

NARCISSUS SMOULDER

A review of the disease and its association with bulb scale mite infestation

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ABSTRACT. The cyclic development of smoulder caused by infection of narcissus with *Botrytis narcissicola*, is described. It is shown that, although the fungus is widespread, infection affects only a small proportion of plants. The development of lesions is linked to environmental conditions, especially cold and wet, near the time of shoot emergence. Physical damage such as flower harvesting can facilitate infection but does not satisfactorily explain the distribution of the disease. Bulb scale mite (*Steneotarsonemus laticeps*) infestation results in damage with a distribution which coincides very closely with smoulder infection, and a link is drawn between the two organisms. Methods of control of smoulder are reviewed and assessed.

INTRODUCTION

Narcissus smoulder attracted attention when the importation of bulbs from The Netherlands recommenced after the war of 1939-45. Some affected bulbs failed to emerge and in 1949 leaves with brown lesions covered with *Botrytis* conidia appeared in the first commercial daffodil crops to be grown in Kincardineshire. Thereafter smoulder was recorded each year on many of the cultivars grown in fields and in forced lots, but not in those grown in bulb fibre or flowerbeds, or among daffodils naturalized in grass.

Smoulder is caused by the fungus *Botrytis narcissicola* Kleb. (anamorph of *Botryotinia narcissicola* (Gregory) Buchwald) which differs from *B. cinerea* Pers. (anamorph of *Botryotinia fuckeliana* (de Bary) Whetzel) in its greater ability to grow in narcissus tissue and in certain cultural characteristics. This has recently been confirmed by O'Neill & Mansfield (1982a).

B. narcissicola is common on narcissus bulbs as a saprophyte or weak parasite. Infection is widespread on foliage towards the end of the growing season and frequently spreads downwards from the neck (where senescence of leaves terminates in healthy bulbs), into the white storage scales, producing irregular, brown rotting areas which may extend to the base. Infected flower stems also rot to the base. The base plate, however, does not rot and infection does not spread upwards from the base into other scales or, laterally, through the bulb. Sclerotia form in late summer occasionally on senescing leaves but mainly in the acute angles where daughter bulbs adjoin in multiple-nosed bulbs, and persist as dry, flattened structures on the brown, dry outer scales. Bulbs can remain infected for several years without producing foliar symptoms.

Conidia are produced on the sclerotia in autumn and early spring and

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are moved around by the bulb mites, *Rhizoglyphus echinopus* (Fum. & Rob.), which are active at the same period, but there is no evidence from experiments that this association of mites and conidia plays a part in spreading infection (Gray, Shaw & Shiel, 1975). During the colder part of the winter, when conidia are not formed, *Trichoderma viride* Pers.: Fr. is common on sclerotia. It has been observed in association with collapsed sclerotia in wet soil (Gray & Shiel, 1975).

Infection of floral shoots becomes evident from December onwards as brown lines on the otherwise white sheath leaves as these emerge from the neck of the bulb. At this stage mycelium grows rapidly from rotting parts of the white storage scales over the surface of the sheath on to the emerging floral leaves. In cold, wet soil the entire bulb nose may rot and the shoot may fail to emerge.

If the infected shoot extends above ground, smoulder takes the form of a yellowish-brown area, bearing conidia, along the inner curve of leaves which are stiff and curled 'like a black-cock feather' (the growers' description), or on the rigid distorted tips of leaves. The tips tend to adhere and to retain the flower bud which also becomes infected. In either case it is usual for more than one leaf from the nose to be infected and for the lesion to form at the same level on each leaf from the one nose. The affected leaves are at first brighter green in colour than normal and may show yellow flecks suggestive of virus infection.

Smoulder lesions above ground level are confined to the rigid malformed tissue and soften and extend only as the leaves wither towards the end of the normal period of growth. In the later stages of growth of the crop, because of natural senescence and as a result of the damage caused by picking flowers, by other operations and by weather, foliage becomes generally soft, discoloured and rotting and therefore, smoulder lesions are not easily distinguished. The very late flowering double white narcissi show little smoulder, while in other late flowering cultivars, such as cv Verger, infection of leaves within the bulb neck occurs late in the season in prematurely senescent foliage with no symptoms of smoulder above ground.

FACTORS AFFECTING OUTBREAKS OF DISEASE

Although infection with *B. narcissicola* is widespread, only a small percentage of plants exhibit foliar symptoms. The incidence in a single stock varies somewhat under differing soil conditions (Gray, 1971). The absence of clusters of shoots with symptoms supports observations that the disease does not spread from bulb to bulb or even from nose to nose of individual bulbs. Records made in crops confirm that symptoms not only tend to occur at the same level on infected leaves from a single nose but, in addition, a high proportion of the lesions in a field occurs at one level or at only a few. Depth of planting may determine the date at which the shoots, and therefore the lesions, appear above ground but the location of the symptom on the shoot does not vary.

Such uniformity indicates that some environmental factor triggers the development of smoulder on shoots. The adhesion of infected leaves

carried up into the canopy suggests also that the lesion must have been initiated within the bulb where the developing leaves are in close contact. In bulb noses rotted by smoulder, the original centre of infection is to be found within the neck, while any foliage which has extended beyond the neck before the lesion forms remains unaffected until the crop senesces. The neck is the region where the sheath leaves of the emerging shoot come into contact with infected tissue in the storage scales.

If bulbs with sheath leaves which have become infected are brought into a dry atmosphere, the smoulder lesion does not develop on the floral shoot even though the mycelium of *B. narcissicola* can be seen extending over the surface. This might be regarded as a form of latent infection. In conditions of high atmospheric humidity, but with no free moisture on the epidermis, the sheath leaves gape and the inner leaves emerge quickly, showing no infection. The water-soaked state favourable for infection has been recorded, in periodic samplings, in bulbs with mycelium spreading to the sheath from areas of storage scales bearing sclerotia. A possible localized source of wetness occurs when emerging leaf tips, enclosed within the noses of bulbs with long residues of withered leaves, show guttation, taking the form of many small drops of liquid on the surface of the centimetre length of leaf tip protruding from the sheath leaves. A more important source of wetness is heavy rainfall, especially when the soil is frozen. Trigger periods prior to outbreaks of smoulder on shoots have been found to coincide with periods of moderate or heavy rain, with the soil described as 'wet', i.e. with standing water in small or large pools on the surface according to the Meteorological Office definition. Similar waterlogging conditions led to serious outbreaks of smoulder where bulbs boxed for forcing were placed on a concrete surface without adequate drainage. It now seems probable that attempts to induce the shoot symptom of smoulder experimentally in conditions of high humidity, or on a high water table, failed because the soil around the neck of the bulb was not sufficiently wet.

A critical period of wetness may account for the uniform level at which the lesion occurs throughout a population; the extent of the lesion is most likely to be determined by soil temperature. The type of infection which destroys the shoot takes place early in the growing season, almost before emergence, when low soil temperature does not favour shoot elongation. At higher soil temperatures, the infected part of the shoot is carried up into the canopy where it does not progress. In general, tip lesions are most frequent in later flowering cultivars which otherwise exhibit relatively small amounts of smoulder.

But although relative position of lesions on shoots may be related to physical conditions, there must be some other factor which accounts for fungal proliferation, since the incidence of shoots with smoulder is low, whereas infection of bulb stocks is widespread. All those who have worked with the disease have experienced difficulty in inducing smoulder artificially (O'Neill & Mansfield, 1982a). The crude type of injury required for successful experimental inoculation does not occur in the bulb neck in natural conditions but both roguing (Gray, 1970) and flower picking (Dixon, 1985) cause damage which can lead to smoulder. The organism best placed to introduce infection into the shoot is the bulb scale mite,

Steneotarsonemus laticeps (Halb.). It is commonly found in association with smoulder in forced bulbs. We therefore examined the possibility of a link in field crops.

ASSOCIATION WITH BULB SCALE MITE INFESTATION

The curling of leaves with smoulder resembles the distortion caused by bulb scale mites in forced narcissi. In samples of bulbs from field crops with appreciable smoulder, mites were located only after dissection of individual bulbs (Gray, Shaw & Shiel, 1985). From 1972–1977, mites and their eggs were recorded in numerous samples of bulbs dug from commercial crops throughout northern Scotland. Mites were already present on the flower initials of cv Golden Harvest by September. The characteristic yellow feeding areas were found at the base of leaves and on the white storage scales. The mites were observed moving detached clumps of smoulder mycelium from the brown tissue in the bulb neck to the adjacent white storage scales. In all cases observed, the irregular areas of storage scale affected by smoulder, extending downwards from the neck, originated on the yellow lines where mites had fed and not on the white tissue. The incidence and activity of the mites in the crops correspond with the account by Hodson (1934). The loss of 'bloom' on the surface of the leaves and the abnormally bright green colour, which he noted, proved useful characters in locating plants in the field with evidence of feeding by mites at the base of the leaves.

Attempts to assess the role of mites in inducing smoulder were made by placing bulb shoot initials, cut from above the root plate, on water agar to which had been added blocks of mycelium of *B. narcissicola* from cultures and portions of white scales with mites. The mites did not move from the scales; *B. narcissicola* infected tissue damaged during excision at the base of some initials, and there was limited infection of the delicate tissue of the tip of the outer sheath leaf, but the leaf initials became green and were not infected. Finally, *Ophiostoma narcissi* Limber grew from the scales over the bulb initials.

A stock of bulbs derived from excised initials, and maintained for three years in sterile conditions, was used to examine the effect of adding scales with mites and sclerotia to pots of peat/sand compost. Of the four bulbs so contaminated, two gave rise the following spring to a single curved leaf with marks of feeding by mites at the base and with *B. narcissicola* growing on the curve. The eight uncontaminated bulbs remained free from smoulder. The fungus on the lesions corresponded with *B. narcissicola* in culture and in tests of pathogenicity.

This is the only experimental evidence for inducing smoulder by a combination of mites and fungus, although in the laboratory *B. narcissicola* grew on white storage scales which had been colonized by mites from hay. These findings agree with the results of O'Neill & Mansfield (1982b) who obtained significantly more foliar lesions from bulbs contaminated with debris bearing sclerotia (and presumably mites, although this is not stated), but only infection of senescent tissue where

washed sclerotia were placed on or above the bulb nose. It is interesting that in his first experiment with sclerotia, Klebahn (1907) mentioned a little growth of *Botrytis* on some yellow flecks on the leaves which could correspond to areas of mite damage. It may therefore be that the injuries caused by feeding by bulb scale mites are sufficient to allow infection. Mite infestation also leads to a reduction in the 'bloom' described earlier, and this 'bloom' may influence the reaction of the leaf to infection by *B. narcissicola*. The composition of the 'bloom' was examined with the help of Dr D. Jones on material prepared for the scanning electron microscope at the Macaulay Institute for Soil Research. There were indications that the covering of wax, which is present on the leaves from an early stage, was less complete or less uniform on samples lacking 'bloom', but differences were not always clear.

In inoculation experiments, *B. narcissicola* infected the undamaged surface of segments of leaves of cv Mount Hood with reduced 'bloom' and evidence of feeding by mites. Segments of leaves with normal 'bloom' were infected only where the epidermis was damaged by scraping. Blocks of agar with mycelium from three isolates of *B. narcissicola*, supplied by Dr J. Mansfield, were placed on series of terminal lengths of normal leaves and leaves with less 'bloom' of cv Golden Harvest. Areas of leaf surface senescent, rotting and covered by mycelium, measured five days after inoculation, varied between the isolates but were consistently greater on material lacking 'bloom'. On the uninoculated leaves smoulder did not occur but *B. polyblastis* Dowson (*Botryotinia polyblastis* (Gregory) Buchwald) grew on one normal leaf and one without 'bloom'. This fungus is rarely seen and its presence in the crop was not suspected.

In commercial crops in the first year after hot water treatment of the bulbs, it is usual to find foliage distorted by bulb scale mites, with yellow feeding areas at the base, although few shoots show smoulder. Presumably the treatment, the breaking up of large bulbs, drying out, and the removal of debris, reduce the amount of inoculum available for infection of storage scales and emerging shoots at the time when the mites become active. In the second and subsequent years, a proportion of shoots damaged by the mites are also affected by smoulder. We were not able to find plants with primary smoulder symptoms which did not also show signs of feeding by bulb scale mites. The malformations and curled leaves characteristic of primary smoulder are too extreme to be attributable only to infection by a *Botrytis*. Late flowering narcissi, which have a high incidence of shoots damaged by bulb scale mites, show little smoulder as the warmer, drier soil conditions at their time of emergence favour the pest rather than the fungus.

Bulb scale mite causes damage which leads to infection with *B. narcissicola*, and under certain environmental conditions this proliferates and smoulder results. The site of mite infestation, its distribution in the crop, the low level of infestation, and the clear association between mite and smoulder symptoms support the view that there is a link between smoulder and mite infestation. Other sources of tissue damage—such as flower picking—can also lead to smoulder development but do not occur with a distribution that coincides with field symptoms.

CONTROL OF SMOULDER

As *B. narcissicola* is so widespread as a saprophyte wherever bulbs are grown or handled, there is little prospect of excluding infection by hygiene. Even the virus-tested stocks produced by 'twin scaling' show evidence of both mite and smoulder activity. Roguing of commercial crops brings no improvement in health of bulbs, and the damage and disturbance involved may increase expression of foliar symptoms (Gray, 1971).

The apparent improvement in bulb yield and flower quality with benomyl applied to bulbs of cv Verger (Gray & Shiel, 1975) was probably due to an acaricidal effect, since the smoulder developed in the neck of the bulb after the leaves had withered prematurely, and that is more likely to have been an effect of mites feeding in the neck, a condition not appreciated at that time.

It is unlikely that any chemical treatment could prevent infection of the emerging shoot within the neck. We obtained no control from combinations of acaricides and fungicides applied to bulbs, as soil drenches and to foliage, from pre-planting until after senescence. Commercial spraying would be impractical because of the difficulty of predicting environmental 'trigger' periods, and very damaging because of the soil conditions of extreme wetness associated with infection.

Commercial stocks, which have been treated with hot water, and usually also with a fungicide, are relatively free from primary smoulder in the first year of growth, although malformations in foliage caused both by hot water damage and mite infestation occur. In subsequent years smoulder reappears due to the proliferation of senescent tissue round the bulb neck and because mite activity becomes concentrated in the neck region. In consequence, high levels of smoulder are common in the second and third years after planting. In bulbs 'naturalized' in grass, the soil structure about the neck of the bulb is maintained by the grass and there may be quite severe drying out of the soil during summer. Competition from the grass keeps the bulbs smaller, and as a result of their slow growth they retain a 'round' shape. These effects serve to reduce both infestation by mites and development of smoulder.

Since adverse growing conditions are so important for the formation of shoot lesions, the provision of a good growing environment would seem to offer the best prospect for control. A freely drained rooting medium, with a good soil structure around the bulb neck would be beneficial on several counts. In bulbs for forcing, measures could be taken to avoid low soil temperatures and this would ensure rapid shoot development. Plants grown in an improved environment would still be liable to infestation by mites and infection by *B. narcissicola* but death of the shoots would be less likely.

REFERENCES

- DIXON, G. R. (1985). The relationships of narcissus smoulder (*Sclerotinia narcissicola* Greg.) to flower harvesting practice. *Research and Development in Agriculture* 2:37-39.

- GRAY, E. G. (1971). Observations on *Sclerotinia* (*Botrytis*) *narcissicola*, the cause of smoulder in northern Scotland. *Acta Horticulturae* 23:219–222.
- & SHIEL, R. S. (1975). A study of smoulder on narcissus in northern Scotland. *Ibid.* 47:125–133.
- SHAW, M. W. & SHIEL, R. S. (1975). The role of mites in the transmission of smoulder in narcissus. *Plant Pathology* 24:105–107.
- HODSON, W. E. H. (1934). The bionomics of the bulb scale mite *Tarsonemus approximatus* Banks var. *narcissi* Ewing. *Bulletin of Entomological Research* 25:177–185.
- KLEBAHN, H. (1907). Weitere Untersuchungen über die Sklerotienkrankheiten der Zwiebelpflanzen. *Jahrbuch der Hamburgischen wissenschaftlichen Anstalten* 24:1–53.
- O'NEILL, T. M. & MANSFIELD, J. W. (1982a). The cause of smoulder and the infection of narcissus by species of *Botrytis*. *Plant Pathology* 31:65–78.
- & — (1982b). Aspects of narcissus smoulder epidemiology. *Ibid.* 31:101–118.