his permission.)
Figure 6, top right: Symptons of Tobacco Rattle Virus. Left — healthy, right — diseased. (Photograph made from slide furnished by the Bulb Research Centre, Lisse, The Netherlands.)
Figure 7, bottom left: Color Break in Cultivar Infected with Narcissus Yellow Stripe Virus. (Photograph made from slide furnished by Willis H. Wheeler and used with his permission.)
Figure 8, bottom right: Symptoms of Narcissus White Streak Virus. (Photograph made from slide furnished by Dr. Gary Chastagner, Washington State University, and used with his permission.)

Tobacco rattle virus (TRV)

Tobacco rattle virus, as mentioned earlier, causes stripes (Figure 6) on the foliage which resembles those caused by NYSV (5,8). Complexes of TRV with other viruses, e.g. NYSV, often occur in narcissus (6,8).

Other nematode-transmitted viruses

Symptom expression due to TRV has already been discussed. Arabis mosaic virus (AMV), strawberry latent ringspot virus (SLRSV), and tomato black ring virus (TBRV) commonly occur in both trumpet daffodils and Grand Soleil d'Or; however, they usually do not express any symptoms (5,8,10). Tomato ringspot virus (TRSV) and raspberry ringspot virus (RRSV) have also been reported in narcissus, presumably causing symptomless infections (5,8).

Narcissus mosaic virus (NMV)

Infections caused by NMV are marked by a mild mosaic, e.g. small, alternating light and dark green areas on both foliage and flower stems (5,8). Narcissus mosaic virus is widespread in cultivars which are trumpets, large cups, and doubles; however, NMV is not found in *N. jonquilla* or *N. tazetta* (11). Some older cultivars like King Alfred, Fortune, and Golden Harvest are totally infected with NMV (11).

Narcissus tip necrosis virus (NTNV)

In cultivars which show symptoms to NTNV, expression begins by the formation of elongated chlorotic areas near the leaf tips. These chlorotic areas later turn brown and necrotic with a yellow periphery (Figure 9). Ultimately, the tip necrosis proceeds toward the base of the leaf causing a premature leaf senescence (7).

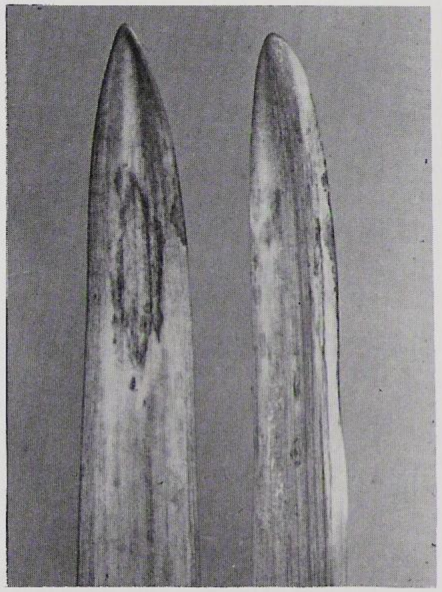
Chocolate spot

Chocolate spot (Figure 10) is a disease of narcissus which is of suspected viral etiology. Perhaps it is due to a virus-complex (6). The obvious symptom of chocolate spot is the brown foliage lesions or spots.

EFFECTS OF VIRUS INFECTION

The effects of plant viruses on narcissus cultivars is often times less than obvious. Certainly, the leaf, flower stem, and flower symptoms of a virus infection would be effects; however, that is not what is at issue here. Instead, effects of virus infection here include such things as deterioration of a cultivar over a period of years as a consequence of infection by NYSV (8). Likewise, the infection of cultivars by NWSV results in early senescence of foliage which results in reduction of bulb yield by 30-40% within 2-4 years from the time the bulbs became infected (8). With reduction in bulb yield in a NWSV-infected cultivar, it is easy to see the effect of a virus infection on a cultivar; however, with deterioration of a cultivar as a consequence of virus infection, e.g. NYSV, the effect is perhaps less than obvious. Perhaps the best way to look at cultivar deterioration as a consequence of virus infection is to look at the only example available of a formerly virus-infected cultivar which has been freed of its viruses by meristem culture, e.g. Grand Soleil d'Or.

Grand Soleil d'Or is a cultivar of unknown origin which was registered first in 1890; however, it has been grown commercially for over 100 years on the Isles of Scilly (10). Over 200 acres of this cultivar, which is not frost-hardy and cannot be field grown elsewhere in Britain, are grown on the Isles of Scilly (10). Grand Soleil d'Or has deteriorated in vigor and productivity over the past 65 years, a deterioration which is due to the



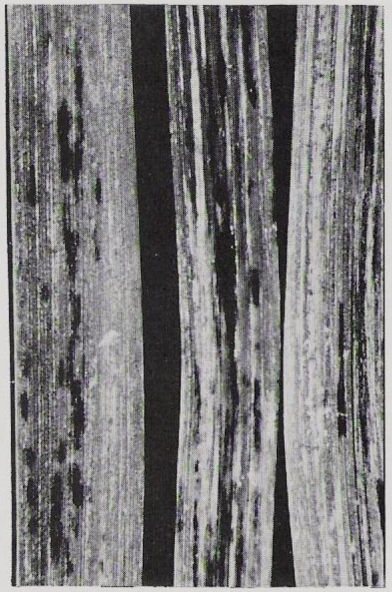


Figure 9, left: Symptoms of Narcissus Tip Necrosis Virus. (Photograph made from slide furnished by the Bulb Research Centre, Lisse, The Netherlands.)

Figure 10, right: Symptoms of Chocolate Spot Virus. (Photograph made from slide furnished by Dr. Gary Chastagner, Washington State University, and used with his permission.)

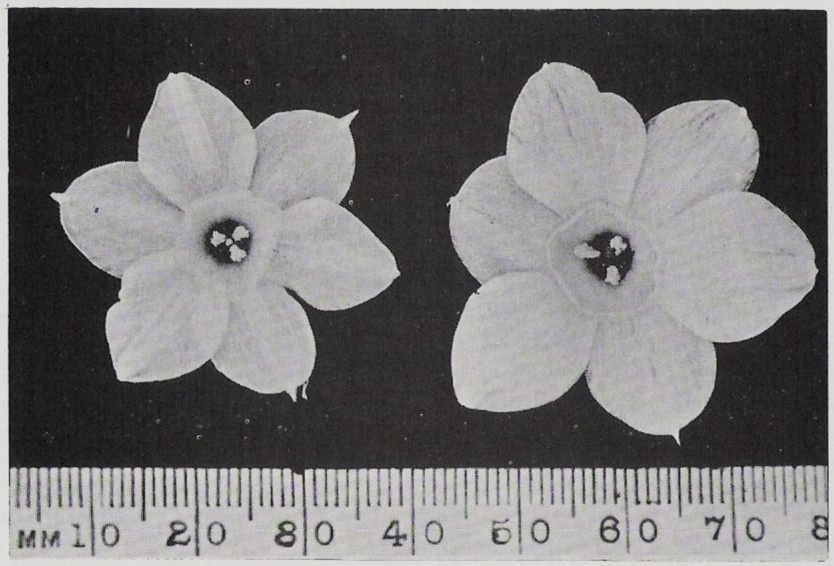


Figure 11 — Grand Soleil d'Or. Smaller flower is infected by NDV and AMV; larger flower is virus-free. (Photograph copied with permission from the Glasshouse Crops Research Institute Annual Report 1977.)

total infection of the cultivar by viruses (10). All samples of Grand Soleil d'Or examined have been found to be infected by NDV and AMV (10). Additionally, this cultivar has often been shown to be infected by other viruses in addition to NDV and AMV, e.g. CMV, TBRV, and SLRV (10). Grand Soleil d'Or, which has been freed of viruses by meristem culture, shows the following: 1) virus-free Grand Soleil d'Or produced twice as many flowers/bulb as did infected bulbs although the number of flowering stems/bulb was only 54% greater than that of virus-infected bulbs; 2) yielded 8.9 flowers/stem as opposed to 6.8 flowers/stem as did infected bulbs; 3) produced flowers which are larger and of richer color than those of virus-infected plants (Figure 11); 4) produced bulbs which showed 239% the weight gain of infected bulbs; and 5) produced 2.94 times as many offsets/bulb as did virus-infected bulbs (10). Wheeler (12) personally observed Grand Soleil d'Or growing in the Isles of Scilly and noted that the virus-free cultivar seemed to grow about twice as tall as the virus-infected cultivar. Thus, from the preceding discussion, it is obvious that what is meant by deterioration of a cultivar can't really be appreciated until one can observe the growth of a virus-free stock of the same cultivar.

CONTROL

Control of narcissus viruses which are aphid-transmitted can only be accomplished by thorough rogueing of all virus-infected stocks so as to remove the source of inoculum for the visiting aphids. With control of the nematode-transmitted viruses, two control measures practiced concommitantly are advised: 1) Rogue and destroy all infected bulbs, and 2) fumigate the soil, e.g. methyl bromide, etc. to rid it of the nematode vectors. With mechanically-transmitted narcissus viruses, e.g. NMV and NTNV, cutting knives and cultivating tools should be regularly disinfected, e.g. trisodium phosphate dip, when moving from cultivar to cultivar.

Control of narcissus viruses in the future is of course speculative to say the least; however, it is through such speculations that new control measures will come. With the aphid-transmitted viruses, control may be accomplished by spraying the foliage with a water/mineral oil emulsion. Such sprays have already been found to interfere with the aphid transmission of some viruses, e.g. potato virus Y (PVY), a filamentous virus which infects potatoes (2,13). The development of genetically-resistant cultivars is an avenue to control of viruses in narcissus which is yet unexplored. However, the use of genetic resistance to viruses is used in a number of agronomic crops today. Control of nematode-transmitted viruses seemingly will continue to be handled by rogueing and the use of soil fumigants. Use of insecticides to control aphid vectors of narcissus viruses does not seem promising as aphids acquire and transmit their viruses too quickly for insecticides to be effective in control.

Although meristem culture as used to free Grand Soleil d'Or of viruses is not really a preventive measure, it does offer the attractive possibility of recovering a desirable cultivar of which there are no known virus-free stocks.

Culture of new cultivars in aphid-proof, screened areas covering soil which has been fumigated offers hope for the introduction of new cultivars which are not already virus-infected as the screening would prevent access to the plants by aphid vectors and fumigation would kill nematode vectors (5).

SUMMARY

Within this article, the attempt was made to discuss viruses in general and narcissus viruses specifically, virus vectors, symptoms and effects of virus infection in narcissus, and control measures for today and possibilities for tomorrow.

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Chapter 5

NEMATODES AND NEMATODE DISEASES

"Many bulb growers look upon the disease (bulb and stem nematode disease) as 'one of Nature's gifts' and are of the opinion that the bulbs will ultimately right themselves. Suffice it to say that if the bulbs are left to themselves, the bulb industry will soon cease to exist." J. K. Ramsbottom (1917)

The exact date at which nematodes became a problem in narcissus is not really known; however, by 1917 the narcissus bulb industry in England and Holland was at a virtual collapse. The euphemistic thought of growers of that day that the bulb and stem nematode disease was 'one of Nature's gifts' had been transformed to one of despair (1). In 1917, the bulb and stem nematode, which had decimated the daffodil plantings of England and Holland, was called *Tylenchus devastratix* (1). Today, the bulb and stem nematode is named *Ditylenchus dipsaci* (2). Amid this devastation to the narcissus bulbs and the concomitant despair of the growers entered James Kirkham Ramsbottom and his hot water treatment of narcissus bulbs to rid them of the bulb and stem nematode (1). Indeed, Ramsbottom did give 'new life' to the narcissus!

In addition to the bulb and stem nematode, *Ditylenchus dipsaci,* there is another nematode which is sometimes a serious problem in narcissus, the root lesion nematode, *Pratylenchus penetrans* (2). Although it is comparatively rare, the bulb and leaf nematode, *Aphelenchoides subtenuis,* has been reported on occasion from the British Isles in Cornwall and on the Isles of Scilly (2).

WHAT IS A NEMATODE?

For years, the English have referred to the nematode as an eelworm; however, the proper term nematode comes from a Greek word, *nematos,* which means thread. Thus, the nematodes are thread-like worms which are classified as follows:

Kingdom — Animalia
Phylum — Nemathelminthes or Aschelminthes
Class — Nematoda

All the nematodes are unsegmented roundworms having bilateral symmetry and a cylindrical form as the name roundworm implies. The exception to this generalization is that a few species have females which become ovoid in shape. All the nematodes have a noncellular cuticle (external covering) which must be moulted and secreted anew by the growing nematode. Additionally, all the nematodes have a digestive system, rudimentary nervous system, reproductive system, rudimentary excretory system, and a muscular system (longitudinal muscles only); however, nematodes do not have a respiratory or circulatory system. All plant parasitic nematodes have stylets which they inject into the host plant cells to derive nutrients. Plant parasitic nematodes are small. The infamous *Ditylenchus dipsaci* adult is approximately 1250 microns in length whereas the root lesion nematode adult, *Pratylenchus penetrans,* is approximately 600 microns in length (3). A micron is one-millionth of a meter. Thus, it is easy to see that individual plant parasitic nematodes are microscopic in size and can only be seen with aid of an ordinary light microscope. In many nematode species, the sexes are separate. Thus, there are both male and female nematodes. In these species, the male adult nematode furnishes the sperm which fertilizes the egg which is produced by the female adult nematode. From this fertilized egg will ultimately come the adult nematode. Secondly, some nematode species are hermaphroditic, i.e. the adult nematode has both male and female gonads. In these hermaphroditic nematodes, the male gonad is usually quite small in comparison with the female gonad. In some instances, the male gonad first produces sperm which is subsequently stored; then the male gonad atrophies. Of course, the ovary would then produce the eggs which are subsequently fertilized by the sperm which had been stored. Lastly, in a few nematodes which are parthenogenic, only the female gonad is present and the eggs which are produced develop into larvae without fertilization ever having taken place. The generalized life cycle of a plant parasitic nematode involves the fertilized egg, four larval stages, and finally the adult nematode. The going from one larval stage to the next involves moulting, i.e. shedding of the cuticle. Several stages of the life cycle of *Ditylenchus dipsaci* are shown in Figure 1.

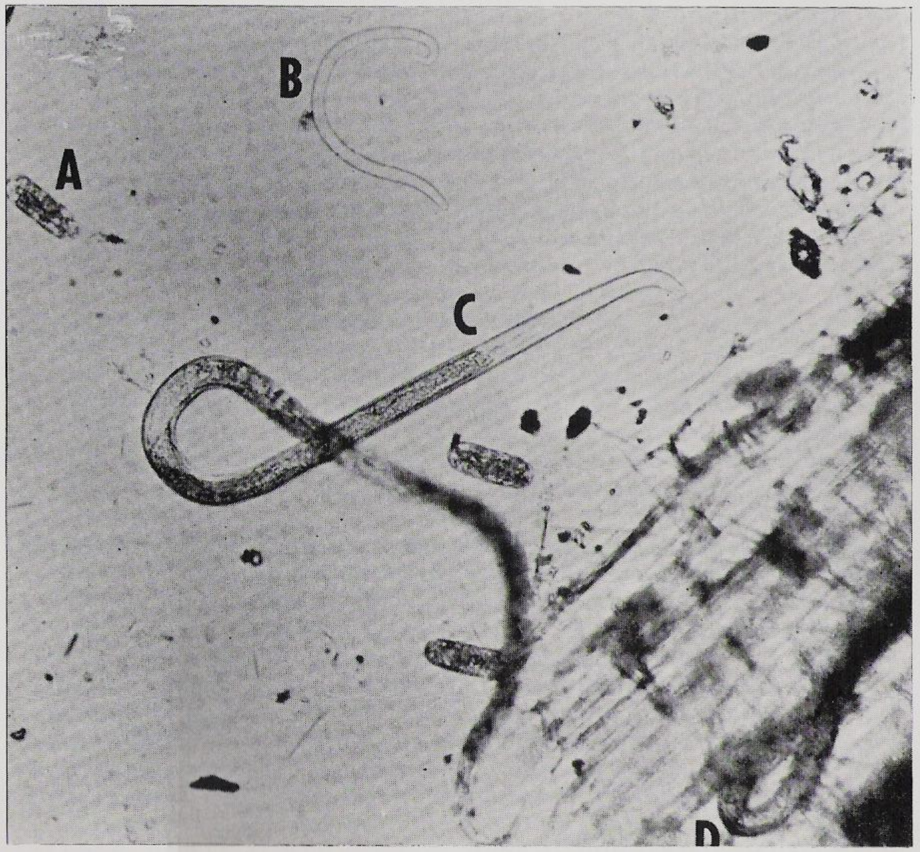


Figure 1 — Photomicrograph of Narcissus Bulb and Stem Nematode. A - egg, B - larva, C - adult, D - adult in leaf tissue

WHERE ARE NEMATODES FOUND?

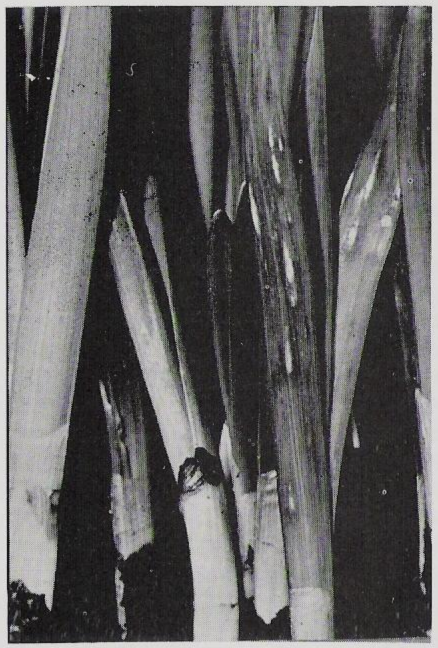
Nematodes can be found in marine, fresh water, and terrestrial habitats. The nematodes are the most numerous of the multicellular organisms found in the soil. Irregardless of their habitat, all nematodes require a film of moisture around their bodies. Some nematode species feed on fungi in the soil while others feed on soil bacteria. Also, some nematode species are parasitic on man and other animals. Lastly, there are an estimated 2000 species of plant parasitic nematodes (4). Some 90 - 95% of the plant parasitic nematode species infect the below ground parts of a plant, e.g. roots, bulbs, etc., whereas the remaining 5 - 10% infect the above ground parts of the plants, e.g. stems, leaves, seeds, etc. Another term which is used for plant parasitic nematodes is phytonematodes (4).

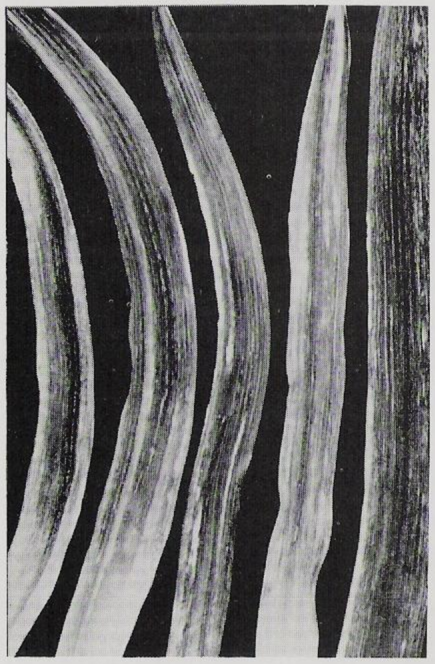
The phytonematodes are divided into two basic categories: 1) ecto-parasitic nematodes are nematodes which remain outside the plant and penetrate with only a small portion of their bodies, and 2) endoparasitic nematodes are nematodes which enter tissues completely or with a large portion of their bodies (4). The discussion here will be limited to the endoparasitic nematodes as no ectoparasitic nematodes have been reported to parasitize narcissus. All the endoparasitic nematodes which infect narcissus, e.g. *Ditylenchus dipsaci, Pratylenchus penetrans,* and *Aphelenchoides subtenuis,* are said to be migratory endoparasitic nematodes (4). All stages of this type nematode, e.g. egg, larva, and adult, may be found either in the soil or in the host plant.

PHYTONEMATODES OF NARCISSUS

BULB AND STEM NEMATODE

Several bulb and stem nematodes, *Ditylenchus dipsaci,* usually enter the bulb from the soil in the region of the neck (2). The inoculum is nematodes which leave infected bulbs and move through the soil to infect healthy bulbs (2). There they invade the young leaf tissue (5). Some of the nematodes are carried upward with the growing foliage while others move downward to the leaf bases. The same is true for flower stems. After this distribution of nematodes within leaves and stems, the nematodes begin to breed and cause small, localized swellings which are often chlorotic. These swellings are called spickels (2,5) or spikkels (6,7). Spickels or spikkels are shown in Figure 2 and Figure 3. Spickels can usually be felt by running the leaves between the fingers (5,6,7). When leaves and flower stems are heavily-infected with *Ditylenchus dipsaci,* the spickels seem to run together causing the leaves to become twisted, distorted, and discolored (2,5,6,7). Leaves showing these symptoms are shown in Figure 4. Flower stems are often affected in the same way as the leaves. In addition, the flower stems are often shortened (Figure 5), and flowering may be delayed, or in extreme cases completely prevented (2). As the foliage of a nematode-infected plant dies down, the nematodes move into the scale leaves of the bulb through the bulb's soft neck (5,6,7). Once the nematode enters the bulb, it begins to reproduce there. The fate of the nematode-infected bulb is always the same if it is left untreated; it rots. The nematodes spread from scale to scale by moving down through one scale and destroying it; then they enter the basal plate and subsequently spread upward in the scales which are adjacent to the destroyed scale (5). Thus, the nematodes move from scale to scale via the basal plate until the bulb is entirely destroyed. If a cross section is cut





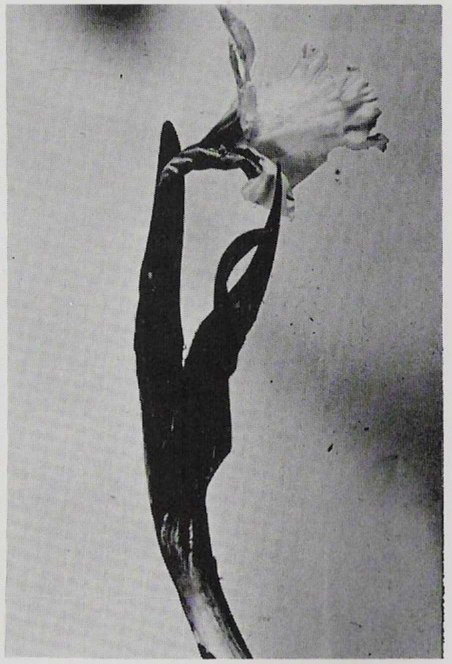
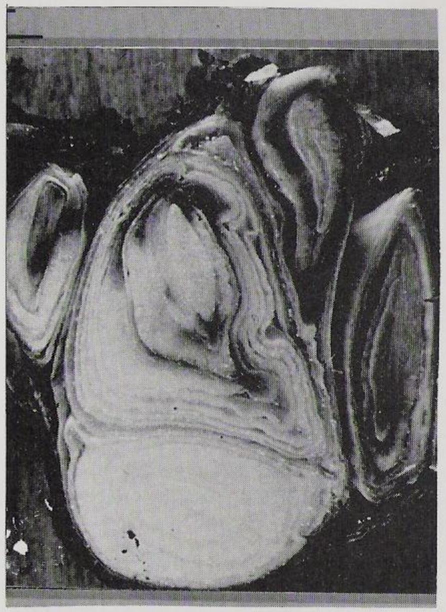


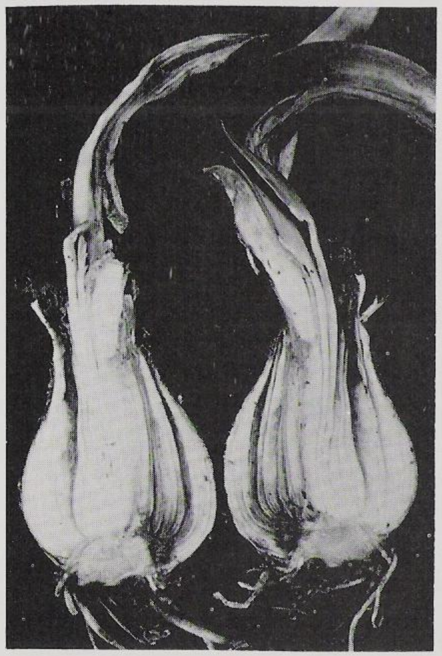
Figure 2, top left: Spickels on Narcissus Leaf
Figure 3, top right: Spickels on Narcissus Leaf (Enlarged)
Figure 4, bottom left: Distorted Leaves Caused by Bulb and Stem Nematode
Figure 5, bottom right: Distortion of Flower Stem and Flower Caused by the Bulb and Stem Nematode

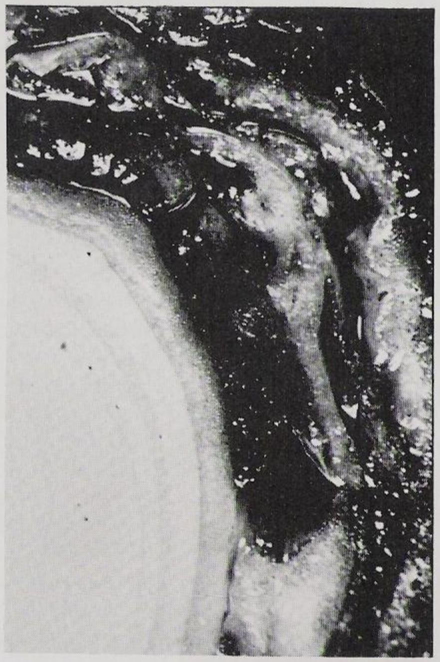
through the bulb; concentric rings of brown or necrotic scale tissue will be seen (Figure 6). A simple technique for inspection of bulbs for nematode infection has been described by Wheeler (14). This technique involves making successive cross-sectional cuts through the bulbs beginning with the nose to reveal the characteristic brown concentric rings indicative of nematode infection. Healthy bulbs do not appear to be adversely affected by this procedure. The formation of these concentric rings is a classic symptom of infection by *Ditylenchus dipsaci* (2). See Figure 7 for a longitudinal section through a nematode-infected bulb. The rotting or destruction of the bulb scales (Figure 8) is due to the production of an enzyme called pectinase which digests away the pectin-containing middle lamellae between adjacent cells of a bulb scale (4). Thus, the bulb scales become completely macerated. Another symptom of nematode infection of the bulb is separation of the basal plate from the bulb (5). Note the separation of the basal plate from the nematode-infected bulbs in Figure 9. As a bulb becomes completely infested with nematodes, pre-adult forms (fourth-stage larvae) begin to ooze onto the outer parts of the bulb, e.g. basal plate or the dry, outer bulb scales; or the nematodes may localize in air space between outer bulb scales (2,5). Here, the ooze of the fourth-stage larvae dries into masses which resemble tufts of cotton (Figure 10). These dried masses of fourth-stage larvae are often called 'nematode wool' or 'eelworm wool' (2,5,6,7). The wool stage is important for survival of the nematode. For in this state, the nematode can remain dormant for several years until moistened to become active again (2,5,6). When the nematodes in the wool become moistened and active, they can serve as the source of inoculum for new infections by *Ditylenchus dipsaci* (2,5,6,7). The nematode wool can be confused with the white mycelium of *Fusarium oxysporum* f. sp. *narcissi* which is often found along the periphery of the basal plate of a bulb with basal rot (2). Nematode-infected bulbs are often secondarily attacked by bulb mites, *Rhizoglyphus echinopus,* and by small narcissus fly larvae, *Eumerus* species (2).

ROOT LESION NEMATODE

The root lesion nematode, *Pratylenchus penetrans,* attacks roots of narcissus bulbs early in the growing season causing small, dark, slit-like lesions on the roots (Figure 11); however, it does not invade the bulb (2,6,7). The only foliage symptom of infection by the root lesion nematode is retarded growth (Figure 12) as a consequence of damage to the roots (2,6,7). Bulb size is reduced in narcissus infected by the root lesion nematode (6,7). Infection of narcissus by the root lesion nematode often shows up in plantings (Figure 13) as patches containing stunted plants (2,6,7). When bulbs from these patches are lifted late in the growing season, the roots will often be found to be brown, rotting, and usually broken off short. This root rot is due to the secondary infection of the lesions by the fungus *Cylindrocarpon radicola* (2,6,7,8). Thus, this root rot is a secondary consequence to the primary infection by the root lesion nematode. On hot, sunny days, plants infected by the root lesion nematode often wilt because of the impaired absorption of water by the damaged roots; premature death of the foliage may follow (6,7). The root lesion nematode is known to infect a number of plant species; therefore, populations of this nematode can build up in soil to a level high enough to pose a serious problem to narcissus plants. Unlike the bulb and stem







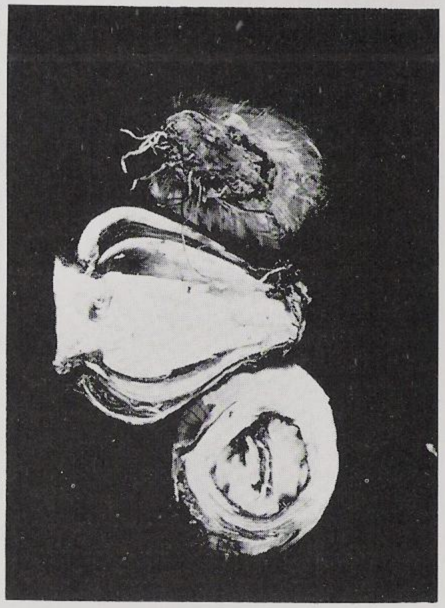
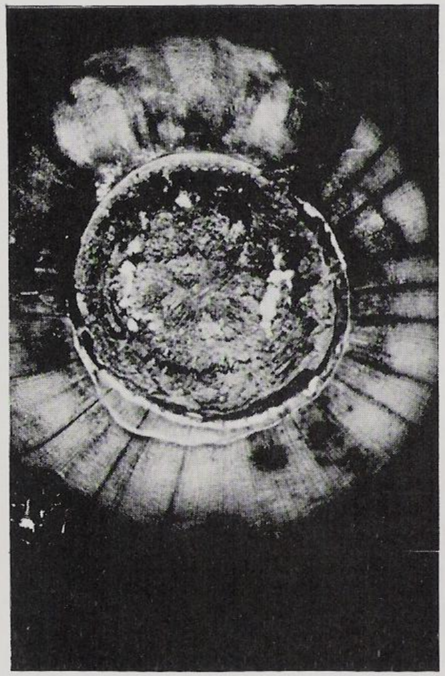


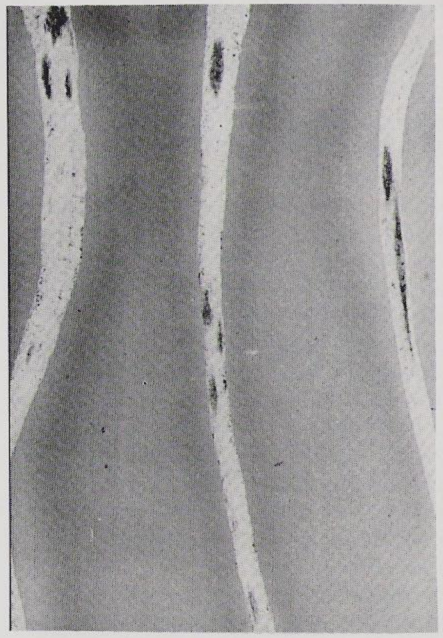
Figure 6, top left: Concentric Rings of Brown or Necrotic Bulb Scale Tissue Caused by the Bulb and Stem Nematode (x.s.)

Figure 7, bottom left: Brown or Necrotic Bulb Scale Tissue Caused by the Bulb and Stem Nematode (l.s.)

Figure 8, top right: Rotting or Enzymatic Digestion of Bulb Scale Tissue Caused by the Bulb and Stem Nematode

Figure 9, bottom right: Separation of Basal Plate from Bulb Caused by the Bulb and Stem Nematode





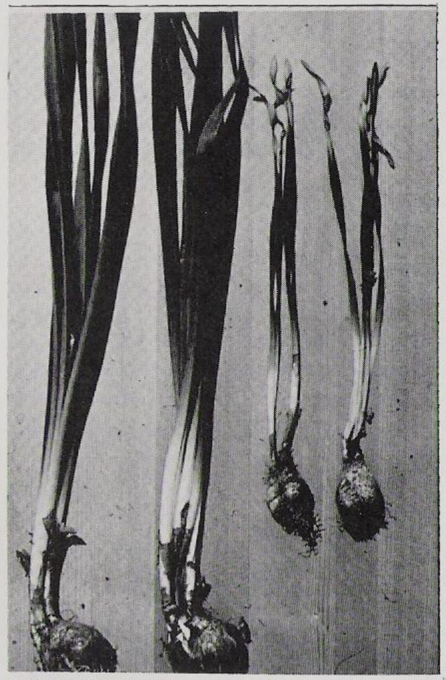


Figure 10, top left: Nematode Wool on Basal Plate of Bulb Infected by the Bulb and Stem Nematode

Figure 11, bottom left: Lesions on Roots Caused by the Root Lesion Nematode

Figure 12, top right: Root Damage and Stunted Growth Caused by the Rood Lesion: Nematode. (Left, healthy; right, infected.)

Figure 13, bottom right: Retarded Growth Caused by the Root Lesion Nematode

nematode, the root lesion nematode is most susceptible to desiccation; therefore, there is little chance of introducing this nematode into clean soil as a consequence of it being carried on the dry roots of bulbs (2,6,7). Infection of roots of bulbs occurs year after year in the spring in soils where the population of the root lesion nematode is high.

BULB AND LEAF NEMATODE

Infections of narcissus plants by the bulb and leaf nematode, *Aphelenchoides subtenuis,* are rare (2). Infected bulbs show crinkling and blistering of outer bulb scales; additionally, the cut bulb shows a diffuse, grayish discoloration rather than the concentric rings of bulbs infected by the bulb and stem nematode (2). During the growing season, the foliage becomes heavily infected, chlorotic, and may die down prematurely; however, spickels are not formed on the foliage as is the case with plants infected with the bulb and stem nematode (2).

CONTROL

Control of nematodes in narcissus, depending upon the nematode species, variously involves hot-water treatment (HWT), nematicides, crop rotation, etc. Unfortunately, the control of nematodes in small plantings by narcissophiles is difficult because of the difficulty in obtaining nematicides or equipment for HWT.

BULB AND STEM NEMATODE

It was control of the bulb and stem nematode, *Ditylenchus dipsaci,* to which J. K. Ramsbottom turned his attention in 1917, culminating in the development of HWT for control of this nematode (1). The HWT method as developed by Ramsbottom is essentially the same as that which is used today. Basically, there are two HWT programs which are used to control the bulb and stem nematode (9). Firstly, there is the HWT program of bulb stocks which are obviously infected by the bulb and stem nematode. In this program, obviously infected bulbs are removed from the planting by roguing. As soon as the foliage dies down, the bulbs are lifted and immediately given a pre-soak for three hours in cold water containing 0.5% formalin (1 liter commercial formaldehyde/200 liters water) and a non-ionic wetter. After the pre-soak, the bulbs are given HWT for 3 hours at 44.4°C (112.0°F) in 0.5% formalin containing a non-ionic wetter. This early treatment for bulb stocks which are known to be infected by the bulb and stem nematode will damage the first year flowers but not the bulbs (5). The second HWT program is a late treatment which is designed for bulb stocks which are clean, i.e. show no visible signs of infection by the bulb and stem nematode. In late HWT, the bulbs are lifted at the regular time and can be given HWT at anytime up until the root initials have started to grow (5). The order in which cultivars bloom does not dictate the order in which they should be given HWT. Instead, poeticus-type cultivars should be treated first followed by short cup, long cup, and trumpet cultivars in that order. Doubles should be treated according to their origin, e.g. double whites of poeticus origin should be treated early (5). As mentioned earlier, HWT can damage first year flowers. Thus, if first year blooms are not required, the bulbs are given HWT for 3 hours at 44.4°C (112°F) in 0.5% formalin containing a non-ionic wetter. Ideally, these bulbs should be planted immediately or stored in a cool, dry place until planting. If first year flowers are

required, the bulbs should be first warm-stored for 7 days at 30°C (86.0°F) to minimize flower damage caused by HWT. Then, pre-soak the bulbs for 3 hours or overnight in 0.5% formalin containing a wetter. Lastly, give HWT for 3 hours at 46.7°C (116°F) in 0.5% formalin plus a wetter. After HWT, the ideal situation is to plant the bulbs; however, if this is not possible, the bulbs should be given cool, dry storage. In either early or late HWT, fungicides, e.g. 0.5% Benlate, can be added to the 0.5% formalin plus wetter for the control of the basal rot fungus, *Fusarium oxysporum* f.sp. *narcissi.*

The use of 0.5% formalin in the pre-soak is important as the formalin will kill any nematodes which become activated from the nematode wool state by the soak (2,5). Additionally, the formalin will kill spores of the basal rot fungus. Thus, HWT without formalin will never be as effective as HWT with formalin.

To the hobbyist it is clear by now that HWT, though desirable, may not be practical because of the lack of the appropriate equipment; however, Marie Bozievich's husband designed and built an inexpensive, but effective, 'bulb cooker' for her (10). Thus, where there is a will and an inventive spirit, there will always be a way!

Another way to attempt to control the bulb and stem nematode is crop rotation where a field or bed known to be infested with the bulb and stem nematode is not planted again in daffodils for at least three years (5). In theory, it is possible to starve out any bulb and stem nematodes from the soil if a suitable host is not present (2). In the intervening period, non-hosts of the bulb and stem nematode can be planted, e.g. potatoes, lettuce, and nonbulbous flower crops (2).

Chemical control by use of a nematicide is also sometimes feasible to rid the soil of the bulb and stem nematode. One problem with nematicides is that they are not available to the hobbyist unless he (she) has a private pesticide applicator certification. In Tennessee, this certification is issued by the Tennessee Department of Agriculture after attending a short training session which is taught by personnel from the County Extension Agent's office. Perhaps, the same is also true of other states. Another problem with nematicides is that they are all toxic to humans and other animals and should be handled with great caution. In England, D-D (dichloropropene-dichloropropane) is recommended for injection in the soil at the rate of 400 lbs/acre; applications should be made six weeks before planting (2). Although covering with black plastic after injection with D-D is not required, its efficacy might be increased by covering the soil with black plastic so as to reduce loss of D-D by volatilization. Other brand names for D-D are Telone, Vidden-D, and Telone-II (3). Perhaps the use of Nemacur, applied as granules or as an emulsion to the row at planting or in a 10-12 inch band over the row after planting, would minimize infection of bulbs by killing bulb and stem nematodes as they move through the soil. Nemacur is the brand name of a contact nematicide (3) which is known as phenamiphos (11).

The use of thionazin (Zinophos or Nemaphos) as a chemical dip offers the attractive possibility of controlling the bulb and stem nematode provided the stock of bulbs shows only a light infection (2). However, a thionazin dip is decidedly inferior to HWT if the stock of bulbs is obviously infected with the bulb and stem nematode. Thus, the thionazin dip might best be used as a precautionary measure in seemingly healthy bulb stocks. Thionazin dip of bulbs is sometimes a desirable alternative to

HWT if first year flowers are required as thionazin does not adversely affect flowering. The standard thionazin dip procedure of bulbs is done soon after lifting and cleaning. The bulbs are dipped for 2½ hours in a cool (65°F or 18°C) dip containing 0.23% thionazin (1 pint commercial 46% concentrate per 25 gallons water). Thionazin is absorbed by healthy, but not dead, bulb tissue. Prolonged contact of the bulb and stem nematode with thionazin may kill the nematode; however, most nematodes are killed by eating bulb tissue which has absorbed thionazin. One problem with the thionazin dip is that the bulb and stem nematode has been shown to leave the thionazin-treated bulb and enter the soil, only to return to the bulb when the thionazin has disappeared from the bulb (6,12). Thus, late-season infection of the bulbs by the bulb and stem nematode may occur, resulting in plants with no external symptoms which could lead to a disastrous loss of bulbs if subsequent treatment is not given (11). For perhaps this reason, the thionazin dip to control the bulb and stem nematode is sometimes not recommended (5).

ROOT LESION NEMATODE

Unlike the bulb and stem nematode, control of the root lesion nematode, *Pratylenchus penetrans*, can not be accomplished with HWT. Instead, control typically involves soil fumigation several weeks before planting with the nematicide D-D (2). The recommended rate of application is 400 lb/acre (2). Other nematicides would probably work as well as D-D on eradicating the root lesion nematode from the soil, e.g. Nemacur. Nemacur in the granular form offers the attractive possibility of control of the root lesion nematode when added to the furrows at planting or when applied above the rows in a 10 - 12 inch band after planting (13). Nemacur (15% granular) is applied at the rate of 40 to 80 lbs/acre (12,445 linear feet of row). Furthermore, Nemacur can be applied to year-old plantings as a fall application over the rows.

In the case of the root lesion nematode, HWT for the bulbs is not necessary as this nematode does not invade the bulb tissue. Furthermore, as the root lesion nematode is extremely susceptible to drying, it dies and poses no further problem to the lifted bulbs which might have originally had the nematode on or in their roots; nor is it a problem to these same bulbs planted in a new area which is free of the root lesion nematode (2).

A possible control measure for the root lesion nematode is the overplanting of beds with the African Marigold, *Tagetes erecta*. This marigold produces secretions which suppress the population of the root lesion nematode (2). Also, some suppression of the root lesion nematode population occurs in beds where residues from a previous crop of the African Marigold has been tilled under (2). Thus, bulbs planted in these beds might be at a lower risk of becoming infected by the root lesion nematode.

BULB AND LEAF NEMATODE

Control of the rare bulb and leaf nematode, *Aphelenchoides subtenuis*, is probably best accomplished by HWT of the bulbs and by crop rotation (2).

SUMMARY

As has been the attempt with the pathogens discussed in previous articles, nematodes have been discussed in terms of what they are, the species which infect narcissus, and possibilities for their control. Hopefully, this article will have provided the information sufficient for a narcissus specialist and necessary for the hobbyist.

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Chapter 6

BULB FLIES AND MITES

This article marks a topical change from diseases of narcissus to narcissus pests. In this paper, the large narcissus fly, *Lampetia (Merodon) equestris*; the small narcissus fly, *Eumerus* species; the bulb scale mite, *Steneotarsonemus laticeps*; and the bulb mite, *Rhizoglyphus echinopus*, will be discussed with regard to their life histories, importance, and control. All four of these pests belong to the phylum Arthropoda. In the phylum Arthropoda, the bulb flies belong to the class Insecta whereas the mites belong to the class Arachnida. Perhaps you will recall the classic distinction between the arachnids and the insects: four pairs of walking legs in the arachnids and three pairs of walking legs in the insects.

BULB FLIES

The large narcissus fly, *Lampetia equestris*, and the small narcissus fly, *Eumerus tuberculatus* and *Eumerus strigatus*, have in common the following: 1) adults of both flies appear at about the same time of the year, e.g. late in the spring after flowering; and 2) the eggs are laid in practically identical fashions, e.g. on the foliage near the neck of the bulb or on the bulb itself (1,2). Conversely, there are various differences between the large narcissus fly and the small narcissus fly: 1) *Lampetia equestris* larvae infect the bulbs singly via the basal plate whereas *Eumerus* species larvae attack the bulb in numbers via the neck of the bulb, 2) *Lampetia equestris* lays eggs only once during the bulb growing season whereas *Eumerus* species may lay eggs several times during the growing season, and 3) *Lampetia equestris* is a primary pest and attacks previously healthy bulbs whereas *Eumerus* species are secondary pests and only attack diseased bulbs, e.g. bulbs infected by the basal rot fungus or by the bulb and stem nematode (1).

LARGE NARCISSUS FLY

The large narcissus fly, *Lampetia (Merodon) equestris*, is a member of the family Syrphidae which contains insects known as hover flies and drone flies (1). The original home of this fly was believed to be Southern Europe; subsequently, it was reported in England in 1865 (1). The date of entry of the large narcissus fly into the United States isn't really known; however, it undoubtedly accompanied bulbs which were imported from Europe after 1865.

The life history of the large narcissus fly involves four stages: egg, larva, pupa, and adult. The adult large narcissus fly (Figure 1) has many color variations (1,2,3). Colors seen in the furry body of the large narcissus fly often appear as bands of black, orange, yellow, gray, and buff. The adult large narcissus fly is about ½ inch (12 mm) in length and resembles a small bumble bee (1). The female large narcissus fly may lay from 40-75 eggs within her lifetime of about seventeen days (1,2,3). These eggs are laid singly at a locus, not in clusters, on foliage near the ground or on the bulb itself in the spring (Figure 2). After ten to fifteen days, the egg hatches, and the larva moves down and enters the bulb through a pin-prick size hole it creates in the basal plate (1,2). See Figure 3 showing a basal plate with a larva entry hole which

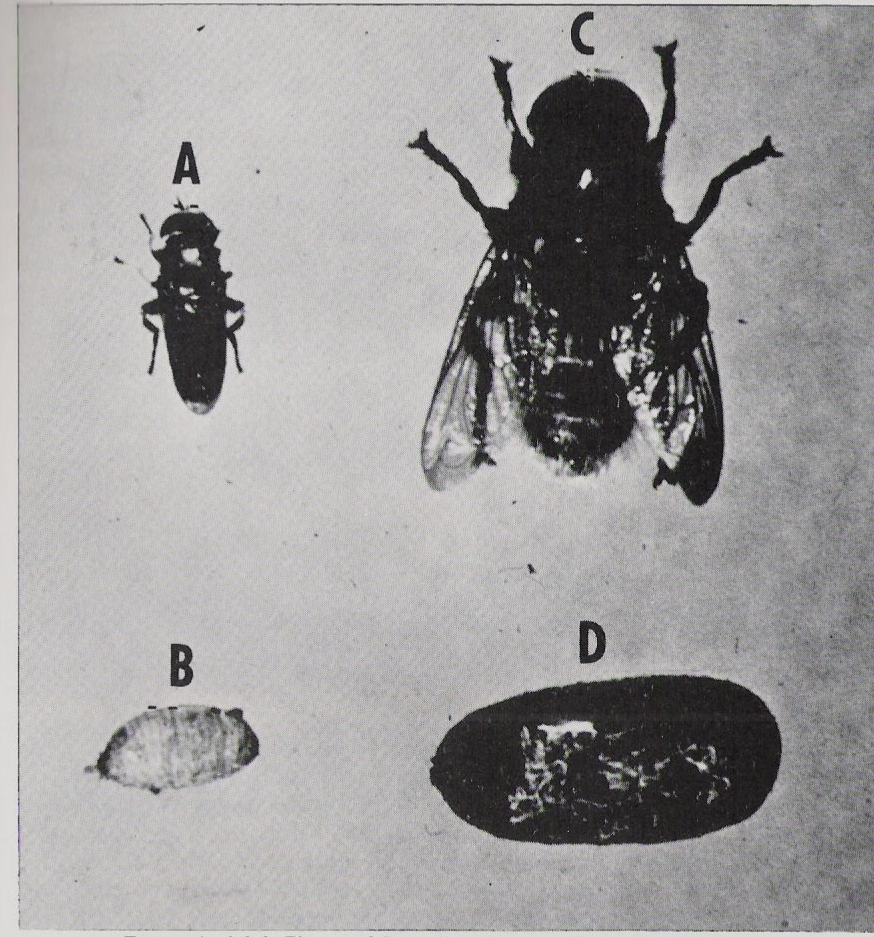
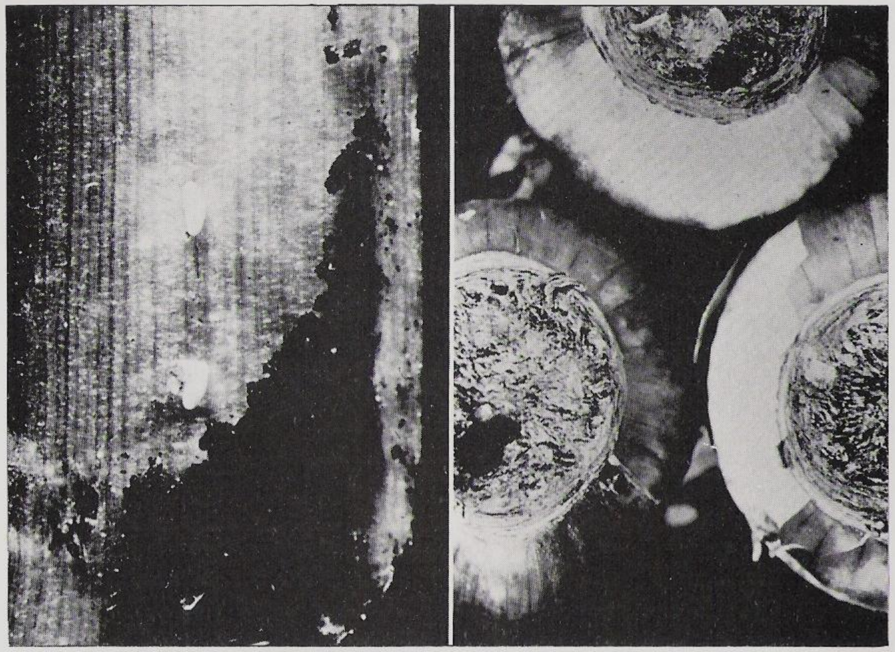


Figure 1. Adult Flies and Puparia.
A—*Eumerus* sp. adult, B—*Eumerus* sp. puparium, C—*Lampetia equestris* adult, D—*Lampetia equestris* puparium.

has been enlarged. Typically, only a single larva enters a bulb through the basal plate (1,2,3). Once the larva is within the bulb, it creates a large cavity by devouring the fleshy scales (Figure 4). A fully-developed larva or maggot is about ¾ inch (18 mm) in length. Winter passes with the larva still within the bulb (Figure 5). In early spring, the larva leaves the bulb via the neck or the hole previously created in the basal plate to move out into the soil to a position just below the surface. Here, the larva forms a puparium (pupal case) within which the larva transforms into a pupa. After five to six weeks, the adult fly emerges from the puparium to begin the cycle anew. The entire life history of the large narcissus fly encompasses a year (1,2,3).

SMALL NARCISSUS FLY

The small narcissus fly is either *Eumerus tuberculatus* or *Eumerus strigatus*. In England, *Eumerus tuberculatus* is the species most commonly found in narcissus (1). It is important to remember that the small narcissus fly is not considered a primary pest but instead is a secondary pest as it only attacks unhealthy bulbs, e.g. bulbs with basal rot or bulbs infected by

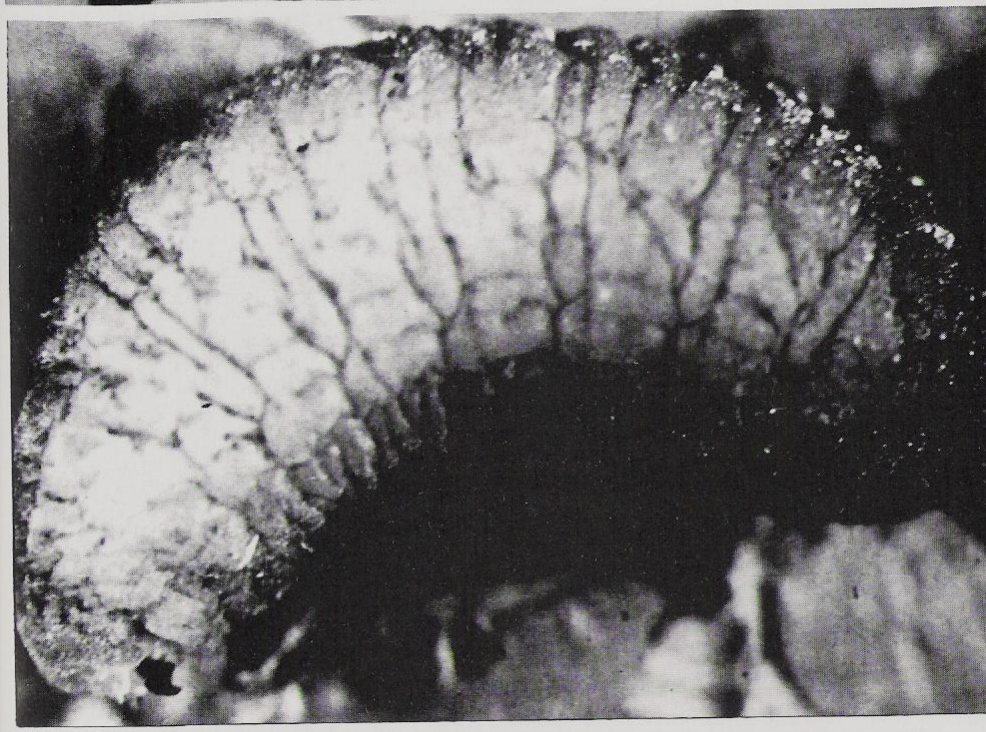


Left, Figure 2; eggs of *Lampetia equestris*. Right, Figure 3; basal plate holes caused by larvae of *Lampetia equestris*.

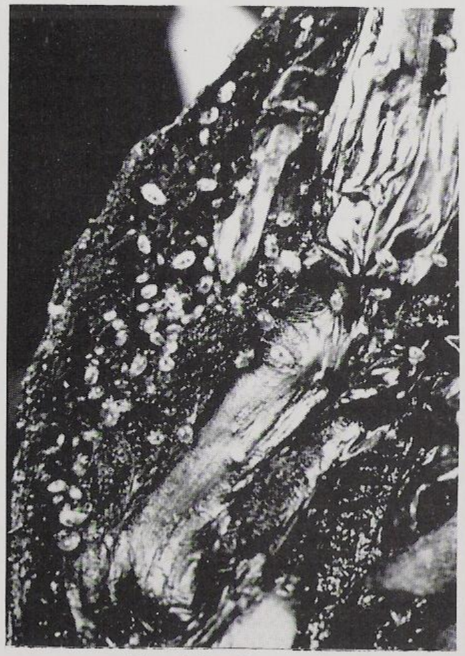
nematodes (1,2). The adult fly is about ¼ inch (6 mm) long (Figure 1). Typically, the small narcissus fly will lay its eggs in clusters of ten or more on the foliage near the ground or on the bulb itself. When the eggs hatch, the mass of larvae enter an unhealthy bulb through the neck of the bulb (Figure 6). The larvae feed on the bulb until they completely destroy it. Small narcissus fly larvae are about ⅓ inch (8 mm) in length (Figure 7). These first brood larvae then pupate. The small narcissus fly puparium is ¼ inch (6 mm) in length. About the first of July in England, these puparia release adults which lay eggs which produce a new generation of larvae to infest bulbs in the soil. Some of these larvae may spend the entire summer feeding on bulbs and will remain on or near the bulbs throughout the autumn and winter until they will pupate and release adult flies to start the cycle anew in the spring (1,2).

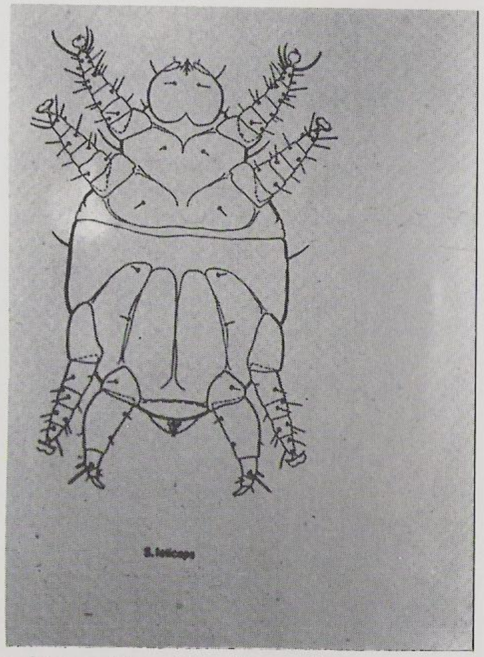
MITES

There are two species of mites which infest narcissus: 1) *Rhizoglyphus echinopus*, the bulb mite, and 2) the bulb scale mite, *Steneotarsonemus laticeps*. Bulb mites seem only to infest damaged or diseased bulb tissue (1,4); hence they would be secondary pests of narcissus bulbs. However, in the case of the bulb scale mite, it infests previously healthy bulbs. The relationship between the bulb scale mite and smoulder has been mentioned previously (5).



Top, Figure 4; larvae of *Lampetia equestris*. Bottom, Figure 5; larva of *Lampetia equestris* (enlarged).





Top left, Figure 6; *Eumerus* sp. larvae. Top right, Figure 7; *Eumerus* sp. larvae. Bottom left, Figure 8; bulb mite, *Rhizoglyphus echinopus*. Bottom right, Figure 9; diagram of the bulb scale mite, *Steneotarsonemus laticeps*.

BULB MITE

The bulb mite, *Rhizoglyphus echinopus*, as previously mentioned, seems only to infest previously injured or diseased bulbs (Figure 8). Thus, it will not be discussed further.

BULB SCALE MITE

The bulb scale mite, *Steneotarsonemus laticeps*, is a major pest of narcissus, particularly forced bulbs. It was first described in bulbs in Ireland in 1923 (1). Now, the bulb scale mite seems to have distribution throughout all parts of the world where daffodils are grown. The adult bulb scale mite is much smaller than the bulb mite. The adult bulb scale mite (Figure 9) is extremely small, 1/125 inch (0.2 mm) long (1,4), and is not visible to the unaided eye as is the bulb mite. There are three stages of the life cycle of the bulb scale mite: 1) egg, 2) larva, and 3) adult. Details of the length of the various stages isn't really known; however, in warm conditions, the bulb scale mite can complete its life cycle in two weeks (4). Conversely, under cool conditions, the completion of the life cycle would require a longer period of time. As the bulb scale mite population increases within the bulb, mites move up out of the bulb onto the foliage and spread to foliage of other bulbs to infest those bulbs (1,4).

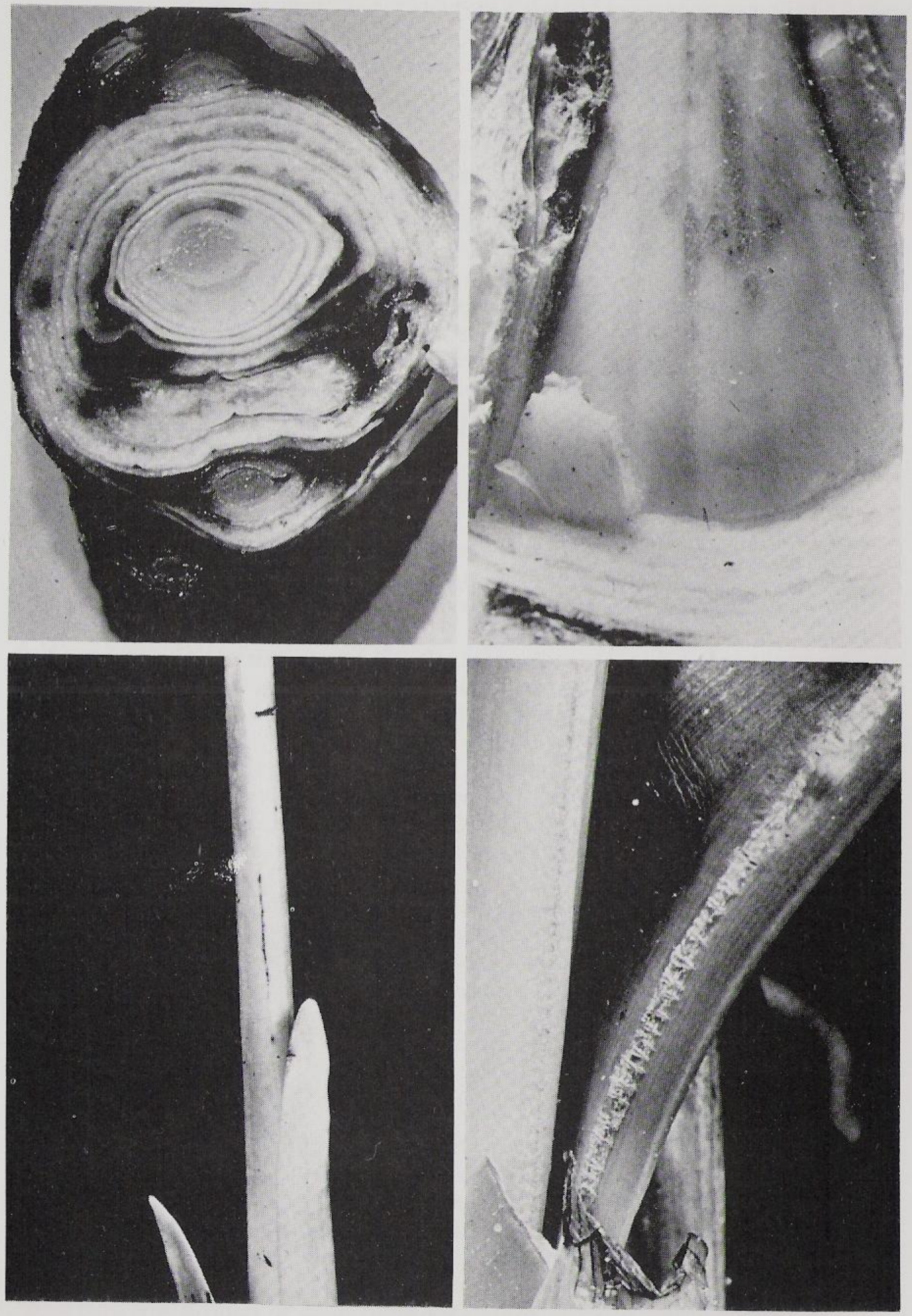
Bulb scale mites are usually found in the neck of the bulb where they feed in the angular spaces between scales (Figure 10). If bulb scales are pulled down from the neck, elongated brown scars on the scale tissue will be seen (Figure 11).

For most of the year, the bulb scale mites live in the air spaces between the scales which are caused by the shrinkage of the bulb scale tissue by the outward flow of nutrients and water. However, in the spring, the scale tissue is fully turgid (filled with water). Consequently, the air spaces between the scales are mostly obliterated as are the mites within them (1,4). Thus, to minimize the effects of bulb scale mites on forced flowers, well-shaped, round bulbs should be selected as the swelling is greatest in such bulbs (1). Thus, here is biological control, i.e. no chemicals, hot water treatment, etc., at its best.

As mentioned earlier, the bulb scale mite is not usually a problem in field grown flowers; however, it is often a problem with forced flowers because the warmer temperatures allow for a dramatic build-up of bulb scale mite populations. Damage to flower stems often shows up as a "saw edge" or as vertical scars (Figure 12). Sometimes the flower bud is killed by the mites. The leaves of mite-infested bulbs are often sickle-shaped (Figure 13) and scar tissue may be seen on the inner edge (1,4).

CONTROL

Because of the previously unhealthy nature of the bulbs attacked by the bulb mite, *Rhizoglyphus echinopus*, and by the small narcissus fly, *Eumerus tuberculatus* or *Eumerus strigatus*, control measures with these pests are of no real importance. However, with the bulb scale mite, *Steneotarsonemus laticeps*, and the large narcissus fly, *Lampetia equestris*, control may be accomplished by the use of hot water treatment (HWT) for three hours at 112°F (44.4°C) which is the same treatment used to control the bulb and stem nematode, *Ditylenchus dipsaci* (1,4,6). This temperature will kill all stages of both the bulb scale mite and the large narcissus fly. Since the removal of granular chlordane from the shelf to control the large narcissus fly, trichlorfon (Dylox R) is recommended as a soil drench to be applied to the



Top left, Figure 10; bulb scale mite damage in the angular spaces between the scales, x.s. Top right, Figure 11; scale damage by bulb scale mite, surface view. Bottom left, Figure 12; flower stem scar caused by the bulb scale mite. Bottom right, Figure 13; leaf damage and distortion caused by the bulb scale mite.

base of the foliage during the time of fly activity (3). Another trade name for trichorfon is Proxol 80 SP, a product of TUCO, Division of The Upjohn Company, Kalamazoo, Michigan. This manufacturer recommends the use of 2 ounces Proxol 80 SP/10 gallons of water as a drench per 100 feet of row. The Proxol 80 SP is applied in a direct stream to the base of the plants at the beginning of adult fly activity (early May to June). The treatment should be repeated yearly. You are cautioned that trichlorfon, like most other biocides, is toxic to many animals including man.

SUMMARY

In this article, the life histories of the large and small narcissus flies were discussed. Significantly, only the large narcissus fly is a primary pest of narcissus bulbs whereas the small narcissus fly is a secondary pest, i.e. it attacks only unhealthy bulbs. Likewise, the bulb mite is a secondary pest whereas the bulb scale mite is a primary pest of narcissus bulbs. Although several control measures exist for the control of the large narcissus fly and the bulb scale mite, HWT is recommended because it is safest to the grower and because it controls the bulb and stem nematode. As the HWT method of control of the large narcissus fly is one of a curative nature, the use of trichlorfon as a soil drench is used as a prophylactic or preventive measure.

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Chapter 7

NONINFECTIOUS DISEASES

It is of the first importance to understand that disease is a condition of abnormal physiology, and that the boundary lines between health and ill health are vague and difficult to define.

Marshall Ward (1901)

Recall that disease is defined as any departure from a state of health and that noninfectious disease is that which is not caused by a microbe(1). It is now important to note that to one degree or another a departure from a state of health also involves a concomitant physiological change, e.g. the browning of a freshly cut apple is due to the oxidation of naturally occurring phenolic compounds to quinones by enzymes which are called polyphenol oxidases. Thus, noninfectious as well as infectious disease is not only a departure from a state of health but a departure which involves physiological change as well. Noninfectious disease of narcissus and other plants is caused by nonbiological agents, e.g. hot water treatment (HWT) damage, freeze damage, herbicide damage, etc.

HWT DAMAGE

In an attempt to control the bulb and stem nematode, *Ditylenchus dipsaci*, by HWT, damage to narcissus may occur which involves the flowers, leaves, bulbs, and roots (2,3,4). Basically, HWT damage occurs as a consequence of being performed at too high a temperature or at the wrong time, i.e. before the Pc stage of internal development has been reached or a long time after it has been reached (2,4). The Pc stage of internal development of the flower in the bulb is said to have occurred when the final floral part, the trumpet or paracorolla, is clearly visible as a peripheral frill outside the base of the anthers (2,4). This stage of development can only be discerned by dissecting the flower bulb to expose the developing flower. Obviously, this is practical only when large quantities of bulbs of a single cultivar are grown. Thus, the hobbyist's concern is not the timing of HWT but rather is the recognition of HWT damage so that otherwise healthy bulbs would not needlessly be rogued. As you will recall (5), there are two basic regimens for HWT: 1) first year flowers not required—HWT for three hours at 44.4°C (112.0F°), and 2) first year flowers required—warm store the bulbs for seven days at 30°C (86°F), pre-soak for three hours or overnight, and HWT for three hours at 46.7°C (116.0°F).

FLOWER DAMAGE

If HWT is given very early, i.e. before the Pc stage of internal development, the flower bud may be killed in the bulb; however, if HWT is applied to the bulbs just before the Pc stage of internal development is reached, the damage usually takes the form of a split trumpet and ragged perianth (2). See Figure 1 showing HWT damage to a narcissus flower.

FOLIAGE DAMAGE

When HWT is carried out later than it should be, i.e. after the Pc stage of internal development has been reached, the foliage will show a pale-green, yellowish or grayish mottling or blotching near the leaf tip (2,3). See Figure 2

Top left: Figure 1, Trumpet Break, HWT Damage; right: Figure 2, Foliage, HWT Damage (both British Crown Copyright). Bottom left: Figure 3, Freeze (frost) Damage to Foliage of *N. italicus;* right: Figure 4, Freeze (frost) Damage to Flower Bud (both Snazelle photos).

showing HWT damage to narcissus foliage. It is important for the hobbyist not to react prematurely and rogue these bulbs because he/she thinks that they are virus-infected. If the symptoms on the leaves are due to HWT, they will not be seen again on the foliage of second-year-down bulbs; however, symptoms due to virus infection would be present on both first and second-year-down bulbs. Occasionally, foliage of a bulb may show symptoms of both HWT and virus infection (3).

BULB DAMAGE

Damage to bulb scales sometimes occurs when the temperature was too high or when the period of treatment was too long. Damage to bulb scales due to HWT shows up as irregular greyish areas deep within the bulb and, often times, extending up from the basal plate. Bruising of bulbs also shows up as irregular greyish areas on the bulb scales; however, damage due to bruising usually is expressed in the outer scales rather than in the scales deep within the bulb (3).

ROOT DAMAGE

If HWT is applied to bulbs very late, i.e. after the root initials have emerged from the basal plate, or at too high a temperature, root damage may occur which may express itself by the bulb making poor growth the next season (3).

SUMMARY

The safest time to minimize HWT damage is to give HWT to the bulbs shortly after they have reached the Pc stage of internal development (2).

FREEZE (FROST) DAMAGE

The available literature to the author on freeze (frost) damage to narcissus was quite limited. Freeze (frost) damage in narcissus is expressed in three ways: 1) damage to foliage, 2) damage to flower, and 3) damage to bulb. Freeze (frost) damage to foliage usually takes the form of death (necrosis) of the leaf tips (Figure 3). Such damage is particularly common in cultivars and species which put forth foliage early in the year. Usually, freeze (frost) damage causes no permanent damage other than creating unsightly foliage. In the case of freeze (frost) damage to the flower, it is most pronounced while the flower is still enclosed in the spathe or sheath, resulting in a killed flower bud which turns brown, fails to open, and may even separate from the stem (Figure 4). Lastly, in particularly tender cultivars and species, e.g. some tazettas, bulbs will actually freeze in the ground and will not put forth foliage again. Fortunately, most daffodil cultivars and species forms have good cold hardiness and survive the rigors of winter nicely.

FLOWERING—LIGHT/COLD EFFECTS

Perhaps the failure to bloom after the first year in a location which receives inadequate sunlight, e.g. the north side of a house, might not be considered by all to be a disease; however, the disease definition by Ward at the beginning of the article clearly points out that disease is ultimately expressed in terms of abnormal physiology. In the case of narcissus, the available literature to the author is sketchy as to the specific light and cold requirements for flowering. Nonetheless, the physiology of flowering in narcissus may be the same as in other plants and involve the production of light/cold induced flowering hormone(s). Thus, one might conjecture that the failure to flower in narcissus might be due to the failure of the hormone(s) to be produced. One thing which is clear is that next year's flower is formed in the bulb immediately after flowering (light dependent effect) and, in most instances, the bulb must go through a period of cold before flowering will occur the following spring (cold dependent effect). Therefore, the question concerning the physiology of narcissus flowering is as follows: What hormone(s) govern(s) flowering in narcissus? Thus, the failure to produce hormones involved in flowering would be a case of abnormal physiology in narcissus; hence, disease.

A simple experiment at Wisley Gardens has shown that the foliage must be left on bulbs for at least six weeks after blooming in order to insure bloom again the next year (6). Of course, the reason for this is that light impinging on the leaves during this period induces formation of next year's flower in the bulb.

HERBICIDE DAMAGE

Damage to narcissus foliage by herbicide drift often shows up as leaves with pale steaks, rusty mottling at the base of leaves, or leaves which are uniformly bright yellow (3). Herbicide drift occasionally occurs when the hobbyist or his neighbor is spraying a herbicide, e.g. 2,4-D, to kill broadleaf weeds, e.g. the dandelion, *Taraxacum officinale.* A specific example of herbicide damage to narcissus has been shown to occur when the pre-emergence herbicide (kills weeds before they emerge from the soil) clorpropham was used on a narcissus planting. Chlorpropham causes chevron-like markings on narcissus leaves (Figure 5). In an effort to control thistle in the Columbus, Ohio, Whetstone Park narcissus planting, Amitrol was used only on the thistle in August 1975. The result of this was the production of some narcissus foliage and flowers the following spring (1976) which were devoid of chlorophyll. Subsequently, some improvement was noticed in the spring of 1977; however, the bulbs of cultivars from areas treated with Amitrol were much smaller than those of the same cultivars from untreated areas. In subsequent years, glyphosate (Roundup) was used for weed control after the foliage had completely died down (7). The reader should be advised that a herbicide like Roundup used on actively growing narcissus plants will not only kill the foliage but will also kill the bulbs as well. Thus, precaution must be taken with use of any herbicide or devastating results may occur. One desirable aspect of the use of Roundup for weed control is that it is extremely short-lived, i.e. it completely disappears from the soil and plant residues after a few days (8).

MINERAL DEFICIENCIES

The literature available to the author on mineral nutrition in narcissus was virtually nonexistent. Thus, the effects of phosphorus and potassium deficiencies cannot be stated by the author with certainty. Nonetheless, to insure adequate amounts of these elements in the soil, a fertilizer like 0-24-24 can be used at the rate of 1-1½ lb/100 square feet for new beds. For

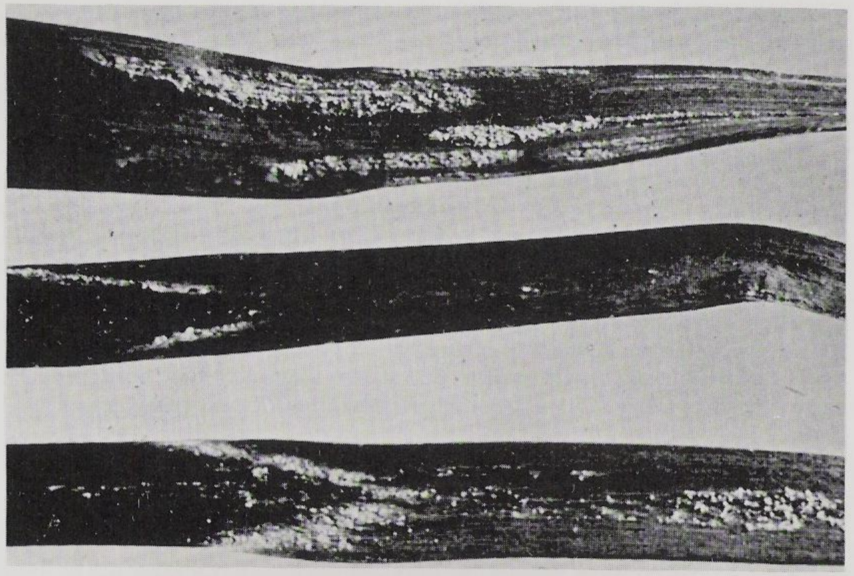

Figure 5, Herbicide (Chlorpropham) Damage to Foliage (British Crown Copyright).

established plantings, a top dressing of ½ lb 0-24-24/100 square feet can be applied in the fall, in the spring at emergence of the leaves, and again after flowering. At Rosewarne Experimental Horticulture Station, Camborne, Cornwall, England, where fertilizer applications have been steadily reduced in recent years because of a high reserve of phosphorus and potassium in the soil, pre-planting fertilizer application has been at the rate of 75 kg P_2O_5 (phosphorus)/hectare and 200 kg K_2O (potassium)/hectare. No additional top dressing was applied during the remainder of the two year cycle (9). In units more familiar to the hobbyist, the Rosewarne fertilizer applications at planting are as follows: 2½ oz phosphorus (P_2O_5)/100 square feet and 6½ oz potassium (K_2O)/100 square feet. The point of all of this is that the hobbyist does not have to religiously apply the same amount of fertilizer to his/her beds every year as it may well be that a good phosphorus and potassium reserve has been established. This can be confirmed by taking a soil sample to your local County Extension Agent for analysis. As is commonly practiced little to no nitrogen is applied to the beds as this may contribute to enhanced basal rot. In summary, it seems rather unlikely that the hobbyist will ever be confronted with the problem of phosphorus and potassium deficiency if minimal application of low nitrogen fertilizer is applied periodically to the planting.

MISCELLANEOUS DISORDERS

Bulbs lying in waterlogged soils produce stunted chlorotic foliage and invariably rot if the condition is prolonged. Obviously, prevention of waterlogging is dependent upon planting in beds which are naturally well-drained or in raised beds. Automobile exhaust emissions which are directed

on a clump of daffodils will severely damage the foliage. Although it would probably occur only in a large commercial daffodil planting, lightning striking the soil would leave a circular area of scorched plants with the effect being greatest at the point of impact and least at the periphery. Repeated urination by dogs on daffodil foliage may cause discolored foliage. Calcium chloride or sodium chloride used to melt ice on the streets may be splashed up onto daffodil beds in sufficient concentrations to severely damage or kill bulbs in the soil. High salt concentrations in the soil can be counteracted to a degree by digging gypsum into the soil. In summary, the list of inanimate objects which will cause a departure from a state of health in narcissus is endless.

AUTHOR'S COMMENTS

This article on noninfectious diseases of narcissus is the last of seven articles on diseases and pests of narcissus. For any errors which may have appeared in the articles, the author accepts full responsibility. The reader should be advised that this series of articles on daffodil diseases and pests doesn't represent the last word on the subject but is only an introduction to it.

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