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Canada

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NOTE

Recommendations for pesticide use in this publication are intended as guidelines only. Any application of a pesticide must be in accordance with directions printed on the product label of that pesticide as prescribed under the Pest Control Products Act. Always read the label. A registered pesticide should also be recommended by provincial authorities. Because recommendations for use may vary from province to province, your provincial agricultural representative should be consulted for specific advice.

CONTENTS

SUMMARY

Amenity turfgrasses in the Prairies of Canada, where winters may be long and severe, are often severely affected by low-temperature diseases. Climatic conditions restrict the range of grass species and/or varieties which can be successfully employed in amenity turf, compared with the remainder of Canada; the pattern of disease is also quite different.

The first part of this bulletin sets out to consider the causes of non-pathogenic winter diseases and their management. The second section deals with the causes, symptoms, epidemiology and management of the principal low-temperature-tolerant fungal pathogens (snow molds) which are involved in winter disease. Symptoms of winter diseases are illustrated. The interactions of these pathogens and antagonists in disease complexes are considered.

Some of the information given may also be useful to those who are concerned with the management of agricultural grassland in the continental interior of North America and other parts of the world with severe winters.

Chapters on the major problems or diseases are self-contained and full reference citations are included.

RESUMÉ

Dans les Prairies canadiennes, où les hivers sont souvent longs et rigoureux, les gazons d'agrément sont souvent atteints de maladies qui se développent à basse température. Contrairement aux autres régions du pays, les conditions climatiques y restreignent le nombre d'espèces et de variétés de gazon que l'on peut implanter avec succès dans les pelouses d'agrément. En outre, les maladies n'y évoluent pas du tout de la même façon.

La première partie du présent bulletin porte sur les causes des maladies non pathogéniques qui se développent pendant l'hiver et sur les moyens de les contrer. La deuxième traite des causes, des symptômes, de l'épidémiologie et des méthodes de lutte associés aux principaux champignons pathogènes psychrotrophes (moisissures nivales) responsables de maladies particulières à l'hiver. Les symptômes de ces maladies y sont illustrés. On y aborde aussi les interactions de ces agents pathogènes et de leurs antagonistes dans des complexes pathologiques.

Certains renseignements peuvent également servir à ceux qui s'intéressent à la conduite des surfaces herbagères en agriculture dans les terres continentales en Amérique du Nord et dans d'autres parties du monde où l'hiver est rigoureux.

Les chapitres où il est question des principaux problèmes ou maladies sont indépendants les uns des autres et sont assortis des citations de référence complètes.

PREFACE

Canada has a great range of climate from temperate oceanic through extreme continental to boreal, and grasslands are found in all of them. While agricultural grasslands have received considerable research attention in this country, amenity grasslands have been an underfunded, unorganized, and largely neglected area of research endeavour (Smith, 1980a).

Amenity grassland may be defined as "all grassland with recreational, functional or aesthetic value of which agricultural productivity is not the primary aim" (NERC, 1977). It can be categorized into intensively-managed turf for games (fields for football of different kinds, field hockey, cricket and polo; golf and bowling greens), trampled grasslands (lawns, parks, golf fairways) and rarely trampled or minimum care grassland (railway embankments, grass airfields, most highway verges and cemetaries).

As far as can be determined, few attempts have been made to estimate the costs of establishment and maintenance of amenity grassland for the Canadian provinces or for Canada as a whole, as has been done for some states in the United States (Nutter, 1965; Kneebone & Hillman, 1969; Rydstrom, 1973) or in the United Kingdom. For the latter, the 1973-74 estimate for amenity turf maintenance was L Stg. 140 million (NERC, 1977). For the City of Saskatoon the annual cost for maintenance of urban turf, expressed as depreciation, in 1974 was estimated as $ Can. 15.5 million (Nelson, 1975). The annual maintenance cost for all types of amenity turfgrass in the Province of Saskatchewan was estimated in 1974 at $ 15.5 million (Smith, 1974). The value of the sod being grown in the province was calculated as $ 3.2 million in 1980 (Smith, 1980a). While the approximate monetary value for establishing and maintaining amenity turf is ascertainable, it is very difficult to quantify its social value. This cannot be expressed in purely economic terms since it also improves our general environment, provides facilities for recreation, exercise and appeals to aesthetic sense. It is probably the latter considerations which account for the generally high standard of turf management found in domestic lawns, parks and campuses in the Prairie Region where other herbage is often dry and brown. There seem to be no published estimates of the proportion of our population which uses amenity turf for recreation in Canadian provinces. In Britain it was estimated that 4% of the people were members of organizations which used amenity turf for such pursuits as sports, camping and horse riding. Many more utilized it for exercise, picnics and other activites (NERC, 1977). In the Prairies the area of turf used for some sports has seen a considerable increase in recent years. In Saskatchewan, for example, golf has been played on grass greens at Regina, Saskatoon and Prince Albert since the first decade of this century. In 1973 there were 111 golf

courses, 36 of them with grass greens (Smith, 1980a). Golf courses with grass greens are now found in the National, Provincial and some of the Regional Parks in the Province. Golf is well catered for also in Alberta and Manitoba and many new courses are under construction or are planned in the Prairies. Despite current financial restrictions, it seems likely that public turf usage is to increase as the size of building lots declines and the trend towards urban, highrise apartment dwelling develops. The reduction in hours of the work week will allow more leisure and favour increased turf usage.

The utilization pattern of sports turf in Canada varies from region to region. In the Prairies, the period of play occurs from 6-7 months from late spring to fall, depending largely on latitude. Northern golf courses may not open until late May. This compares with Vancouver Island where many turf sports may be played throughout the year.

The species and cultivars of turfgrasses which can be used in a region are determined mainly by climate. While bentgrasses, fine-leaved fescues and perennial ryegrass may be used in domestic lawns on the West Coast, perennial ryegrass usually barely survives Prairie winters so lawns in the latter region are usually Kentucky bluegrass monostands or that grass in mixture with creeping red fescue. In the Prairies, the bentgrasses which are used to form golf greens require protection from desiccation, freezing injury and snow molds if they are to survive the winter. By comparison, in coastal British Columbia, wintergreen grasses are needed for all-season golf, football of various kinds and field hockey. Wintergreen grasses are often too cold- and snow mold-susceptible for the Prairies and management to maintain winter greenness does not improve the winter disease resistance of grasses. In any case, few people would choose to, or would be able to, play turf games in Regina, Saskatoon or Prince Albert after October in most years.

Although most of the winter diseases of turfgrasses which occur in the Prairies are also found in the colder parts of the Northern Hemisphere, there are some which appear to be absent or are rare in similar climatic regions. Overwintering problems of turfgrasses are a major consideration when formulating management strategies for amenity turf in the region.

The aims of this text are to examine the causes of winter damage to turfgrasses (mainly in the Canadian Prairies), and to indicate how some of them can be managed. It is hoped that much of the information contained herein may be useful in the remainder of the continental interior of northern North America and other regions of the world with severe winter climates. Some of it is also applicable to winter problems in agricultural grassland and winter cereals, since the same disease agents are involved.

(References to literature cited in this Preface will be found at the end of the next Section).

I. WINTER-HARDINESS AND OVERWINTERING DISEASE - INTRODUCTION

Winter-hardiness was defined by Vasil'yev (1961) as "the overall ability of plants to come through the winter successfully", by Levitt (1956) as "their (plants) ability to survive the severities of winter", and by Ruckenbauer (1974) as "the capacity of a plant to survive the vagaries of the very variable winter environment". According to Steponkus (1979) "winter-hardiness implies avoidance of or tolerance to all the cumulative effects of winter a plant encounters, including freezing, heaving, smothering and desiccation and disease." Fuller (1980) refers to it simply as "the resistance of plants to winter damage". Winter-hardiness is a very complex phenomenon.

Causes of winter damage in turfgrasses

The synonymous terms "winter injury" and "winter damage", with subsequent "winterkill" often appear in the literature. They are not precise enough descriptions when the causes of the failure of plants to survive in good condition are being examined to determine whether winter problems can be solved. A more precise definition of a type of injury is "winter-burn" in which the death of all or part of the existing leaf material reduces the biomass, but does not affect growing points or consequently, the spring growth potential.

Vasil'yev (1961) and Levitt (1956, 1972) have reviewed earlier ideas about the winterkilling of plants generally. Ekstrand (1955, 1955a), in Scandinavia considered the main causes of winter damage to winter cereals and grasses were cold injury, water and ice injury, frost-heaving, suffocating injury, freezing of hydrated tissues and low-temperature fungi. Jamalainen (1960) and Ylimaki (1962), in Finland and Anderson (1963), in northern Norway, reported that factors such as freezing, soil frost-heaving, standing water, ice layers and physiological drought, as well as low-temperature fungi were important in the overwintering of field crops, including grasses. A group of Scandinavian workers (NJFF, 1968) reduced the abiotic causes of winter injury to four, combining ice and water injury:

(1) Cold injury - which develops in winter in very cold locations where there is little or no snow cover and with autumn and spring frost.

(2) Water injury - which may develop under standing water or in frost-free or waterlogged soil under snow or ice.

(3) Frost-heaving

(4) Other non-parasitic injuries such as physiological drought. The latter can develop on snow-free land when the soil is frozen

and water lost by plants cannot be satisfied by the roots.

Five biotic causes of injuries were given:-

(1) Fusarium nivale (Microdochium nivale)

(2) Typhula spp.

(3) Sclerotinia (Myriosclerotinia) borealis

(4) Other fungi

(5) Noxious animals

In the northern USA, Smith, D. (1964) found that winter injury to forages was primarily the result of three factors operating singly or together viz:- low-temperature, including alternating temperature which was related to rapid freezing and thawing, exposure to warm temperatures and frost-heaving, smothering and desiccation.

Ruckenbauer (1974) in Vienna, considered several causes of winter injury in the development of research methodology for testing winter-hardiness of grasses. These were:-

(1) Direct-freezing injury - including (a) low-temperature killing because of inadequate cold resistance or inadequate hardening (see below) - (b) freeze-drying or the combined effects of cold and dehydration by wind - (c) spring injury caused by the diurnal variation in temperature.

(2) Snow damage - caused by the smothering effect of a long-duration snow cover combined with an ice crust.

(3) Indirect frost and snow injury including - (a) heaving of plants and root tearing through soil movement as a result of alternate freezing and thawing - (b) ice-burn caused by ice forming over plants combined with strong solar radiation - (c) fungal diseases under the snow caused by Microdochium nivale, Typhula spp. and other snow molds.

Beard (1978), working in Michigan, considered that the major types of winter injury to turfgrasses on golf courses were:- (1) Desiccation, (2) Direct low-temperature kill, (3) Low-temperature diseases, (4) Traffic effects.

In the Prairie Region of Canada snowfall is generally light when compared with the mid-western states of the USA and eastern Canada. Very low winter temperatures are experienced and there is a short, but usually adequate hardening period.

Early spring winds are usually dry and very cold. The main abiotic injuries to turfgrasses are direct low-temperature killing and desiccation injury during "chinook" periods when mid-

winter thaws occur or in early spring. Frost-heaving and injury associated with ice sheets are not so common. Of the biotic causes, Coprinus psychromorbidus (LTB and SLTB phases - see Sect. IIId) is the most prominent of the snow mold fungi, capable of causing damage under shallow snow covers. Except for M. nivale (see Sect. IIIa) which causes pre-hibernal disease, other snow molds require deeper snow covers. Traffic injury is accentuated by shallow snow covers (Smith, 1981 and unpublished). Meadow vole injury may be significant on turf adjacent to taller vegetation refuges (Smith, 1979a).

In Norway, particularly along the coast and in fjord districts with the prevailing raw, cold and foggy winter weather, low-temperature killing associated with ice- or water-covers is more severe than that caused by other abiotic factors. Where the winter climate is more stable and the snow cover deep, developing on unfrozen or lightly frozen ground, injury is most often caused by snow molds (Andersen, 1963; Arsvoll, 1973; Smith, 1975).

In Denmark, abiotic causes of injury are prominent, categorized as freezing injury, mainly on Lolium perenne, and damage associated with ice- or water-covers. Abiotic injuries are also more important than biotic in southern Sweden and southern and western Finland and in Iceland. Damage associated with ice/water covers and other direct-freezing injuries figure most prominently whenever there is little or no snow cover for long periods (Gudleifsson, 1979; Jamalainen, 1960, 1974; Jensen, 1970; Kristinsson & Gudleifsson, 1975; Smith, 1975 and unpublished; Ylimaki, 1962).

Although the prevailing climate of the British Isles is a cool-temperate one, different regions often have considerable differences in climatic conditions and it is difficult to generalize about the causes of winter injury to turf grasses. "Winter-burn" is a characteristic injury on many grasses, but particularly on perennial ryegrass (Baker & David, 1963; Hunt, 1969) and there are considerable differences in susceptibility between cultivars and large environment/cultivar interactions (Charles et al., 1975; Laycock & Shildrick, 1979). Low-temperature injury to grassland is often associated with ice/water covers, fluctuating temperatures in spring, fertilizer and management factors (Baker & David, 1963; Breese & Foster, 1970; Charles et al., 1975; Hides, 1978) which may lead to "winterkilling" (Hunt et al., 1976). In colder regions and in snowy years, such as 1962/63, winter injury and winterkilling can be related to specific snow molds such as Typhula incarnata and Microdochium nivale (Gray, 1963; Jackson, 1962).

Beard (1966) noted that low-temperature injury occurred principally at two critical periods, during late-winter thaws and just after the spring thaw when turfgrasses were at a reduced state of hardiness. The damage seemed to be most prominent in the region from Chicago east through Michigan, Ohio, Pennsylvania, New York and New England. However, in all the northern states snow molds are also prevalent on turfgrasses.

The usual causes of winterkilling of grasses and other plants in Alaska, particularly the introduced ones, are thought to be their failure to undergo adequate cold acclimation and/or excessive cold stress. In 1975, abundant soil moisture and air temperatures considerably above the 10-year average in mid- to late-October and much below this average from 25 October resulted in interrupted hardening followed by severe cold injury. There was no snow cover and air temperatures reached -12°C. Phleum pratense, which has its growing points at or above soil level, was severely damaged, but Poa pratensis, with rhizomes insulated by the soil, was not affected (Klebesadel, 1977).

II. OVERWINTERING DISEASES OF PHYSIOLOGICAL ORIGIN

a. Dormancy, hardening and overwintering

Cool-season turfgrasses do not go completely dormant, although many approach this state. They assume a quiescent winter condition which may be photoperiod- or temperature-induced. This is an adaptive mechanism for the survival of the plant through periods of low-temperature and other climatic vagaries (Shimada, 1982; Smith, 1978, 1980, 1981). The ability of the plant to overwinter under severe conditions is largely governed by its inherent physiological characteristics and its state of progress towards quiescence. Growth in mild winters and survival through severe ones are negatively correlated. In preparation for overwintering, a prehibernal or early-winter acclimation takes place in those species which have the physiological ability to do so under the influence of declining day-length and decreasing temperatures. This hardening or toughening process results from physiological changes in the plant which, among other things, make it more able to withstand low temperatures, freezing and thawing, desiccation and resistance to low-temperature pathogens. Hardening is a process which usually needs light and concurrent temperatures from 0°C to a few degrees above, sometimes as high as 10°C, depending on the species (Levitt, 1956). Recent studies by Tronsmo (1982) indicate that the relationship between resistance to freezing and snow mold infection in grasses after hardening is a complex one not necessarily originating in common mechanisms. It was found that after hardening there was a significant positive correlation between resistance to freezing and to the snow mold pathogen, Typhula ishikariensis, but in unhardened plants there was no significant correlation. Perhaps hardening only increases the inherent resistance while resistance to freezing is independent of the level of resistance before the plants are hardened.

By varying the temperature and day-length the relative cold-hardiness of three cultivars of Lolium perenne were modified during the hardening period and contrasting hardiness responses were evoked in the cultivars (Fuller & Eagles, 1980). Different ecotypes and climatic varieties of the same species of grasses may have different light and temperature requirements during the hardening period. Differences in temperatures and light energy input in the pre-hardening period may also influence survival through the accumulation and mobilization of organic reserves (Breese & Foster, 1970; Dexter, 1956; Fuller & Eagles, 1980; Lorenzetti et al., 1971; McColl & Cooper, 1967; Smith, D., 1964).

b. Metabolic and physiological changes during the hardening process

Although many changes have been found to take place during the cold-acclimation process, it is not yet certain which of them are causal or which result from low-temperature growth (Mazur, 1966, 1970; Smith, D., 1968; Steponkus, 1979). Some of these changes are:-

(1) A decrease in the total water content and an increase in the bound water content resulting in a low free water content (Levitt, 1939, 1956). This point is controversial.

(2) An increase in the hydrophilic (water-binding) proteins. Bound water held on the surface of these resists freezing by crystallization (Levitt, 1959). These soluble organic nitrogen compounds may also inhibit protoplasm precipitation (Maximov, 1930).

(3) An increase in cell sap concentration, especially storage starch and sugars (Levitt, 1972). With the onset of low temperatures the starch is converted to sugars. While the high concentration of sugars will lower the freezing point of the cell sap (Jung & Smith, D., 1961), the main role of these in frost-hardiness is probably an osmotic effect which may result in the inhibition of protoplasm precipitation.

(4) A physiological change which is related to metabolite concentration and total water content which takes place during the hardening process is an increase in permeability of the cell membranes (Levitt & Scarth, 1936). This is believed to facilitate the rapid withdrawal of water from the cell, reducing the risk of damage by intercellular ice formation (Smith, D., 1964).

(5) Changes in the buffering capacity of the protoplasm (Levitt, 1956).

(6) A reduced respiration rate which would conserve carbohydrate reserves (Dexter, 1933).

(7) Enzymic changes in peroxide isozyme components in plant tissues capable of cold-acclimation (McCowan et al., 1969).

For detailed discussions of these changes and others refer to Levitt (1956, 1972), Mayland & Cary (1970), Smith, D. (1964), Steponkus (1979) and Weiser (1970).

c. Dehardening

Dehardening is mainly caused by increasing temperatures, but

its progress can be modified by several other factors such as freezing and thawing cycles, depletion of available energy reserves, hormonal balance and prolonged periods of cold.

During the winter, grasses deplete carbohydrate reserves by respiration which goes on slowly in living tissues, even at sub-zero temperatures (Levitt, 1972), resulting in gradual dehardening. Further dehardening will take place in plants not covered by snow in warm periods in winter and their rehardening is dependent on adequate carbohydrate reserves and sufficiently long periods of suitable light and temperature (see Hardening). Under a prolonged, deep snow cover dehardening results from an exhaustion of reserves in the absence of photosynthesis, especially if the soil is unfrozen. This also disposes grass plants to attacks by snow molds (Bruehl & Cunfer, 1971; Tomiyama, 1955). If the soil is frozen reserves suffice and plants overwinter (Hunkuna, 1974). Sudden cold spells in spring when nutrient reserves are low, following warm weather when plants start growing, are particularly damaging. Cold resistance in cool-season turfgrasses (Poa pratensis, Agrostis sp. and Festuca arundinacea) increases in fall and winter until about late January and then may decline rapidly in spring (Beard, 1966; Powell et al., 1967; Wilkinson & Duff, 1972).

d. Climate and overwintering problems with cool- and warm-season species

In temperate climates winter is the "off-season" for the growth of turfgrasses. This is mainly because, in winter, temperatures are at their lowest. In the tropics the annual temperature differential may be small and growth is slowest in the dry season. Temperature largely determines the possibility of using a turfgrass species or cultivar in a particular region, although day-length, climatic, edaphic or biotic factors may govern its success there (Hartley, 1950; Hartley & Williams, 1956). The relative importance of the climatic factors which cause winter problems varies considerably in different parts of the world and these are dependent mainly on latitude (or altitude) and location of the place in relation to great land masses, large bodies of water and mountains. These affect the severity of the winter climate, the duration of the winter season, the depth and duration of the snow covers and the prevalence of warm katabatic winds such as the "foehn" or "chinook" which may rapidly melt protective snow covers.

Turfgrass species are used in many regions to which they are not native and they may be maintained there by artificial means, particularly by the use of irrigation, nitrogenous fertilizer and plant protectants. The misuse of these may lead to lowered resistance to winter damage. The demand for an extended playing season may require use of fertilizer and water to maintain colour in turf in autumn when the grass would normally tend to dormancy, a natural protection against winter injury, particularly in very

severe climates. There has been a tendency, mainly in the cool-season grasses, for commercial reasons, to attempt to extend the use of successful turfgrass cultivars much beyond the limit of their best adaptation (Smith, 1981). Cultivars of cool-season species such as Poa pratensis, Agrostis stolonifera, Festuca rubra and Lolium perenne are frequently used as turfgrasses where they encounter much lower temperatures, ice formation, desiccation or snow covers to which they are not adapted. A species such as Poa pratensis, which is widely distributed in Europe, temperate Asia and North Africa (Whyte et al., 1959), shows considerable genetic variability and clones can be found within it showing considerable winter-hardiness, but even so, the limit of winter insults which these will tolerate may be reached, for example, in the Canadian prairies. Warm-season grasses in genera, such as Cynodon, Zoysia, and Paspalum and species such as Stenotaphrum secundatum and Eremochloa ophiuroides, have much lower tolerances to low temperatures than cool-season turfgrasses (Johnston & Dickens, 1976; Smith, D., 1964; Whyte et al., 1959). Some tropical grasses may suffer winter injury by chilling at temperatures above freezing point (Johnston & Dickens, 1976; Sellschop & Salmon, 1928; Smith, D., 1964). Low temperatures in winter induce deep dormancy in some warm-season turfgrasses to the extent that oversowing with an annual cool-season grass is necessary to produce a green playing surface (Younger, 1959; Ward et al., 1974). Complete winter dormancy is not shown by cool-season grasses and they may resume aerial growth in winter when temperatures rise a few degrees above freezing. This may be to the detriment of their winter-hardiness.

Rogler (1943) found that southern strains of warm-season grass species were so late in maturing in the northern USA (North Dakota) that fall frosts injured them while still growing vigorously, but northern strains of warm-season grasses had started to quiesce before the first frost. He concluded that for good survival, warm-season species must be quiescent at first frost. Northern and southern strains of cool-temperature species did not show such marked differences in maturity (at the latitude of North Dakota) and no cases of winterkilling occurred.

Cooper (1964) showed that in natural populations of ryegrasses from a European transect embracing the Baltic to the Mediterranean the proportion of plants surviving freezing temperatures of $-5^{\circ}C$ was greater in more northerly populations. These conclusions were supported by the results of similar studies with a wide range of ecotypes and climatic varieties by Lorenzetti et al. (1971). This evolutionary development was referred to as "resistance adaptation" by Levins (1969). Northerly types had a lower capacity for leaf extension and growth than those from further south, that is, "resistance adaptation" and "capacity adaptation" were negatively correlated (Breese & Foster, 1971). While the lower growth potential is disadvantageous in forage grasses their lower productivity and greater cold tolerance would suit them to a turfgrass role in

more severe winter climates.

e. The effect of soil fertility

Adequate supplies of N, P and K and minor nutrients are necessary to ensure plant vigor and maintain the metabolic processes necessary for cold-acclimation. K and P increase frost-hardiness of plants and high N applications almost always decrease it (Dexter, 1956; Kresge, 1974; Levitt, 1956). There are exceptions to this rule. N applications decrease accumulation of sugars and of bound water, i.e. increased tissue hydration (Carroll, 1943; Carroll & Welton, 1939). Heavy, and late-season applications of N during the hardening period decreased cold-hardiness of seven cool-season turfgrasses (Carroll, 1943). An N to K ratio of 2:1 to 3:1 resulted in the maximum hardiness for Poa pratensis (Beard & Rieke, 1966). In Poa pratensis N fertilizer applied up to 1 November reduced cold resistance most in the autumn of the same year, but late fall fertilization (after 1 November) resulted in greatly reduced cold resistance in spring next year. Cold resistance in all treatments increased until mid-winter, peaked in late January and declined to spring (Wilkinson & Duff, 1972). In bentgrasses and tall fescue reserve carbohydrates increased from autumn to winter and then decreased to spring when the decline was rapid (Powell et al., 1967). Following the very severe winter of 1962/63 and in subsequent milder winters in Britain, the severity of winter-damage on Lolium spp. was proportional to the N dosage (Baker & David, 1963; Breese & Foster, 1970). Northern populations with higher resistance tended to accumulate their assimilates (usually as soluble carbohydrates) in the leaves, leaf bases and roots while the southern populations used these assimilates for new leaf production and extension (Breese & Foster, 1970). In bentgrasses and tall fescue, higher N reduced available carbohydrates; there were more carbohydrates in stems than in leaves and stem carbohydrates declined from 44% of the dry matter in late fall or winter to 5% in spring (Powell et al., 1967). High nitrogen applications to ley grasses in Northern Finland resulted in complete or almost complete destruction of Phleum pratense and Festuca pratensis from abiotic winter injury (Jamalainen, 1960). In Festuca pratensis and Dactylis glomerata heavy N (300-600 kg/ha) resulted in serious sward damage, abiotic in origin, when a snow cover persisted for 6 months on unfrozen ground. The least damage was seen with a dosage of 150 kg/ha N. Swards receiving no nitrogen were weakest. Increasing N resulted in reduction of total soluble carbohydrates in plant crowns and roots (Huokona, 1974).

The warm-season St. Augustinegrass (Stenotaphrum secundatum) is one of the least winter-hardy turfgrasses in the southern USA. Late applications of N alone or in combination with P and K had little effect on winterkill, but increased the period when it remained green in autumn and resumed growth in spring. The level

of K and P in tissues was related to tissue N level (Reeves & McBee, 1972). In Coastal bermudagrass, Cynodon dactylon, winter injury at high levels of N decreased with increasing K levels and at a given level of potash, injury increased with given levels of nitrogen (Adams & Twersky, 1959). In Tifgreen bermudagrass (Cynodon dactylon X C. transvaalensis) nutrient level did not appear to alter the temperature range for killing between -2.2 and -4.4°C. High tissue N levels did not much affect cold-hardiness. A high P/K increased winterkill, but low ratios showed little damage. Tissue N level influenced uptake of P and K. Late fall applications of N extended lateness of fall growth and encouraged early spring growth (Reeves et al., 1970).

Freezing tolerance of Phleum pratense decreased signficantly with increasing supply of N and increased with increasing supply of P. There was no consistent effect of K. Total soluble carbohydrates in leaves and roots was positively correlated with freezing tolerance. The N content of hardened plants, increasing with increasing supply of N was negatively correlated with freezing tolerance, but not with the P and K content (Arsvoll & Larsen, 1977).

In Pennsylvania, Jung & Kocher (1974) found that N fertilization at rates from 0-240 kg/ha differentially affected winter survival of 39 clipped grass cultivars (see Effect of Mowing). Of the turfgrasses little or no winter injury occurred on Park and Pennblue P. pratensis and significantly less on Norlea L. perenne of Canadian origin than on Lolium spp. of European and more southerly origin in the USA.

Andersen (1960) found that autumn applications of N alone at 31 and 62 kg/ha did not improve overwintering of grasses. Where P was deficient in the soil 31 kg N, 50 kg K and 12 kg P per hectare improved wintering but did not where P was in satisfactory amounts.

f. Differential cold-hardiness of plant tissues

Generally, older tissues are more severely damaged by frost than younger ones, provided that energy reserves are adequate. Younger tissues have less free water and more cytoplasm which enables them to resist freezing better (Vasil'yev, 1961). In wheat, the leaf tip is not quite as hardy as the lower part of the leaf (Dr. L. V. Gusta, pers. comm.). Studies made by Beard & Olien (1963), Beard (1964), and Peake (1963) have suggested that grass leaves and roots are more sensitive to low-temperature injury than crowns. More recent results obtained by Gusta et al. (1980) suggest that this may not be so for all grasses. Using an electrolyte leakage test they found in Fylking Poa pratensis that leaves were the hardiest part of the plant followed by crowns, with roots and rhizomes the least and of equal hardiness.

Noshiro (1982) has found that grasses with above-ground crowns, such as Dactylis glomerata, Festuca pratensis and Lolium perenne were injured by freezing at -7 to -10°C for 16 h and were

killed at -15°C. Primordia of flowering tillers were injured at -7 to -10°C. Of other grasses Phleum pratense was the hardiest with a critical temperature for crown death of -25°C. Poa pratensis was nearly as hardy. The rhizomes or shoot bases (corms) of Festuca arundinacea, Bromus inermis, an Agropyron sp. and Phalaris arundinacea were heavily damaged by freezing at -13 to -20°C, but they could survive in the field because the organs were placed 1-5 cm below the soil surface and hence protected from being cooled to the critical death temperature. Leaves were the hardiest organs and roots and rhizomes generally the least hardy in 9 grass species.

Young leaves are more frost-hardy than old ones (Baker & David, 1963; Vasil'yev, 1961). However, unless there is a complete ice or water cover, roots and crowns receive some thermal protection from low air temperatures by their immersion in or proximity to the soil (Klebesadel, 1977; Kolosova, 1941; Nostura, 1982). The plant cover provided by dead or living leaves over the crowns gives additional insulation from low air temperatures. Crown tissues differ in their resistance to cold injury. The upper crown of Poa annua with its smaller, closely-packed, undifferentiated cells (Kolkunov, 1931; Levitt, 1956; Vasil'yev, 1961) is more frost-tolerant than the lower crown tissues where roots arise. When fully hydrated, following immersion in melt water, these lower crown tissues are susceptible to direct freezing injury (Beard & Olien, 1963). Thomas and Lazenby (1968) found that there were differences in the cold tolerance of shoots and roots of Festuca arundinacea. In three populations, some tillers were killed after exposure of roots to temperatures of -8°C, but even at -11°C there was not complete kill. When shoots were exposed to temperatures below -6°C in two synthetics and below -7°C in the other, some tiller death occurred. Complete killing took place when shoots were exposed to -11°C. Cold tolerance of a plant per se apparently cannot be assessed by the subsequent survival of the whole. Plant roots and shoots are indispensable in that healthy shoots could replace damaged roots but not vice versa. The importance of the crown or tillering node being submerged in the soil where it is protected from low winter temperature has been stressed by Kolsova (1941) and Smith, D. (1964). It is this area where roots are regenerated and the more cold-susceptible meristematic tissues are located.

g. The effect of mowing or clipping on winter-hardiness

Grasses which are mown, clipped or grazed often or late in the season are often in the position of not having accumulated sufficient nutrient reserves at the start of winter to acquire sufficient hardiness or they lose these reserves and hardiness before the winter is over (Andersen, 1960, 1963). Photosynthetic tissues are necessary to synthesize the carbohydrates needed to develop hardiness, to survive the winter and to initiate growth

in spring (Larin, 1962; Smith, D., 1964a). Huokuna (1974) in Finland, found that the most important factor affecting the overwintering of Festuca pratensis was the harvest date. Late clipping (in mid-September) reduced total soluble carbohydrates and greatly reduced winter survival. This could also be a hormonal effect, not just carbohydrate depletion. Jung & Kocher (1974) in Pennsylvania, found that clipping differentially affected winter survival of 39 cool-season grass cultivars nearly as much as nitrogen fertilization (q.v.). Plants were either clipped before heading and then in early June, late July and October or after heading and again in late August and early October. Of the turfgrasses Poa pratensis was little affected regardless of clipping treatment. Festuca arundinacea and Lolium perenne cultivars were injured more by after-heading clipping than by clipping in the vegetative state. The effect of autumn and winter mowing of 17 strains of the warm-season lovegrasses, Eragrostis curvula and the more cold-hardy E. lohmanniana on winter survival was compared by Voigt (1975). After mild winters strain survival was strongly correlated with limited, late autumn regrowth. After a severe winter, survival was associated with the presence of live, green leaves in late autumn.

h. Ice and water injury

Ice injury is a broad term referring more to the situation when damage occurs rather than to the cause of injury. It occurs when turf has been frozen in an ice sheet or when plants have been encased in ice. Water lying in depressions over a frozen or waterlogged soil or developed from sleet showers may freeze in autumn and early winter and persist until spring (Fig. 1). An ice cover may also develop from diurnal thawing and refreezing of a snow cover during winter and early spring, particularly in northern and coastal areas (Andersen, 1963; Gudleifsson, 1971, 1975, 1979; Sjoseth, 1959). Three different types of ice/water situations have been suggested by Ekstrand (1955) as leading to plant injury and death under Scandinavian conditions: Root-suffocation which occurs when the soil is waterlogged and unfrozen in autumn and a snow deck builds up on this: Ice-suffocation when waterlogged soil is covered with an intact ice sheet resulting from successive snowfalls, rains, thaws, snows, etc. Plant death was considered to have been caused by a combination of smothering or suffocation and direct freezing injury: Ice-burning occurs in patches in shallow depressions where the water is a centimetre or so deep. This water impregnates the crowns of the plants and freezing injury results. Usually complete patches of plants are killed. This injury appears to be identical to that described by Beard & Olien (1963) as direct low-temperature kill in lower crowns of Poa annua.

Since ice is a good conductor of heat in comparison with fresh snow it will permit low-temperature damage to plants embedded or encased in it in direct relation to air temperature

(Ylimaki, 1962).

Oxygen starvation (Ylimaki, 1962) or the accumulation of carbon dioxide (Smith, D., 1952), alcohols (Vasil'yev, 1961), lactic acid (Andrews & Pomeroy, 1979), or toxic gasses, such as hydrogen cyanide produced by fungal metabolism (Lebeau, 1966) have been suggested as possible causes of death or injury to plants under snow or ice/water covers. While there seems to be some experimental evidence in support of these as causes in particular cases, during early and mid-winter there is probably sufficient oxygen, even under deep snow, to permit adequate respiration of dormant plants (Gabran, 1935; Tumanov, 1940; Tumanov et al., 1935). As temperatures rise in spring, plant respiration rate increases and the snow becomes compacted by thawing, sufficient oxygen lack and carbon dioxide concentration may develop to reduce plant-hardiness to a level which they become susceptible to snow mold injury (Bruehl & Cunfer, 1971; Tomiyama, 1955; Tumanov et al., 1935). However, it is doubtful whether a sufficient concentration of carbon dioxide would develop, even under deep snow covers, to produce plant injury (Pichler, 1948). Ice up to 6 cm thick has been shown to be quite permeable to air (Tumanov, 1940) and Vasil'yev (1961) noted that perennial grasses wintering under ice in ditch bottoms showed no injury.

Sjoseth (1959) examined the effect of ice encasement on nine strains of Phleum pratense and one of P. alpinum. Plants were held at 6-8°C after the seedlings had grown for 2-4 months to slow down their growth rate*. Some were then fully hardened at 1.5°C/14 days. The soil in which they were growing was then frozen at -2.5°C and then the plants were covered with water and frozen in ice at the same temperature for 40 days. Controls held at -2.5°C, without ice cover, were provided. In the hardened plants survival in ice ranged from 82-58% and in the unhardened 81-28%. It was concluded that the ability of strains to withstand ice encasement was broadly related to frost-hardiness as determined by laboratory freezing tests (unspecified) and to winter-hardiness of strains in field experiments.

Beard (1965, 1965a) showed, in controlled environment studies, that there were differential responses in turfgrass species and cultivars. Freezing in an ice block at -4°C resulted in 100% mortality of Poa annua after 15 days and P. pratensis after 45 days. Toronto Agrostis palustris survived completely at 60 days; there was 70% kill at 75 days and 90% kill at 90 days. Death was not caused by intercellular ice formation in lower crown cells (Beard & Olien, 1963).

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*Probably some hardening took place at this temperature (J.D.S.).

A snow cover on the plants, with an ice cover on top, produced only minor injury to older leaves in all three species. An ice cover formed over flooded and frozen soil resulted in complete survival of all species at 60 days, but in 50% kill in P. annua and 25% kill in P. pratensis after 90 days. When submerged in stagnant water at 1°C, A. palustris survived completely and there was little damage to P. pratensis and P. annua after 90 days. In similar studies in the field, a compacted frozen slush on the turf caused severe injury to leaf, crown and rhizome tissues of P. pratensis, but less in A. palustris. Layering of ice over snow for 6 days resulted in no significant crown, rhizome or stolon kill, but ice layers formed directly on plants caused some leaf injury by no crown, rhizome or stolon injury to either species (Beard, 1965a).

Andersen (1963, 1971) at Tromso, Norway, at latitude 69°N, found that only the hardiest cultivars of Phleum pratense, derived from local and northern Scandinavian lines, overwintered satisfactorily with a prolonged covering. Similarly, only a local strain of Poa pratensis, Holt cv. was fully hardy when ice covered. Festuca pratensis strains were not as hardy as the poorest of Phleum pratense.

Andrews & Gudleifsson (1983) found that although seedlings of timothy, (Phleum pratense L.) showed relatively low cold-hardiness, compared with winter wheat, they had about a threefold greater ice tolerance than the cereals in controlled environment studies. There was little association between cold and ice-tolerance in P. pratense. An Icelandic timothy cultivar, Korpa, was more tolerant than the Norwegian, Engmo, and much more ice-tolerant than the Canadian cultivar, Salvo. It was considered that high ice tolerance was a major reason for superior survival of P. pratense in high winter-stress conditions.

i. The nature of frost injury

Although the way in which freezing kills plant cells is still imperfectly understood, it is related to ice formation and probably cell dehydration (Steponkus, 1979). Undercooling of cells of hardy and non-hardy plants may result in the formation of intracellular ice crystals (Siminovitch & Scarth, 1938), but undercooling without ice formation can take place in plants of temperate and cool regions to temperatures below which they would be killed if ice had formed (Scarth, 1944). Although the freezing point of cytoplasm is usually about -1.5°C, cells generally remain unfrozen, because they are undercooled, at temperatures of -10 or even -15°C, even when surrounded by a medium which is frozen. The cell membrane prevents the growth of extracellular ice into the cell interior which is undercooled (Mazur, 1970). When hardened plant material is slowly cooled, ice forms first in the extracellular spaces (Levitt, 1956). This is equilibrium freezing. Under natural conditions temperature drops are usually gradual. Ice formation is initiated by a

process known as nucleation, the exact mechanism of which is uncertain (Mayland & Cary, 1970). As this freezing occurs, water from the surrounding cells diffuses through the semi-permeable membranes as a result of an attempt to maintain osmotic equilibrium, since freezing lowers the osmotic equilibrium pressure of ice. Once equilibrium temperature is reached between the plant tissue and the external environment osmotic equilibrium will also develop. If extracellular freezing has occurred, dehydration of the cells should also be at maximum and further damage to the cells should cease. However, the length of time the cells remain frozen and how close the temperature is to the lethal temperature appears to influence whether further damage occurs (Pomeroy et al., 1975). It has been shown in laboratory experiments that if the temperature is decreased more rapidly, ice crystals may form in the cytoplasm (Siminovitch & Scarth, 1938). In these circumstances, the cells become fully permeable because of rupture of or structural changes in the plasma membrane (Mazur, 1966). Ice formation within the protoplast, that is intracellular freezing, which is a non-equilibrium process, seems always to be fatal to the cell (Habeshaw, 1973; Levitt, 1956). However, Levitt (1956) suggested that it occurred rarely in the field because high rates of temperature fall were usually not observed. Habeshaw (1976) agreed generally with Levitt (1956) that it would occur rarely in hardened grasses under any but exceptional circumstances (under British climatic conditions?). However, he (Habeshaw, 1973) had found by microscopic observation that intracellular freezing could occur under much less severe conditions than were generally assumed and that tissues frozen intracellularly could change their appearance to that of extracellularly frozen as temperatures continued to fall. Unhardened plants would undercool considerably under much milder conditions than hardened ones and would be at risk from intracellular freezing during autumn and spring. If the cooling rate is above the minimum rate for undercooling then the plant could undercool without ice formation and survive, but if it is below then extracellular ice would form. Grasses differ considerably in their ability to undercool without freezing (Habeshaw, 1976). Unhardened Festuca pratensis and Phleum pratense would not undercool much without freezing. Agrostis and Poa spp. were intermediate and Lolium multiflorum and Lolium perenne would undercool to a lower temperature without freezing. This can be related to the ability of unhardened plants of the different grass species to survive sharp frosts, i.e. radiation frosts, in otherwise warm periods.

j. Low-temperature killing

There are many factors which are related to the susceptibility of species and cultivars of turfgrasses or forage grass species, which are used in turf also, to low-temperature injury, both direct and indirect. These include:- clipping (Jung

& Kocher, 1974; Thompson, 1977; Voigt, 1975); degree of acclimation (Beard, 1966; Lorenzetti et al., 1973; Wilkinson & Duff, 1972); duration of freezing (Lorenzetti et al., 1973; Vorst, 1966); freezing temperature (Adachi et al., 1976; Carroll, 1943; Gusta et al., 1980; Habeshaw, 1976; Lorenzetti et al., 1973); frequency of freezing (Vorst, 1966); ice or water cover (Andersen, 1963; Beard, 1964; Ekstrand, 1955; NJFF, 1968); inherent ability to harden (Adachi et al., 1976; Baker & David, 1963; Cooper, 1964; Habeshaw, 1976; Lawrence et al., 1973; McCowan et al., 1969); plant health (Cormack, 1948; Esau, 1957; Paliwal & Andrews, 1979); snow, slush or ice suffocation (Andersen, 1963; Beard, 1965; Ekstrand, 1955; Ylimaki, 1962); soil fertility (Adams & Twersky, 1959; Arsvoll & Larson, 1977; Baker & David, 1963; Beard & Rieke, 1966; Breese & Foster, 1970; Carroll, 1943; Carroll & Welton, 1938; Cook & Duff, 1976; Cordukes et al., 1976; Huokuna, 1974; Jamalainen, 1970, 1978; Johnson & Dickens, 1976; Jung & Kocher, 1974; Kresge, 1974; Nissinen, 1970; Powell et al., 1967; Reeves & McBee, 1972; Wilkinson & Duff, 1972); soil moisture (Peake, 1964); stage of growth (Arakeri & Schmid, 1949; Rogler, 1943; White & Horner, 1943); topography (Andersen, 1963; Gudleifsson, 1978; Smith, 1975); unavailable soil moisture or desiccation (Beard, 1966, 1978; Ekstrand, 1955; NJFF, 1968).

k. Desiccation injury

Injury and death of turfgrass may result from the inability of conducting systems of roots and shoots to supply sufficient water to make up for moisture losses from plant crowns and other aerial parts. In winter, this may occur because turf is not protected by snow cover, the soil is frozen or short of water, or the grass root systems have been damaged. Cold, drying winds may cause desiccation by "freeze-drying". Exposed, elevated turf sites are particularly prone to desiccation injury in late winter and early spring in the northern plains of North America (Fig. 2). Some desiccation of leaves following snow melt, called "winter- burn" frequently occurs, but it is when crown tissues become dried out that severe plant damage may result. Desiccation injury is the result of physiological drought, and in most cases drought-hardiness and frost-hardiness are correlated (Gusta et al., 1980; Levitt, 1956).

l. Protection from frost injury by snow covers

In cold weather, a snow cover will insulate turfgrasses from air temperatures which are much lower than those of the soil. This is because the thermal conductivity of snow is many times lower than that of soil, especially when the snow is fresh and uncompacted. As snow ages and consolidates, its insulating properties decline (Ylimaki, 1962). Snow also protects soil from further heat loss. On the other hand, because the reflectivity

of snow is high and because of its good insulating properties it moderates the heating effect of any sunshine on the soil below it. The length of grass can influence the development, maintenance and rate of disappearance of a snow cover. This was illustrated by Monteith (1956) in relation to the differential effects of short turf of a cricket square and longer turf of the outfield on snow covers. Long grass will trap snow more effectively than short, but where the grass is short there is better thermal contact between it and the snow cover and in the case of the latter this increases with compaction such as results from rolling, a commonly used management practice on cricket wickets.

The deeper the snow cover the greater the insulation and the more stable the temperature regime is in the turf (Geiger, 1965; Noshiro, 1982; Smith, 1979; Ylimaki, 1962) up to a maximum of about 20 cm depth. Even 1 cm of snow gives some plant protection, 5 cm gives effective protection, and at about 20 cm the maximum protection for plants (Buhrer, 1902; Kokkonen, 1942; Ylimaki, 1962). Deeper snow takes longer to melt and, apart from encouraging snow molds, may kill grasses by smothering, particularly if partly converted to ice. When the air temperature is $-30^{\circ}C$ a 20 cm-thick snow cover can maintain the 5 cm soil temperature at $-10^{\circ}C$ (Smith, 1979). For a 6-week period from 20 December 1979, under a snow cover of 20-28 cm, the turf surface temperature varied from -5 to $-13^{\circ}C$ when air temperatures ranged from -10 to $-35^{\circ}C$ (Smith, 1979). A comparison of meteorological data in a snow mold season (1973-1974) and a non-snow mold season (1974-1975), at Saskatoon is given in Fig. 3. The greatest protection from freezing injury is given to turf when a permanent snow cover develops early and goes late in spring, but if the soil is not frozen or only slightly frozen before the development of a thick, permanent snow blanket, humidity and temperature conditions may be very suitable for the development of snow molds.

m. <u>The effect of alternate freezing and thawing</u>

When there is no snow and little vegetation cover to act as insulation, temperatures which vary from above to below freezing point may cause damage to overwintering plants (Sprague, 1955). Periods like this occur mainly during late autumn and early winter and in late winter and early spring. Rapid freezing and thawing cause more cell damage than when these processes are slow (Gusta & Fowler, 1977; Sprague, 1955; Vasil'yev, 1961). Warm periods of a few days duration at the end of the growing season may interrupt hardening, particularly if growth recommences, but periods of a few hours duration probably interfere little with the hardening process (Pomeroy et al., 1975). In late winter when carbohydrate reserves are low, rehardening after a warm period is less certain.

n. Frost-heaving

In frost-heaving plants are lifted up in the soil and their crowns and roots exposed to desiccation and low-temperature injury. In mature plants there is damage to roots, stolons, rhizomes and shoot bases while grass seedlings may be lifted almost completely from the soil. On established turf an undulating surface may be produced but there is usually little permanent damage other than to levels, which may be severely disturbed (Fig. 4). Frost-heaving is not caused by simple freezing and thawing but by the formation of vertically positioned ice lenses made up of bundles of needle-like ice crystals (McCool & Bouyoucos, 1929). The grass roots are firmly fixed in position by freezing and a thin ice forms on the soil surface. This continues to grow from below, being supplied by water moving through the soil, molecule by molecule to the point of freezing. The mechanism is not entirely understood. This movement is only possible in soils with a high capilliary pore content (Jackson et al., 1966; Nikki, 1974). The frozen soil acts like a desiccated soil layer and attracts water from the surrounding unfrozen soil (Dr. Y. W. Jame, pers. comm., 1980). This movement will continue so long as the temperature gradient persists and heat is provided by the freezing process. Further water is provided by the melting of the surface ice (Beard, 1973). Gradually the plants are raised while fixed in the ice crust, sufficiently in some cases, to break or tear roots and shoot tissues. In the case of seedlings, these may be left lying on the surface when the ice melts.

Cavities which develop under plants, particularly in grasslands at high altitudes and in Northern Japan have been attributed to ice lens formation. Many of these cavities are lenticular in shape and are interconnected by long tunnels. Nikki (1974) explained their formation in terms of ice needle accumulation under a frozen surface layer of soil with the lens developing horizontally. The lenses do not form directly under plants but as they build up, the surface soil layer is heaved as whole, tearing plant roots. In undisturbed soils, lens needle crystals are more abundant than in bare soil or on soils carrying plants other than grasses, but the soil is more deeply frozen under grass. A densely developed root system appears to possess the ability to prevent ice lens formation and frozen soil under plant roots has a lower moisture content. Fine soil particles are needed for the initiation of crystal formation and there must be enough soil moisture to permit their development. A similar explanation is advanced for the heaving of mature turfgrass such as is shown in Fig. 4.

o. Effect of topography and traffic

Topographic profiles play a major role in determining location, depth and duration of snow covers, accumulation of

precipitation and formation of ice sheets. They influence wind speed, katabatic temperature effects and frost development. These factors affect the type and severity of direct and indirect freezing injury. Where water and ice injuries are important severity of damage has been shown to be negatively correlated with raised areas in grassland and positively correlated with depressions (Andersen, 1963; Gudleifsson, 1975). The latter found that soil samples from damaged areas had greater loss on ignition, less air space, less exchangeable Mg and Ca, lower base exchange and a lower pH than undamaged turf. Topography was more important than fertilization in determining injury. Even microtopographical features resulting from tractor and golf cart ruts and skid marks (Dahlsson, 1975), ski pressure and foot traffic may be associated with winter-damage (Fig. 5). Frost formation is often greater round rut profiles than elsewhere because more moisture is supplied to the frost-face leading to ice/water injury (Andersen, 1963). Foot and ski traffic on shallow snow covers over turf reduces their protective insulation. Footmark injury occurs on grass already frozen by crushing and tearing of tissues before the development of a snow cover.

p. Stage of development and survival

Low-temperature-hardiness is related to stage of development of the grass plant. White & Horner (1943) in tests over 5 years in Saskatchewan found a positive correlation between stage of development of seedlings of Agropyron cristatum, A. trachycaulum and Bromus inermis at freeze-up and winter survival. Seedlings which had not emerged or had just emerged 7-10 days before freeze-up survived poorly, but those which had reached the 3-leaf or later stage survived well. Although there was a difference in survival from season to season there was the same relationship between size of plant and season over the whole test period. Similar results were obtained by Arakeri & Schmid (1949) in Minnesota with the cool-season grasses, Phleum pratense, Festuca elatior, Phalaris arundinacea and Poa pratensis. The observations of Scandinavian workers indicate that first-year agricultural grasslands are generally more severely damaged by the snow mold fungi Typhula ishikariensis and Myriosclerotinia borealis than older ones (Arsvoll, 1973, 1977), but in Norway, at least, first-year grassland showed the highest tolerance to the stress of abiotic factors (Arsvoll, 1973). Seedling resistance to Microdochium nivale, T. ishikariensis and M. borealis in Phleum pratense increased between 2 and 16 weeks and there was a highly significant, positive correlation between fungal resistance and freezing tolerance (Arsvoll, 1977). In the Canadian prairies, first-year turfgrass usually escapes severe snow mold but this may be due to lack of inoculum (Smith, unpublished).

Rogler (1943) found that the ability to survive cold

temperatures of northern and southern climatic types of warm-season grass species of the USA was inherited. Both seedlings and mature plants showed the survival characteristic. Southern types were less able to harden-off as seedlings or mature plants in North Dakota than were northern types. Seedlings of warm-season species from the south were more susceptible to freezing injury than those from the north and less capable of withstanding low temperatures than the latter.

q. Evaluation of turfgrasses for cold-hardiness

Annual variation in severity of winter injury to turfgrasses is considerable, particularly in regions with more temperate climates. The relative importance of the different causes of winter injury also varies from year to year, even when overall damage is high. Although breeding material can be evaluated for cold-hardiness in the field (Baadshaug, 1973; Weibel & Quisenberry, 1941) it requires several seasons since winters severe enough to damage the most hardy plants occur infrequently and injury escape is common (Levitt, 1956). Although winter injury is usually due to a complex of causes, the ability to withstand cold temperatures is often the most important and is amenable to study in controlled environments. Techniques have been developed to simulate the natural processes associated with cold-hardiness, hardening or acclimation, freezing, thawing, dehardening and recovery. These have been reviewed by Dexter (1956), Levitt (1956), Steponkus (1979) and Weibel & Quisenberry (1941). It is then possible to have control over the severity and consistency of some climatic stresses to the exclusion of others and to make more rapid progress in the understanding of physiological and biochemical processes and in the preliminary stages in the development of more cold-resistant cultivars. The winter-hardiness of winter cereals and forage crops have received considerable attention in the colder regions of northern temperate climates of North America, Britain, Scandinavia, northern Europe, USSR and Japan. Most of the work on forages, including some grasses which are used in turf, has been concerned with general winter-hardiness and survival sometimes including a consideration of abiotic and biotic factors. Until the 1970's, most of the studies on cold-hardiness of turfgrass species had been done in the USA (Beard, 1973; Wit, 1952). Damage to grasslands in Britain in the severe winter of 1962/63 was considered to be due mainly to cold injury aggravated by high usage of nitrogenous fertilizer (Breese & Foster, 1970; Hunt, 1969; Monson & Wright, 1972; Thompson, 1977). This further stimulated the development of techniques and the evaluation of cold-hardiness of ecotypes, cultivars and breeding material of Lolium perenne, an important forage and turfgrass species (Breese & Foster, 1970; Fuller & Eagles, 1978; Lawrence et al., 1973; Lorenzetti et al., 1971; Thomson, 1974; Thomson & Wright, 1972). Cooper (1964) used a freezing test to study the cold-hardiness of

natural populations of Lolium spp. and Dactylis glomerata from a north-south transect in Europe. Arsvoll (1973) found that there was a highly significant, positive correlation between freezing tolerance and fungal resistance in Phleum pratense and Festuca pratensis. Adachi et al. (1976) used a freezing test to show whether after hardening, perennial ryegrass cultivars had acquired enough winter-hardiness to tolerate low temperatures experienced before a snow cover developed. Gusta et al. (1980) have used a controlled freezing test to study low-temperature-hardiness of cool-season turfgrasses. Cloutier (1982) has described a rapid method for determining small differences in frost-hardiness in winter cereals which may be applicable to turfgrass studies. The long period of cold-acclimation required to induce frost-hardiness is replaced with a desiccation-stress technique. Survival is assessed by the regrowth and greening of epicotyls on agar and regrowth of whole seedlings in vermiculite.

Instead of using a direct-freezing technique some methods of determining potential for effective hardening are indirect. These are based on empirical correlations between some anatomical or physiological character and cold-hardiness. These include such characters as width of leaves, habit of growth, date of heading, dry matter or sucrose content of sap, osmotic pressure, viscosity of protoplasm, electrical conductivity and respiration rate (Dexter, 1956; Steponkus, 1979; Wilner & Brach, 1979; Wit, 1952). These are generally much less reliable than direct freezing and assessment of plant survival (Levitt, 1972).

r. Relative winter-hardiness of turfgrasses

Considerable differences in cold-hardiness are shown by species and cultivars of turf grasses, but their reactions to cold injury may also be related to their snow mold resistance which is another important factor in winter survival. Cold-acclimation, which involves the acquisition and maintenance of adequate carbohydrate reserves is also important in resistance to snow mold fungi (Arsvoll, 1975; Bruehl & Cunfer, 1971; Tomiyama, 1955). Arsvoll (1977) found that there was a highly significant positive correlation between snow mold resistance and freezing tolerance.

Carroll (1943) found that the lethal soil temperature for cold-hardened, cool-season grasses was between -10 and -15°C. Some Poa pratensis plants survived at -20°C. Poa nemoralis, Agrostis tenuis and Festuca rubra fallax were the least injured, while Lolium perenne, L. multiflorum, Cynosurus cristatus and Anthoxanthum odoratum were most injured at -10°C.

Peake (1964) subjected crowns of hardened grasses to -23°C while holding roots at -7°C, a characteristic winter soil temperature in Alberta. Agropyron cristatum and Phleum pratense were most resistant, Festuca rubra was moderately resistant and Lolium perenne had poor resistance to freezing. Beard (1966) found that field-hardened plants of Toronto, Cohansey and

Washington Agrostis palustris (vegetatively propagated) suffered no serious injury at -23°C; Seaside, Penncross and Congressional cultivars of the same species (seeded) showed serious injury at -20.5°C. Astoria, A. tenuis, was severely damaged at -15°C. Poa trivialis showed no serious injury at -20.5°C while the Merion cultivar (Poa pratensis) was hardier than common and Newport which showed severe killing below -18°C. Poa annua was badly damaged at -15°C. Pennlawn (Festuca rubra), showed severe damage at -15°C. These hardiness levels were obtained in early December, but by late January serious injury occurred 3-4.5°C higher.

Lorenzetti et al. (1971) found that of 19 acclimated cultivars and ecotypes of Lolium perenne, those from northern Europe, were more cold tolerant than those from the Mediterranean when frozen at -8°C. However, winter-hardiness in the field and cold tolerance in the controlled environment was not highly correlated. The indigenous cultivars S23 and S24 were more winter-hardy in Wales than foreign accessions, even though the latter came from colder climates and showed greater cold tolerance when grown in a controlled environment.

Adachi et al. (1976) in northern Japan noted that of 116 cultivars of Lolium perenne and Lolium spp. the best field survival was in those of northern origin from Canada, Finland, Sweden and Norway. The lethal temperature for field-hardened, artificially frozen entries lay between -13 and -16°C. Canadian and Finnish cultivars were more cold tolerant than those from Denmark, Great Britain and the Netherlands.

Larin (1962) in the USSR classified Poa pratensis, Phleum pratense and Agrostis alba as having good frost resistance, Festuca rubra as moderately frost resistant and Festuca pratensis and Lolium perenne as having little or no frost resistance.

Oullet's hardiness index for forage grasses in Canada (1976) reflects the resistance of species and cultivars to low temperatures with freezing tolerance as the main factor in survival, particularly in the Prairies. In the latter regions Agropyron cristatum had the highest survival index; Poa pratensis, Phleum pratense and Festuca rubra showed high indices while Festuca pratensis was low (Table 1).

In New Zealand, Ritchie (1973) ranked Festuca rubra L. spp. commutata, Holcus lanatus L. and Agrostis tenuis Sibth. as the most persistent at altitudes of 1250 and 1430 m where winter injury was concerned. Cossens (1977) reported a reduction of the Lolium perenne in a mixed sward from 50-30% following a severe winter at high altitudes. Short clipping before the first heavy snow was associated with survival in this species compared with almost complete kill on longer, unclipped grass.

Gusta et al. (1980) determined the LT50 (0°C) for several cultivars of 9 species of field hardened perennial grasses collected in mid-February. Of the turfgrasses, 3 cultivars of Agrostis palustris tolerated -35°C, 7 cultivars of Poa pratensis tolerated -30 to -21°C, 2 cultivars of F. rubra subsp. rubra

-24°C, Festuca longifolia -21°C and 11 cultivars of Lolium perenne -15 to -5°C. Some hardiness was lost between mid-January and the time of the test in mid-February. By 11 June only -5 to -7°C of crown hardiness remained in the P. pratensis. After 3 weeks of dehardening at 20°C by day and 17°C by night they could only tolerate -3°C. These findings are in general agreement with those given by Beard (1973). Lolium perenne crowns had the highest water content of the species tested and this was the least cold-hardy species, but there was no association between water content and freezing resistance in the 11 cultivars. Poa pratensis cultivars were more frost-hardy than L. perenne and generally had lower crown moisture contents, but there was no definite association between the water content and cultivar resistance (Gusta et al., 1980).

In Britain, turfgrass cultivars of L. perenne were rated into three broad categories from good to poor. Most cultivars were considered only of medium winter-hardiness (STRI, 1980). It was found that ratings for winter-hardiness in cultivars of this species at Aberdeen, in northeastern Scotland after the severe winter of 1976/77 agreed reasonably well with those from the National Institute of Agricultural Botany at Cambridge (NIAB, 1978) and from the Netherlands (RIVRO, 1980). Manhattan and Sprinter cultivars were the most winter-hardy (Laycock & Shildrick, 1979). In Poa pratensis at Aberdeen, four cultivars showing the least melting-out disease (Drechslera poae (Baudys) Shoem.) in 1976, Enmundi, Nugget, Parade and Sydsport were four of the five best for winter survival in winter 1976/77. This correlation did not apply to the cultivar Bensun, the most winter-hardy or to Birka with the highest resistance to D. poae melting-out which lost 93% ground cover to winterkill. Prato, Monopoly and Baronie suffered more severe damage than Birka (Laycock, 1980). The results obtained did not agree with those for some of these cultivars tested in Pennsylvania and Finland (NE-57 Tech. Comm., 1977; Rainenko & Laurila, 1975).

Most warm-season turfgrasses have low frost tolerance, even at maximum cold-hardiness.

Table 1. Relative Cold-hardiness of cool-season turfgrasses

Species	Relative winter-hardiness	Reference
Agropyron cristatum	High	Peake, 1964; Oullet, 1976
A. riparium	High	Knowles, 1961
Agrostis tenuis Sibth.	High to moderate	Carroll, 1943; Beard, 1966; Ritchie, 1973

A. palustris Huds.	Very high to high	Beard, 1966; Gusta et al., 1980
A. stolonifera L.	High	Larin, 1962
Anthoxanthum odoratum L.	Low	Carroll, 1943
Cynosurus cristatus L.	Low	Carroll, 1943
Elymus junceus Fisch.	High	Knowles, 1961
Festuca rubra L.	Moderate	Peake, 1964
F. rubra ssp. rubra L.	High to moderate	Knowles, 1961; Beard, 1966; Gusta et al., 1980
F. rubra ssp. commutata Gaud.	High to moderate	Carroll, 1942; Knowles, 1961; Ritchie, 1973
F. longifolia Thuill	High	Knowles, 1961, Gusta et al., 1980
F. pratensis Huds.	Low	Larin, 1962; Ritchie, 1973
Lolium perenne L.	Moderate to low	Carroll, 1943; Larin, 1962; Peake, 1964; Lorenzetti et al., 1971; Adachi et al., 1976; NIAB, 1978; STRI, 1980; Gusta et al., 1980
Holcus lanatus L.	Moderate	Ritchie, 1973
Poa annua L.	Low	Beard, 1966
P. compressa L.	Moderate	Knowles, 1961
P. pratensis L.	Very high to moderate	Carroll, 1943; Knowles, 1961; Beard, 1966; Oullet, 1976; Gusta et al., 1980
P. trivialis L.	Very high	Beard, 1966; Gusta et al., 1980
P. nemoralis L.	High	Carroll, 1943

s. Winter-hardiness of dryland turf

The need for low-maintenance, drought-resistant turf is apparent for climatic areas such as the plains of western North America when irrigation water is not available or its economic use not justifiable. A lower turf quality than on irrigated lawns is acceptable for school- and farm-yards, playing-fields, memorial gardens and cemeteries, road-sides and banks of irrigation ditches. A good cover and low maintenance is attainable.

Drought- and frost-hardiness are broadly related and plant response to drought and low temperatures appear integrated. When plants become drought-hardy because of reduced water supply they usually become more frost-hardy and vice versa (Vasil'yev, 1961). It is often difficult to determine the effects of drought and low temperature in a particular situation.

Grass species found acceptable in practice for dryland turf in the Prairies of North America include: Fairway crested wheatgrass Agropyron cristatum (L.) Gaertn. (Kirk, 1932); Russian wild rye, Elymus junceus Fisch. (Heinrichs & Lawrence, 1958); sheep's fescue, Festuca ovina L. (Morrison et al., 1957); and streambank wheatgrass, Agropyron riparium Scribn. & Smith (Douglas & Ensign, 1954).

Knowles (1961) compared several species and cultivars of turf and forage grasses for resistance to winterkilling, wear and turf quality between 1951 and 1960 at Saskatoon in the Canadian Prairies. He came to the following conclusions. Crested wheatgrass and Russian wild rye were highly resistant to drought and frost, were quick to establish and wore well. Some strains of sheep's fescue and hard fescue, Festuca longifolia Thuill, were almost as drought- and frost-hardy as crested wheatgrass and Russian wild rye, but were slower to establish. Streambank wheatgrass and native western wheatgrass, Agropyron smithii Rydb., were inferior in quality to the crested wheatgrass, Russian wild rye, sheep's and hard fescues. Creeping red fescue, Festuca rubra L. spp. rubra, suffered considerable winterkilling but not to the same extent as Chewing's fescue, F. rubra L. spp. commutata. Canada bluegrass, Poa compressa L., and the Merion cv. of Poa pratensis L., were both susceptible to winter injury, much more than common Kentucky bluegrass. The relative winter-hardiness of cool-season turfgrasses is summarized in Table 1.

t. Winter damage to turf caused by geese and meadow voles

During periods of autumn migration geese may graze the short grass of golfcourse fairways and parks adjacent to open water. While their foot traffic probably causes little injury at this time, their droppings which are high in nitrogenous compounds (Kear, 1963; Marriott, 1973) have a phytotoxic affect and intensify low-temperature injury.

In the Prairies of Canada, meadow voles (Microtus

pennsylvanicus Ord.) may cause severe localized damage to turfgrass of different categories in winter (Smith, 1979a). These small rodents make runs under the snow and eat crowns, stolons or rhizomes of different grass species, usually rejecting grass blades and upper parts of shoots. Plants are sometimes killed or turf to the sides of runs are mulched with discarded leaves which encourages snow mold. The debris is unsightly when exposed at snow melt. Tell-tale droppings may be found in the runs.

u. Practical diagnosis of the causes of winter injury

The determination of the cause of physiological winter disease may be more difficult than where snow molds are involved. It is not often that complete reliance can be placed on plant symptoms alone. It is advisable to have local weather and plant data on such as:

Temperature of air and surface soil.
Date of development and thickness of a snow cover.
Autumn soil moisture content and state of the ground when a snow cover developed.
Duration of ice covers and their thickness.
Duration of winter thaws and state of the ground after thaw.
Duration of exposure of turf after thaw and wind conditions.
Species and cultivar concerned.
Autumn management.

Often much of this information will be available only in very general terms, but it may provide valuable clues for the investigator to pursue.

Snow molds should first be eliminated as the cause of the injury (see Snow Mold Diseases). The first indication of their activity at snow melt is the occurrence of mycelium and then the appearance of associated, discrete patches of killed or damaged foliage bearing fungal structures such as sclerotia or spores. Damage associated with heavy snow drifts is often likely to be fungal in origin.

"Winter-burn" of old or senescing leaves of grasses is common in early winter before the development of a snow cover, but may also occur in spring. This "tipping" and the later "browning-off" which results from low-temperature killing and desiccation of older leaf tissues, must be regarded as the normal onset of near-dormancy. Crown tissues are usually little affected.

On turf areas which are free from snow or have been inadequately covered, frost injury should be suspected if air temperatures are known to have fallen rapidly to lower than the lethal temperatures for the hardened species and cultivar (see Relative Winter-hardiness). This damage is often associated with

freezing and thawing of an ice cover in late winter or early spring when plants have lost cold-hardiness. Then they may be killed at higher temperatures. Affected plants will turn limp and go brown in large patches. Examine plants for injury to lower crowns. Leaves of plants killed by freezing in early winter and then covered by snow may appear green at snow melt, but after thawing they go limp and brown.

Desiccation injury occurs when a snow cover is lacking and plants are exposed to winds which dry them out. Desiccation may take place at sub-zero temperatures when the soil is frozen or at higher temperatures when the soil moisture is inadequate. Raised, exposed turf is more likely to suffer desiccation injury than low-lying, sheltered areas. Leaves killed by desiccation are usually bleached. Patches of injured grasses may be localized or general. In the case of low-temperature desiccation the soil is often quite moist after thawing, but in above-zero situations the soil is usually dry.

Although Poa pratensis and Agrostis stolonifera, which are important turfgrasses in northern regions, will tolerate complete freezing in ice for 15-60 days respectively, at -4°C when fully hardened, Poa annua, which is also a very common turfgrass will not. When they have lost hardiness in spring, grasses are more susceptible when frozen in ice. Under natural conditions temperatures are not as stable and uniform as those in controlled environments and this makes it uncertain if ice injury per se is a major cause of damage in turfgrasses. In the unstable winter weather found in the northern parts of western Europe or in northeastern and northwestern North America with alternating periods of rain, sleet, snow, freezing and thawing, probably much of the injury ascribed to ice covers is caused by direct freezing injury of hydrated crowns when the ice cover melts. In some years, when an ice cover develops in winter and persists until spring, as in the "isbrand" of northern and coastal region of Norway (Sjoseth, 1959) and in eastern Ontario and western Quebec (Edey, 1973) grass death is probably due to ice suffocation. Dead plants will be found only in areas which were covered by the ice sheets.

Frost-heaving in seedling turfgrasses may be noted particularly, in regions with unstable winter climates in late sowings on high organic or fine-textured, poorly-drained soils. Seedlings may be lifted right out of the soil and mature plants raised and their roots, stolons or rhizomes torn. Mature turf may heave under similar climatic and soil conditions leaving an uneven surface. In the shallow depressions direct-freezing injury may occur in spring.

Traffic injury is usually easy to identify since it takes the outline of feet, skis, snowmobile tracks or wheels. Traffic patterns developing before frosts are usually less distinct than

where frozen grass blades are broken or crushed by pressure. Damage to plants is usually proportional to the traffic intensity. Damage may occur with or without a snow cover. While deep snow may reduce crushing injury, when compacted the snow loses much of its insulating properties.

v. Management of physiological winter diseases

i. General

1. Select species and cultivars capable of adequate hardening for winter survival. Use a cultivar bred in and/or adapted to the region. Within a species, cultivars may show considerable variability.

2. When establishing new turf sow early enough to allow the plants to mature so that they will fully acclimate before the onset of severe winter weather. Plants should have reached the 4th-leaf stage at the minimum by that time. Where frost-heaving is a problem, the better the root development the less damage the plants are likely to sustain.

3. Practices which delay the onset and progress of the grass plant towards dormancy reduce the attainment of full potential for cold-acclimation (and also resistance to snow molds). This is particularly the case with late-season or excessive N fertilizer usage. Vigorous growth of roots and shoots should not have been encouraged by cultural practices during the growing season. Diseases and insect pests should have been controlled well before winter because weakened plants are less frost-hardy. Adequate supplies of K, P and minor nutrients are necessary for vigorous growth and timely tissue maturation, essential for cold-hardiness.

4. Management practices which improve turf surface conditions such as dethatching and vertical mowing and those which improve surface drainage such as pricking, coring or aerifying are likely to be of benefit in getting rid of surface water from rain or melting snow. Although coring may be done late in the season in milder or snowy regions, where turf may be unprotected by snow, desiccation injury is frequently seen on turf round unfilled core holes.

5. Raise height of cut gradually in autumn to provide better temperature protection for grass crowns, but keep on mowing until near-dormancy is reached. Pick up clippings as these may mulch the turf unevenly and slow up drying of the surface and favour snow molds.

6. The growing medium for the turf should be free-draining; high organic matter and a high proportion of fine soil particles (clay

and silt) should be avoided.

7. In site construction make slopes or rises as gentle as possible especially in regions where desiccation injury is known to be a problem since steep slopes make effective watering difficult. Build greens and lawns with a slight slope or crown and pay particular attention to levelling to avoid ponding and permit free drainage. Install drains or cut temporary channels to dispose of dammed-up thaw water at spring run-off.

8. Provide logically placed, rather than landscaped roads and walkways to lessen winter traffic injury on turfed areas to which the public has access. Designate and sign areas for ski and motor toboggan use.

ii. Particular problems

Desiccation injury: The grass species most commonly affected is Poa annua, which frequently invades turf when the sown species, i.e. Agrostis stolonifera, Poa pratensis, Festuca rubra or Lolium perenne, have been weakened or killed out by disease, insects, summer drought, chemical injury etc. P. annua regenerates freely from seed lying in the base of the sward. Considerable control of the effects of desiccation can be achieved indirectly by excluding P. annua and maintaining an adequate, seasonally-controlled growth of the desired cultivar. In particular, the prevention of snow mold injury in bentgrasses, which are particularly susceptible constitutes a main line of defence against invasion by P. annua.

Mulching the turf lightly and evenly with screened topdressing, similar in composition to the root zone soil[1] covering with 6 mil. polyethylene sheets which should be clear, not black (Evans, 1975) pinned with large wire staples, screen cloth (Ledeboer & Skogley, 1967), or special winter protection blankets are suggested for light or moderate snowfall areas only where desiccation risk in late winter or early spring is great. When or where snowfall is heavy, unperforated polyethylene sheets increase smothering risk. Temporary snow traps formed from regular snow fence, tree branches and twigs laid on the

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[1]Or with lumpy, unscreened, well-rotted animal manure, applied when the turf has gone quiescent and removed with snow pushers just before grass growth starts in spring. This has been very successful on some golf greens in Saskatchewan in minimizing desiccation injury (pers. comm., Mr. D. Campbell, Riverside Golf & Country Club, Saskatoon).

turf, or permanent wind breaks of shrubs or trees may be used to reduce wind velocity and drying rate. These will also drop snow on the turf and in years when persistent snow drifts develop or in snowy regions this will increase the risk of snow mold injury. Where covers or snow traps are used so should snow mold fungicides (see Snow Molds). Polyethylene covers should be removed as soon as possible in spring to prevent the turfgrass being forced under the highly humid "greenhouse" conditions which develop under them.

Turf should be adequately supplied with water in the autumn, particularly on light soils. Frequent shallow wetting should be shunned in favour of infrequent but deep watering.

Low-temperature killing: Only the most cold-hardy cultivars of species such as Agropyron cristatum, Agrostis stolonifera, Poa pratensis, Poa trivialis, Festuca ovina, F. duriuscula or F. rubra should be used where low-temperature killing is a severe problem unless reliance is to be placed in electrical soil-heating. Then less cold-hardy species such as Lolium perenne and Phleum spp. may be used. Cultivars of northern origin may be expected to survive better than those from further south.

An adequate nutrient supply and correct nutrient balance between the major nutrients are particularly important for the full development of cold-hardiness. Generally, high K and P to N ratios increase frost-hardiness and a high N ratio almost always decreases it. Where the use of K is necessary (Canadian prairie soils have abundant K), fertilization in a ratio of 2 or 3 to 1 N to K should be aimed at, towards the end of the growing season for cool-season grasses in severe climates. For temperate, oceanic climates, where appreciable growth takes place in winter, this may be increased to 3 or 4 to 1. In the warm-season grasses, in southerly regions, where risk of freezing injury is low, the N to P and K ratio usually has little effect on cold injury.

Because a high crown water content predisposes grasses to freezing injury, free surface and soil drainage of snow melt water should be facilitated, especially in spring, and irrigation in autumn should be adequate to take care of possible desiccation, but not so excessive so that the turf goes into winter waterlogged.

Fresh snow provides excellent insulation over turf and a layer 20-25 cm deep may be encouraged by ventilated snow fencing or brush placed on the turf. As the snow compacts its insulating properties decline and at the end of the winter may be quite low at the same time as the hardiness of the plants has begun to decline also. While it is doubtful whether turfgrasses are often killed by suffocation or smothering under even very deep snow in the Prairies, if there is inadequate drainage the melting of a heavy snow blanket encouraged by trapping may increase hydration of grass crowns and provide the necessary conditions for freezing

injury. Special insulating blankets which are available provide reasonable thermal protection for small valuable turf areas such as golfgreens, but straw and other organic mulches are rarely practicable. Cereal straw compacts under snow and then loses much of its insulating properties. This material and others which are applied in bulk are difficult to keep in place and remove after winter.

Sub-surface soil warming by means of electrical resistance wires, warm-air ducts and warm-water pipes has been employed effectively to keep sports turf of various categories frost-free and playable during winter months. Soil warming also protects against low-temperature injury and permits the use of less cold-hardy grasses, i.e. Lolium perenne where Poa pratensis would normally have been used. Temperatures of the soil must be carefully adjusted so that they do not favour the development of particular snow molds (see under LTB Snow Mold).

Ice injury: Where ice injury is a problem resistant species should be used. Some Agrostis stolonifera cultivars are resistant to long-duration encasement. Poa pratensis and Phleum pratense are moderately resistant, Lolium perenne and Festuca pratensis are fairly susceptible and Poa annua quite susceptible. Select the most frost-hardy cultivars and improve the drainage of the site.

On shaded turf snow and frost may persist in spring longer than in unshaded. It may be necessary to remove snow from such locations with a snow blower. Where ice sheets form these should be dusted with dark-coloured top dressing to encourage quicker melting.

Frost-heaving: Frost-heaving is a particular problem in late-sown turfgrass which has not had time to develop a root mass before winter, especially on fine-textured soils, those with high organic matter or inadequately drained. Similar soil conditions favour heaving in mature turf. Management practices that improve vigor of rooting and facilitate drainage in spring will reduce risk of injury from heaving. Where it is necessary to make late sowings the insulation of the turf surface with brushwood, peat, compost or snow trapped by snow fences (Russell, 1961) may prevent heaving. Heaved seedlings or turf should be lightly rolled. Raising the height of cut in autumn allows the development of a heavier plant cover in spring which reduces heaving by giving a better insulation (Andersen, 1960).

Fig. 1. Ice/water injury on a golf green of *Agrostis/Poa annua*. Fig. 2. Desiccation injury — differential reactions of *Agrostis* strains on a sand-base green. Fig. 4. Severe frost-heaving on an established *Agrostis* turf. Fig. 5. Traffic injury after snow melt — *Poa pratensis* turf.

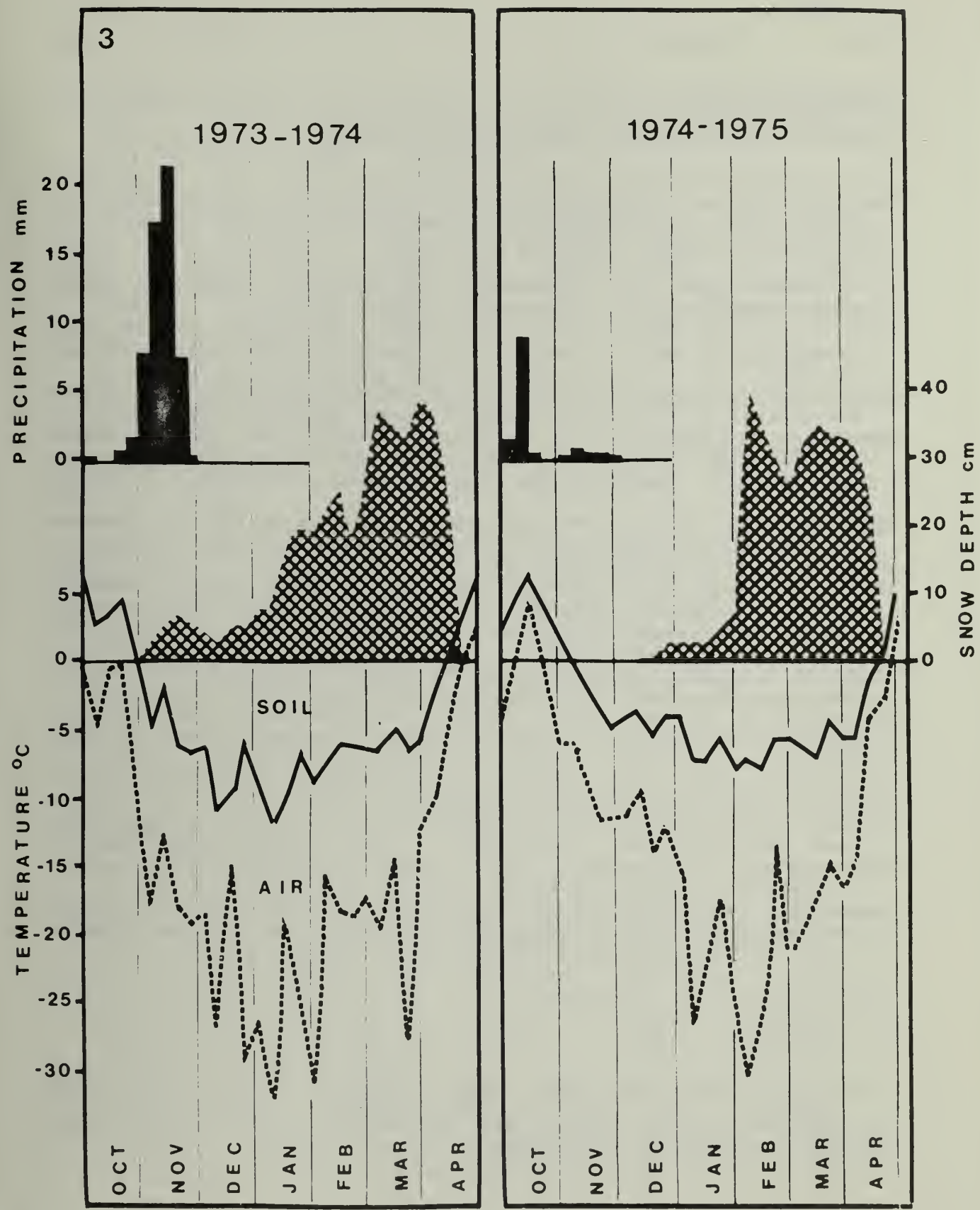

Fig. 3. A comparison of meteorological data in a "snow mold year" (1973–1974) and a "non-snow mold year" (1974–1975) at Saskatoon.

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FUSARIUM OR MICRODOCHIUM PATCH AND PINK SNOW MOLD

Fusarium patch describes the disease as it develops in turf in the absence of a permanent snow cover and pink snow mold as it appears at the end of the winter after snow melt.

Fusarium patch disease is probably the most common, disfiguring, and damaging disease of golf and bowling green turf in the colder seasons of the year in many parts of western Europe and Scandinavia (Bennett, 1933, 1933a; Bjorklund, 1971; Bourgoin et al., 1974; Courtillot, 1976; De Leeuw & Voss, 1970; Gram, 1929; Gray, 1963; Hermann, 1982; O'Rourke, 1975; Sampson, 1931; Schoevers, 1937; Skirde, 1970; Smith, 1953, 1959, 1965, 1974, 1975a, 1978; Welling & Jensen, 1970; Ylimaki, 1972). It is also commonly found on the coarser types of turf of soccer fields and cricket outfields (Smith, 1965).

Attacks weaken and may kill turfgrass and permit invasion by weeds. In cool-temperate oceanic climates such as in the British Isles, coastal regions of the Eastern and Northwestern States of the USA, in British Columbia and New Zealand, fusarium patch disease is important and in some regions it may develop at any time of the year during cool, wet weather (Conners, 1938; Foster, 1949; Latch, 1973; Smith, 1953, 1957). However, when the summers are drier, fusarium patch is most prevalent in autumn, winter and spring when the seasonal decline of grass growth occurs (Smith, 1965) as in New South Wales (Anon, 1967; Siviour, 1975) and Victoria (Jones, 1961; Mebalds & Kellock, 1983) in Australia. It may develop in places where there is never any snow (USGA, 1957). Fusarium patch is probably more common than has been reported in autumn and spring in regions where it is usual to regard the pathogen as causing a snow mold disease. Meiners (1958) in the Pacific Northwest of the USA and Smith (1974) in the Canadian Prairies have noted autumn outbreaks subsequent to cold, rainy weather, sleet showers or temporary light snow covers developing on unfrozen ground. Since there is little grass growth at this time, recovery from these attacks is slow and under the permanent, winter snow cover, snow mold develops.

"Snow mould" (English) or "mold" (North American), "sneskimmel" (Danish), "snomugg" (Norwegian), "snomogel" (Swedish), "sneeschimmel" (German), "snaesveppur (Icelandic) and "pourriture des neiges" or "moisissure des neiges" (French) are general terms describing symptoms of disease which appear on plants after snow melt. In older or uncritical European or North American literature they have been applied to disease caused by the attacks of complexes of several low-temperature tolerant pathogens including Microdochium nivale. The latter causes "pink snow mold" which is so described because the fungus on the leaves takes on a pink coloration on exposure to light. In cool-temperate continental climates and at high elevations in warm-temperate ones across North America pink snow mold is often an important overwintering disease of turfgrasses (Broadfoot,

1936; Dahl, 1934; Fushtey, 1975; Howard et al., 1951; Lebeau, 1968; Madison et al., 1966; Meiners, 1955; Monteith & Dahl, 1932; Platford et al., 1972; Smith, 1974, 1978; Stienstra, 1974; Vaartnou & Elliott, 1969; Wernham, 1941). The disease may also be important on grasses generally in regions with cool or cold continental climates in Europe (Jamalainen, 1959, 1974; Skirde, 1970; Ylimaki, 1972) and northern Japan (Maki, 1976; Tomiyama, 1959; Yoshikawa, 1969). However, Woess (1972) considered it less important than rust in the pannonic region in which Vienna is situated (western Hungary, northern Yugoslavia, eastern Austria).

Symptoms - fusarium patch

The disease first appears in turf as small patches, roughly circular and 2.5-5 cm in diameter, at first water-soaked and then yellow to orange-brown. Around the margins of these patches of dying and dead grass shoots and leaves there may be seen in moist conditions, a faint fringe of white or pale pink mycelium which tends to mat together the aerial plant parts. Patches may increase in diameter to approximately 25 cm with irregular margins and adjacent patches may coalesce. Very early symptoms are illustrated in Fig. 6 and late fall symptoms in Fig. 7. The fungus may penetrate as far as the crowns of the plants, but complete killing in North America may result from subsequent winter injury or from the activity of secondary invaders.

Patches may fill in gradually from surviving plants when the disease has passed over. This recovery from damage may be slow in winter or spring when leaf production is low. In some cases a patch may show concentric-ring symptoms with fungal attack proceeding actively at the periphery and the green recovering centre with a ring of dead leaves between. If attacks occur before a moderate snowfall, and/or short duration snow cover, on snow melt, patches may have a bleached appearance with little increase in diameter having taken place under the snow. Under prolonged, thick snow cover existing patches increase in size, numerous latent infections develop and abundant aerial mycelium may be produced. This is not as common in the British Isles as in North America or continental Europe.

Symptoms - pink snow mold

As the winter snow cover melts, exposed patches appear bleached and are often covered with abundant, white mycelium which may mat together the leaf blades. The patches may gradually turn pink because of colour change of the mycelium and the development of pink sporodochia of the fungus. The margins of the patches, especially on Poa annua turf may take on an orange-brown tone with bleached centres (Fig. 8) under moist conditions because of the continued activity of the pathogen. Recovery may be slow if the weather is dry in the spring.

The causal fungus

The most recent taxonomic classification for the anamorph of the fungus is Microdochium nivale (Fries) var. nivale Samuels & Hallett (1983) with Monographella nivalis (Schaffnit) E. Muller (1977) as the teleomorph.

Lanosa nivalis (Fries, 1849) is the oldest name for the conidial state. Other synonyms include: Fusarium nivale (Fries) Sorauer and F. nivale Cesati ex Berlese & Voglino.

There is a variety with multiseptate conidia as the main distinguishing feature: Microdochium nivale (Fries) Samuels & Hallett var. majus Wollenweber with the synonym Gerlachia nivalis (Cesati ex Berlese & Voglino) W. Gams var. majus (Wollenweber) W. Gams & E. Muller (1980) (as Gerlachia nivale (Ces. ex Sacc.) W. Gams & E. Muller var. and major (Wr.) comb. nov.).

For discussions on the taxonomy and synonomy see Samuels & Hallett (1983) and Boerema & Verhoeven (1977).

There is considerable variation in morphology in isolates of M. nivale whether obtained from grasses or cereals (Smith, 1957, 1965). Grass isolates are pathogenic towards cereals and vice versa (Smith, 1957, 1983). Although Booth (1971) reported that there was little evidence for physiologic specialization there were distinct strains differing in minimum temperature for growth. This contradicts Bennett's finding (1933) that significant morphological and physiological differences between British and Continental strains were accompanied by differences in pathogenicity. Bennett (loc. cit.) also showed that the Continental strain was more virulent towards cereals and less virulent towards grasses than the British strain. The teleomorph has never been found on turfgrasses (Smith, 1965, 1983) and the only record on grasses is on Glycera fluitans (L.) and Phragmites communis Trin. R. Br. from the Isle of Rhum in the Hebrides (Dennis, 1964). Isolates from cereals form perithecia readily in culture on cereal straws (Booth, 1971a) and these fruiting structures have been reported immersed in the leaf sheaths on stems of cereals in many countries (Cook & Bruehl, 1966; Dobrozrakova, 1929; Noble & Montgomerie, 1956; Wollenweber & Reinking, 1935), although not in Canada (Gordon, 1952). Sampson (1931) found that none of her isolates of M. nivale from turfgrasses ever produced the teleomorph, but she was prepared to accept that it was the anamorph of Calonectria graminicola[1].

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[1]Bennett (1933) reports correspondence from Dr. H. W. Wollenweber in which the latter states that only 10% of the isolations of M. nivale will produce perithecia, but that forms in which this happens will produce the teleomorph on many kinds of substrate.

Smith (1983) showed that none of 50 isolates of M. nivale (mostly from turfgrasses) produced perithecia or perithecial initials while 14 of 24 isolates[1] from cereals did so in culture on wheat straws. It seems questionable whether the fungus from cereals is the same as that from grasses. Perhaps the strains of M. nivale which have developed on turfgrasses have lost the ability to produce the perfect state in the absence of suitable natural substrates, viz. the bases of mature flowering stems, which are not characteristic of mown turf.

On grass showing fusarium patch symptoms, aerial mycelium is white, cobwebby, occasionally knotted and often quite sparse, stretching from leaf to leaf at the patch borders. In the case of pink snow mold, on emergence from the snow the mycelium is white, but is often more felted, matting together the leaf blades, gradually changing to a pink colour. Sporodochia may be found on leaves and suspended in mycelium under moist conditions. On turf grasses from British sources 1-septate macroconidia were most common, and aseptate, 2-septate and 3-septate forms were quite common also. Four- and five-septate forms are less common. One- septate spores measured 8.0-18.0 µm X 1.8-3.0 µm (most 12.5-15 µm X 2.0-2.75 µm) (Smith, 1953). Three-septate conidia measured 19-30 um X 3.5-5.0 µm (Booth, 1971). Sprague (1950) gave the dimensions for 0-septate as 8-12 X 2.0-2.8 µm, 1-septate as 13-18 X 2.4-3.0 µm, 3-septate as 19-27 X 2.8-3.8 µm and 4- to 7- septate as 19-30 X 2.5-4.0 µm. Most turfgrass isolates from Saskatchewan had 0- or 1-septate conidia (Smith, 1983). Bennett (1933) noted that conidia of a Continental strain grown on the same media as a British strain were considerably longer and wider. There are no microconidia or chlamydospores. Conidia (Fig. 9) are curved, slightly broader in the lower half, narrowing upwards to a slightly curved, sharp apex. They are heelless or with a minute heel.

Light is necessary for the production of sporodochia but the duration of exposure to light necessary to initiate this is quite brief, i.e. for the period of opening and closing an incubator door. An oat-extract starch medium proved very suitable for spore production; on this medium very little aerial mycelium was produced. Spore production was enhanced by incubating cultures in light from a tungsten filament bulb at 33 cm distance for 8 h per day (Smith, 1953). Nirenberg (1981) recommends the use of an agar medium with low nutrient content and growth under continuous "black light" (nuv) at 17°C to encourage formation of sporodochia. Sanderson (1970) has examined in detail the effect of light on sporulation of M. nivale in culture. On semi-synthetic medium with yeast extract (Smith, 1957) isolates from turf produce abundant mycelium, which may be loose or dense, white or assuming a faint salmon tint in good light. Some isolates remain mycelial. In other cases pink or salmon-pink, salmon-orange and finally rufous spore masses are produced on the surface of the medium in good light. On potato-dextrose agar in diffuse light the mycelium may be white, rose pink or salmon pink

(Sprague, 1950). Nirenberg (1981) has described a simplified method for identifying Fusarium spp. occurring on wheat.

In cereals, perithecia develop in the leaf sheaths covering the lower portion of the culm. They are oval or flattened oval, papillate, appearing as black dots, sometimes abundant, 150-260 um across and up to 300 um high with plectenchymatous walls 10-30 um thick, gold to dark brown. Asci are parallel-walled or spindle-shaped, straight or curved with a thin wall and an amyloid apical spore-discharge ring. Ascospores are hyaline, 2- to 4-celled, with usually 8 to the ascus. They measure 50-70 X 7-9 um (Muller, 1977).

Perithecia of the teleomorph, Monographella nivalis, were induced in isolates from cereals, in culture or on wheat straw at 20°C (Booth, 1973; Smith, 1983) although they did not develop in turfgrass isolates (Smith, 1983).

Isolation of the fungus and pathogenicity tests

This fungus may be isolated from mycelium produced on infected leaves by incubation in cool (4-10°C), moist chambers or from spores on sporodochia or from suspensions of spores from "pink" leaves. If isolates are then grown on potato-dextrose or potato-sucrose agar at 13-17°C in 12 h nuv light, pale to bright orange sporodochia will usually develop. For pathogenicity tests on turf, cultures of the isolate in potato-dextrose broth are macerated briefly in a blender and applied with an atomiser. The inoculated turf is placed in a moist chamber with wet cellulose wadding or newspaper and polyethylene sheeting and incubated in the dark at 1-2°C for 6-8 weeks. For individual plants the technique described by Smith (1981a) may be used. For field inoculation, the fungus grown on sterile rye grain is suitable. The cultures must not be allowed to stale. After they have been air dried they are broadcast over the turf in autumn. The inoculated turf may then be covered using wet hessian (burlap) until the disease develops (Smith, 1953).

Susceptibility of grass species and cultivars

Agrostis spp. - bentgrasses. Dahl (1934) in Wisconsin reported that the creeping bentgrasses Columbia, Washington and Seaside were susceptible, but Metropolitan was resistant as was browntop bentgrass. He also commented that the variation in wild bentgrasses was from resistant to susceptible. On the other hand, Tyson (1936) in Michigan found that of the creeping bentgrasses Seaside was susceptible, but Washington and Metropolitan were resistant. Of the browntop bentgrasses, Astoria was resistant and Rhode Island and Prince Edward Island susceptible although less so than German mixed bentgrass. For Madison et al. (1960) Seaside creeping bentgrass was much more susceptible than Highland browntop. Howard et al. (1951) considered that browntop and the creeping bentgrasses Washington

and Metropolitan more resistant than Seaside. Smith (1958, 1965) and Jackson (1962) observed the resistance of common species and cultivars of turfgrasses in Britain to fusarium patch disease from 1951 to 1962. New Zealand, Highland, Steinacher and Holfior browntop bentgrasses, Barenza and Brabantia brown and velvet bentgrasses, Compacta and Z103 creeping bentgrasses were very susceptible to susceptible. Wild strains of creeping bentgrass varied from resistant to susceptible. Jackson (1964) found that the velvet bentgrasses, Mommersteeg's, Barenza, Brabantia and Opal and brown bentgrasses, were all susceptible in the early establishment period and this allowed invasion by P. annua. Skirde (1970) reported that Highland browntop and creeping bentgrasses were severely damaged by M. nivale at Giessen in West Germany in the 'snow mold' winter of 1969/70. Gray & Copeman (1975) found that both browntop and creeping bentgrasses on short, sheep-grazed, natural grasslands (of low-fertility) in the North of Scotland were severely damaged by M. nivale. Goss et al. (1974) in western Washington noted that browntop bentgrasses Astoria, Bardot, Exeter, Highland, Holfior and New Zealand were susceptible. Velvet bentgrasses Kingstown and Novobent were moderately susceptible as were creeping bentgrasses, Seaside, Arlington, Congressional and Old Orchard; Cohansey and Toronto creeping bentgrasses were susceptible. Emerald, A74, A75, Nimisila, Northland, Waukanda and Yale creeping bentgrasses were resistant. Gould et al. (1978) tested 160 lines of bentgrasses for resistance to snow mold injury and for turf quality at Puyallup in western Washington and 138 of the most promising entries of these in eastern Washington. They found that in general, varieties or selections from northern climates had the greatest resistance to M. nivale and that the stolonized bents were more resistant to this pathogen than the seeded sorts. A. tenuis lines were less resistant that those of A. canina or A. palustris.

Smith (1980) found that the A. palustris Huds. (= A. stolonifera L.) and A. canina L. entries were considerably more resistant to pink snow mold than those of A. tenuis. There is general agreement in North America that Penncross is still one of the most resistant cultivars of A. palustris currently available, but there are several promising newer cultivars showing differential resistance (Fig. 10). In Denmark, Thuesen (1975) found that Tracenta browntop was resistant and Bardot moderately so while Kromi and Prominent creeping bents were moderately resistant to fusarium patch disease. Taylor (1971-1978) reported fusarium patch scores from many cultivars of bentgrass established vegetatively (stolonized) or from seed in the coastal region of British Columbia. The variation in ratings obtained for the same cultivar from year to year even, in one location, illustrate the difficulties in evaluating resistance to fusarium patch when general disease incidence is low. However, in late autumn in 1975 and again in 1976 bentgrasses showed up to 25% of turf area infected with fusarium patch. In the sown cultivars,

browntop bentgrasses Barbinet, Highland, Tracenta, Contrast, Enbenta, Orbica and AT-4 showed much disease; Enate, Boral, New Zealand, Exeter, Bardot and Astoria were moderately affected and Ligrette little affected. In the creeping bentgrasses (A. palustris), Penncross, EWS-2, Prominent, Emerald and Seaside were only slightly diseased, but OE-0332 was moderately damaged. None of the velvet bentgrasses (A. canina) Novobent, Kingstown, Agrettina or Rusta showed more than slight spotting. However, the amount of damage from fusarium patch in late autumn 1976 increased during winter until by mid-February 1977, all browntop and velvet bentgrasses cultivars showed heavy to moderate infections. Of the creeping bentgrasses, Penncross still showed slight infection and EWS-2, Emerald and Prominent were only moderately infected. At this time the vegetative creeping bentgrasses Arlington, Keen 53, Keen 36, Toronto and Huffine HCC-7-2 showed very heavy patching but Nimisila, Huffine, MCC-3 and Smith-732 showed only slight infection. Smith (1980) found Penncross, Kingstown, S-4979, Emerald and Seaside (creeping and velvet bentgrasses) less susceptible than the STRI (1978) browntop cultivars Boral, Bore, Varmland, Colonial, Bardot, Exeter, Highland and Astoria to pink snow mold in Saskatchewan. Data, mainly from European sources, indicates that the browntop bentgrasses, except Highland, showed generally good resistance to fusarium patch while creeping bentgrasses were fairly good and velvet bentgrasses good except for Aca 61. In the Netherlands, RIVRO (1978, 1980) lists Bardot, Tracenta, Contrast, Mamelou, Holfior and Enbenta browntop bentgrasses as moderately resistant; Prominent creeping bentgrass and Barbella velvet bentgrass are considered as highly and fairly resistant to fusarium patch, respectively.

Fine-leaved fescues. There is less information on the resistance of fine-leaved fescues in turf than for bentgrasses; they are not used as extensively in the finest turf as the latter and usually not in monostands which probably reduces their liability to infection. Jamalainen (1951, 1955) considered Festuca rubra as one of the more resistant species to M. nivale in Finland. Gray & Copeman (1975) noted that F. rubra was not infected in sheep-grazed, semi-natural grassland in upland pastures in the North of Scotland. Dawson and Wilton slender creeping red fescues were moderately resistant and S59 slightly resistant while Puma and Barfalla chewings fescues were slightly resistant to fusarium patch in Denmark (Thuesen, 1975): Biljart hard fescue was slightly resistant. Skirde (1970) reported that fine-leaved fescues were heavily attacked and F. rubra was attacked moderately severely in West Germany. In the Netherlands, F. rubra was considered as in the least severely attacked category (de Leeuw & Voss, 1970). In Sweden, Jonsson & Nilsson (1973) compared the resistance of 12 cultivars of F. rubra to artificial infection in the laboratory and their resistance in the field to artificial and natural infections.

The correlation between the two ratings was poor, perhaps because of differences in pathogenicities in the fungus in the natural attack and artificial inoculations. In the natural attack, taking the average of two seasons, Polar, Dawson and Golfrood were moderately, and Oasis and Pennlawn of the slender creeping red fescues, slightly damaged; Reptans, Illahee, Boreal and Novorubra of the strong creeping red fescues were also only slightly damaged. S59 and Dawson slender were resistant, New Zealand chewings red fescue moderately resistant, and Cumberland slender, susceptible to fusarium patch in Britain (Smith, 1965).

Perennial ryegrass (Lolium perenne L.). Most cultivars are resistant or moderately resistant to fusarium patch disease (Smith, 1965) and reaction to this specific pathogen is not usually recorded (RIVRO, 1980; STRI, 1978; Taylor, 1971-1978) although resistance to snow mold generally may be. However, resistance to M. nivale is likely to break down under heavy snow covers, such as occurred in Britain in 1963 (Gray & Copeman, 1975; Jackson, 1963; Jamalainen, 1974). Pasture types, some of which are used in amenity turf, suffered considerable damage from M. nivale especially where high rates of N fertilizer had been applied late in the season in pasture tests in northern Scotland (Gray & Copeman, 1975). The Finnish cultivar Valinge (Jamalainen, 1951) was considered resistant to M. nivale, as were Ejer, Pleno, Barlenna, S.23, Lamora, Barenza and Vigor in Denmark (Thuesen, 1975).

Annual bluegrass - Annual meadowgrass (Poa annua L.). This species is very susceptible to fusarium patch disease and is often killed out completely by the disease in late winter.

Smooth-stalked meadowgrass - Kentucky bluegrass (Poa pratensis L.). Most cultivars are resistant or moderately resistant to fusarium patch disease (Jamalainen, 1955, 1974; Smith, 1965) and if snow mold develops under persistent snow covers crowns are not usually severely damaged and plants recover; this is characteristic of the response of this species to slight to moderate attacks of the common snow molds (q.v.). Vargas et al. (1972) reported severe infection (greater than 50%) of snow mold caused by M. nivale on Arista, WK-411, Park, Cougar, Primo and NJE P-111 and lesser amounts on two other sorts. Thuessen (1975) reported that Fylking had some resistance to M. nivale.

Other species. Crested dogstail (Cynosurus cristatus L.) is sometimes slightly damaged by M. nivale when grown in a monostand, timothy (Phleum pratense) is not usually damaged by fusarium patch or snow mold (Jamalainen, 1954; Smith, 1965), but Poa trivialis is susceptible.

Epidemiology

Source of inoculum for disease outbreaks. M. nivale has rarely been isolated from soil directly, but it can be cultured from plants growing in it. Although the pathogen commonly occurs in cereal crops in Britain, Rawlinson & Colhoun (1969) failed to isolate it from soil on Warcup plates of pentachloronitrobenzene agar. Gordon (1954) did not recover it from soil samples when examining 1,674 soil samples from cereal plots in Canada, yet the pathogen is quite common on both turfgrasses and cereals in Canada (Smith, 1978). However, it has been isolated from old grassland soils in both North America and Britain (McElroy et al., 1954; Nicholls, 1956). The fungus is seedborne on cereals in Britain and Europe (Booth & Taylor, 1976, 1976a; Colhoun, 1969; Noble & Richardson, 1968; Rawlinson & Colhoun, 1969) and on Lolium perenne (Matthews, 1971; Richardson, 1979). In North America and Finland the fungus is regarded as a soil-borne pathogen (Bruehl et al., 1966; Jamalainen, 1962; Sprague, 1950). In Britain it has been shown in greenhouse and plot experiments with cereals and grasses to be soil-borne (Hewett, 1983; Holmes, 1979; Rawlinson & Colhoun, 1969). M. nivale survived within naturally infected or artificially infected cereal straw for up to a year (Bruehl & Lai, 1966; Sanderson, 1967), but was not recovered from the soil (Snyder & Nash, 1968). M. nivale is capable of spreading through non- sterile soil from agar discs, seeds and artificially infected straw (Booth & Taylor, 1976a). Dahl (1934) used infected grass clippings for plant inoculation. Smith applied inoculum of a pathogenic turfgrass isolate of the fungus grown on a sterile, wheatmeal/ground dried grass/sand mixture (1957) or on sterile rye grain (1976) to infect large field plots of fine turfgrass for fungicide studies in late autumn. In the British tests (1957) the turf was then covered with moistened sackcloth to encourage fungal growth and to increase susceptibility by reducing carbohydrate reserves in the grass leaves. This was not necessary in Canadian tests (1976). The mycelium of the pathogen grew out from the pieces of inoculum, spread from leaf to leaf and produced characteristic patches of the disease. Spore suspensions of M. nivale applied to the soil around L. perenne seedlings were not effective in causing disease, but mycelial macerates were (Holmes & Channon, 1975). Mycelial macerates of isolates from oats, wheat and turfgrasses were infective towards Poa annua (Smith, 1957) and spore suspensions of isolates from turfgrasses, rye and wheat were infective on rye leaves (Smith, 1981a). Leaf spot symptoms which appear on winter wheat after snow melt in spring are typical of discrete infections initiated by spores rather than from mycelium (Smith, 1975; Sprague, 1950). Although direct evidence is lacking it appears likely that under snow, most infection of grass seedlings and mature turf is from soil-borne mycelium. Splash dispersal of conidia by raindrops or in water films, mechanical turf operations such as brushing, scarifying,

aerating or mowing, and pedestrian traffic could carry the sticky conidia or mycelium in leaf or soil fragments. These are all possible methods of spread of the disease when snow cover is lacking. Dahl (1933) has described the infection process by mycelium. In turfgrass, wind dispersal by ascospores may be neglected if perithecia are shown to be absent. Bennett (1933) suggested that in turf the fungus persists saprophytically, since there are no chlamydospores or sclerotia, as dark brown aggregates of mycelium in plant residues. Recent studies by Petrini et al. (1979) suggest that M. nivale is usually a harmless endophyte and only becomes pathogenic under a snow cover lasting for several weeks. However, as outbreaks may occur at any time of the year in climates like those of the British Isles (Smith, 1965), and where there is no snow (Arsvoll, 1973), it is probable that sub- clinical infections of leaves and crowns may always be present. In some cases the fungus will develop in apparently healthy turf if it is shaded and kept moist and cool (Smith, 1965).

Conidia from an aqueous suspension dried on glass rods germinated normally after 6 weeks storage in the laboratory. Germination vigor declined after 3 months when the spores were stored in full light or in the dark at room temperature, refrigerated at 4°C or exposed to full light outside the laboratory during spring and summer. After 3 months vigor was highest when spores were stored in the dark at room temperature (Smith, 1953).

The effect of temperature. Although M. nivale is a mesophilic fungus with an optimum temperature for growth in culture of 18-20°C and a maximum of 30-32.5°C, (Bennett, 1933; Broadfoot, 1938; Dahl, 1934; Ekstrand, 1955; Endo, 1963; Smith, 1953; Tasugi, 1935), some isolates will also grow at temperatures below 0°C (Arsvoll, 1975, 1980a; Ekstrand, 1955; Smith, 1953). It should not be labelled as cold-loving or psychrophilic, but rather cold-tolerant (Bennett, 1933). This wide range of temperature over which it is active is probably the main factor responsible for its wide geographical range as a pathogen on members of the Gramineae in all the continents other than Antarctica (CMI Map 432: 1967). Isolates differ in their minimum temperature for growth and the rate at which they will grow at low temperatures (Booth, 1971a). Bennett (1933) found that a British isolate showed a mean increase in colony diameter at 0 and 1°C of mm and a Continental isolate from Schaffnit of 1.25 mm in 24 h. Smith (1953) compared a British isolate and one from Zurich, Switzerland, both from diseased turf, on artificial culture media under a snow cover at 0 to +1°C. The British isolate showed a mean increase of 0.86 mm and the Swiss one 1.25 mm in 24 h. The Continental isolate produced more abundant aerial mycelium under the snow than the British one and also grew more rapidly at 21°C than the British one. Mycelium can withstand temperatures as low as -20°C and remain viable

(Bennett, 1933).

Dahl (1934) found that the most rapid and severe infection on turfgrasses and cereals in moist chambers took place at 0-5°C, while at 15-20°C infection was slight and slow. Results obtained by Endo (1963) on Seaside bentgrass (Agrostis palustris L.) grown in sterile quartz sand without nutrient were in apparent contradiction to Dahl's (1934). In 2 weeks a trace of infection was found at 21°C and in 3 weeks, 10, 45 and 10% infection at 15.5, 21 and 27°C, respectively. There was no disease at 32°C. Sparse sporodochia formed at 21°C and abundantly at 15.5 and 10°C. These results reflect the artificial conditions of culture of the bentgrass rather than simulating the pathogen/host relationship in the field where lower temperatures restrict the growth of the grasses, increasing their susceptibility more than they reduce the pathogenicity of the fungus, making the disease more obvious. Favourable natural meteorological conditions for the fungus are those of a moist (British) summer with shade temperatures of 12-13°C average minimum and 18-19°C average maximum. Couch (1962) in the USA suggests a temperature range from 0-7.2°C for optimum disease development during periods of high humidity, with economically important outbreaks to 18.3°C. This latter is probably too high for British conditions. Bruehl & Cunfer (1971) found that M. nivale caused no disease on winter wheat at -1.5°C. Lebeau (1964), who regulated soil temperature by means of buried heating cables, found that M. nivale became the dominant snow mold pathogen when the minimum soil temperature was raised above 3°C. Severe pink snow mold sometimes develops on turf influenced by the warmer temperature adjacent to buildings (Fig. 11) or over heating ducts. Continuous cold weather is not necessarily a meteorological characteristic for regions where M. nivale causes fusarium patch damage to turfgrasses, but rather, short intermittent periods. Holmes & Channon (1975) have shown that several short intermittent periods at 0°C arrested the recovery of L. perenne seedlings inoculated with M. nivale and caused further root damage. In autumn, winter and early spring attacks on aerial parts of the turf grasses frequently follow sharp frosts which check grass growth or damage aerial tissues (Smith, 1965).

Effect of moisture and snow cover. The effect of moisture and snow cover on attacks of M. nivale can only be stated in general terms. Wet weather, even persistent drizzle, especially if temperatures are low, temporary snow cover in autumn, sleet showers, thick fogs, heavy dews during clear weather, especially in spring and autumn, predispose turf to attacks of M. nivale and typical fusarium patch symptoms may develop. Bennett (1933) considered that M. nivale is favoured by the moist weather conditions prevailing in summer and that the most numerous attacks occurred from May onwards. However, from late spring through summer infections are masked to a greater or lesser extent by the vigorous leaf and shoot production in most cool

season grasses while autumn and winter infections are not masked as grass growth is then slower. Patches of diseased grass are more noticeable during autumn, winter and early spring (Smith, 1965).

Environmental and management factors may lead to the creation or maintenance of moisture in the upper layers of the turf. Inadequate soil drainage, too much shelter, which restricts airflow, often referred to as "air drainage", mulches of tree leaves in autumn and the development of a mat or thatch of fibre, grass left too long, covering with straw to keep out frost, or with tarpaulins and turf covers to keep off rain increases humidity and provides favourable conditions for attacks to develop or become more severe. Dew may persist in shaded areas if not dispersed by switching or hose-dragging and this favours spore germination and fungal growth.

In a study on the effect of irrigation on turf of A. castellana Highland, in California (Madison et al., 1960), the number of patches on plots which received morning irrigation was higher than those which received afternoon water. When plots were watered in the morning, daily irrigation was associated with greater disease incidence than was irrigation once every 7 days. On A. palustris L. Seaside turf, irrigation practice had very little effect on disease incidence. Since the irrigation terminated several months before the disease appeared it could be that watering influenced the disease incidence by changing the environment of the fungus in the soil during the summer when soil temperatures are too high for disease development. Under British conditions it is usual practice to complete watering in summer in time for the turf surface to dry off so that cool, moist conditions favourable for infection will be presented for the shortest possible time.

A deep snow cover developing on unfrozen turf early in winter provides favourable environmental conditions for the growth of cold-tolerant fungal pathogens, including M. nivale (Fig. 3). Although the snow cover protects the grass from frost injury it also maintains a humid, stable microclimate favourable for mycelial growth of the fungus. The longer the cover persists the longer are the conditions likely to be favourable for fungal growth, substrate colonization and plant invasion. During the time the grass is covered by snow its carbohydrate reserves are being depleted. Under a deep snow cover there is insufficient light for photosynthesis, yet respiration continues at measurable rates at temperatures as low as $-30^{\circ}C$ (Meyer & Anderson, 1952; Scholander et al., 1953). This drains carbohydrate reserves leading to lowering of both snow mold resistance and winter hardiness (Kneen & Blish, 1941; Tomiyama, 1955). The depth of the snow cover, whether the ground was covered before it froze, and the subsequent air temperature determines what the temperature at the turf surface will be (Bruehl et al., 1966; Ylimaki, 1962 and Fig. 3). It is possible to determine only a broad, general relationship between the duration of snow

cover and and incidence of different snow mold diseases, as Arsvoll (1973) has done for Norwegian grasslands, including meadows, pastures and amenity turf, the latter of Agrostis tenuis, Festuca rubra and Poa spp. Over three seasons, between 1968 and 1971, 1,410 fields out of 2,401 surveyed showed damage by M. nivale. Slight attacks were often noted after 30-day snow covers and but also at localities with no snow cover at all. After 90 days of snow cover there was little increase in frequency of attacks. In the Prairies of Canada pink snow mold on turfgrasses assumes epidemic proportions only under several months of snow cover which melts to produce very wet conditions in spring (Lebeau, 1968), particularly on turf against buildings with heated basements, and over buried heating systems (Fig. 9). There is often a pre-hibernal history of fusarium patch disease. The pathogen also occurs in disease complexes with other snow molds.

Effect of pH, lime applications and soil texture. Schaffnit and Meyer-Hermann (1930) found that the optimum pH for growth of their strain of the fungus was 7.9 and that it did not grow in acid soils. Bennett (1933) found that M. nivale in culture would grow throughout the range of pH 2.5-13.0 with an optimum of pH 6.6-6.9. Conidia did not germinate below pH 5.0. He also found that the British strain of the fungus would persist and attack cereals in all normal field soils of acid to alkaline reaction. Bennett (1933) suggested that the reason why the Continental isolate of M. nivale was unable to grow in acid soils was that its presence and pathogenicity might be suppressed by the presence of antagonistic organisms. The disease occurs in Britain on acid to alkaline turf but its incidence is more common where alkaline amendments are used, i.e. of bone meal, steamed bone flour, sodium nitrate, Nitrochalk, basic slag and urea-formaldehyde (Escritt & Legg, 1969) or following the application of lime. Smith (1958a) showed in a field experiment over three seasons that the amount of disease which appeared following the liming of very susceptible turf of Poa annua was related to the amount of lime applied and to the pH of the top 2.5 cm of turf. The higher the pH (test range 4.3-7.2) the more severe was the infection. However, slight disease occurred at the lowest pH where no lime was used. This relationship between soil pH and fusarium patch is not a simple one because major plant nutrients may modify the soil reaction (Brauen et al., 1975; Goss, 1967), and also influence plant response to the pathogen through nutrition. They may also directly influence the pathogen and its antagonists in the sole of the turf before it establishes a relationship with the host plant. This appears to be the case with the LTB snow mold (q.v.).

Fusarium patch disease occurs on all soils from sands to clays, but since heavy soils tend to be less well-drained and more fertile they also favour outbreaks. Attacks on poor soils take longer for recovery because of lack of nutrient and

inadequate water supply in drier regions. However, since microbial activities take place mainly in the mat or thatch, the character of this probably influences progress of the disease more than the texture of the soil below.

Effect of nutrition. Bennett (1933a) noted that fertile bowling greens were more severely affected by M. nivale than those of low fertility. Applications of ammonium sulphate with activated sewage sludge made in late autumn were shown by Dahl (1934) to predispose putting green turf to pink snow mold attack. A 5-cm cover of straw in combination with the fertilizer more than doubled its severity. It was reported as practical experience in Britain that the worst attacks of fusarium patch disease followed very intensive management of turf which produced a succulent growth which had little resistance to the disease. The most important factor in producing this type of growth was the application of fertilizers high in soluble nitrogen and 'unbalanced' with respect to potassium and phosphorus. Even 'balanced' fertilizers applied at a sufficiently high rate at the wrong time of the year would encourage this type of growth (Dawson & Gregg, 1936). Smith (1957) showed that late autumn application of ammonium sulphate to turf of Poa annua influenced the amount of fusarium patch disease appearing in late winter. Increases in rate of this fertilizer led to increases in the amount of disease. Gould (1965) concurred with the latter finding from experience in western Washington. The form in which nitrogen is applied as well as the rate influences the severity of attacks by M. nivale. Tyson (1936) in Michigan found that snow mold was more severe on plots treated with organic nitrogenous fertilizers, such as cottonseed meal, dried blood or Milorganite than on those given ammonium sulphate, urea or sodium nitrate. The least damage occurred on a plot treated with calcium nitrate. Madison et al. (1960), in a test on irrigated turf in California, found that on 'Highland' bentgrass (A. castellana) increasing the rate of urea formaldehyde fertilizer did not significantly increase the amount of disease, but that applications of the latter fertilizer resulted in more disease than with urea at the same rate. On Seaside bentgrass (A. palustris); however, the increased incidence of the disease was a function of increasing nitrogen level not significantly affected by previous irrigation history or nitrogen source. Escritt and Lidgate (1965) reported that urea formaldehyde behaved like organic fertilizers in producing a soft, disease-susceptible turf and that the amount of disease increased as the nitrogen level was raised. Powell et al. (1967) found that bentgrass and tall fescue turf in Virginia, fertilized with up to 5 kg/100 m^2 of N in the form of ammonium sulphate or slow-release urea granular material from October to January was not affected by snow mold. Handoll (1966) found that the addition of other nitrogenous fertilizers to ammonium sulphate increased turf susceptibility to fusarium patch compared with the

latter alone. Summer applications of sodium nitrate or nitro-chalk gave the greatest increase in susceptibility. Goss (1968) showed that the disease decreased with increasing rate of 0-3.3 kg of K per 100 m^2 when N levels were not greater than 6 kg per 100 m^2 per season. Increasing N to 10 kg per 100 m^2 per season increased the disease sharply and there was little effect from K. Goss & Gould (1968) found that the numbers of spots of the disease on Agrostis tenuis Colonial were reduced in number as N applications, in the form of urea, were reduced from 10 kg to 3 kg per 100 m^2 per season without regard to the P and K applications made with the N. When N rates were at 3 and 6 kg less disease occurred when P was applied. The addition of K in the absence of P seemed to increase the number of disease spots at 3 and 6 kg of N. The greatest amount of disease was when K was absent, N at 10 kg and P at 0.9 kg per 100 m^2. This agrees with the practical experience in Britain reported by Dawson & Gregg (1936). Studies by Brauen et al. (1975) on A. tenuis cv. Astoria turf showed that N at 2.9 and 5.9 kg per 100 m^2 significantly increased disease over no N and that an increase to 9.8 kg when no sulphur was applied reduced it to 8.2% but plant vigor and turf density were lowered because of stress induced by no sulphur. No fusarium patch was noted on plots treated with sulphur at 2.2 kg per 100 m^2. Although P tended to reduce the amount of disease, the reduction was not significant. The highest levels of N and S are considered too high for British conditions. Interactions between N, P and K were similar to those previously obtained (Goss & Gould, 1968).

In Finland, Nissinen (1970) found that N reduced resistance to winter attacks of M. nivale in L. perenne while K increased it. Survival of plants given Ca but no NPK was relatively poor on soil of pH 4.7, but good at pH 6.5. Mn, Cu, and S appeared to increase resistance.

Cultural control of fusarium patch disease

1. Moisture control. Operations which will assist the rapid removal of surplus water, such as drainage, pricking, spiking or removal of surface mat or thatch, scarifying, vertical mowing and switching or hose-dragging to disperse the dew assist in the prevention of the disease. Screening or shading by hedges, rows of trees, fences or herbaceous borders which prevent "air drainage" should be reduced. Turf should not be smothered with heavy top dressing or left uncut in late autumn or winter. It is also important to rake off mulches of fallen leaves for fusarium patch will often start under the humid conditions they provide.

2. Maintenance of turf vigor and fertility control. Adequate drainage and aeration, regular mowing and other surface operations, suitable fertilizers and top dressings are all required to maintain good turf. If the turfgrasses are vigorous, the turf will stand up to usage, recover from damage, withstand

disease and so retain a good appearance. Although the nitrogen, phosphate, and potassium status of turf must be adequate to maintain grass vigor, judicious nitrogenous fertilization is the key to the control of disease caused by M. nivale.

Fine turf in particular, is predisposed to attack by M. nivale if heavy applications of quick-acting nitrogenous fertilizers such as sodium nitrate, urea, ammonium nitrate or dried blood are made in autumn. Care must be taken in the use of the slower-acting nitrogenous fertilizers (such as hoof and horn meal, ground leather or casein waste) for heavy applications of these may favour the disease. Synthetic slow-release fertilizers and the slower-acting organics applied before a dry summer may not break down until moist conditions return in autumn and produce a flush of susceptible growth at this time. Frequent light (split) applications of quick-acting nitrogenous fertilizer from spring to summer enable a more precise control of growth to be effected. The level of nitrogenous fertilization adopted will depend on several factors such as climate, management intensity and species employed. Where irrigation is used extensively, as in drier regions of North America, a higher nutritional plane is employed than in the British Isles on more "natural" amenity turf. If it should be necessary to apply a quick-acting nitrogenous fertilizer late in the year in a mild winter climate where grasses remain actively growing or for some special purpose, appropriate fungicide applications should be made to prevent the appearance of the disease.

On the coarser types of turf of soccer pitches, later applications of nitrogenous fertilizer are necessary to maintain plant vigor and colour in late autumn, winter and early spring, the seasons during which much play takes place in western Europe and the west coast of Canada. This may result in some unseasonable growth, but as these types of turf often contain the less susceptible Lolium perenne and Poa pratensis the risk of the disease developing may be much less than where finer and generally more susceptible turfgrasses are concerned.

There is abundant evidence that some of the organic fertilizers (in particular dried blood) make the turf more susceptible than do fertilizers supplying equivalent amounts of nitrogen in inorganic form. The cause of this is uncertain. The use of such materials should be avoided, particularly in autumn. If annual applications of nitrogen at up to 6 kg/100 m^2 are made, K applied at up to 3.3 kg/100 m^2 is likely to reduce susceptibility, but the balancing effect of the latter is likely to be overridden if N is increased excessively (Goss, 1968; Goss & Gould, 1968). A similar effect may be expected with P which will reduce susceptibility at lower N levels. Use balanced fertilizers according to indications of soil analyses.

Applications of lime are likely to encourage the disease but the use of sulphur, ammonium sulphate or other acidifying materials discourage it. It is difficult to determine whether this is because pH changes in the sole of the turf alter the

balance of pathogen and microbial antagonists or because of nutrient effects, since sulphur and calcium are also essential nutrients. Following liming a fungicide effective against M. nivale should be used.

3. Use of resistant species and cultivars. The resistance of grass species and cultivars varies according to region, level of fertility and management practice. Local experience must govern their selection (Smith, 1980). Poa annua is the most susceptible species and a major effort should be made to exclude it from turf by chemical seedbed treatment and later by suitable management and fungicidal treatment of the selected species to prevent takeover of killed-out patches. Of the bentgrasses, A. tenuis cultivars are probably the most susceptible, but their liability to disease is reduced by using them in polystands with less susceptible species such as F. rubra. Although stolonized types of A. canina and A. palustris are generally less susceptible than seeded forms they are not as extensively used as the latter. Other grass species are generally less susceptible than the bentgrasses and the latter should be used only where the finest turf is needed. There are several cultivars of Lolium perenne and Poa pratensis (RIVRO, 1980; STRI, 1978, 1980) which will withstand close mowing and are wear-tolerant. These may be used successfully in medium fine turf of lawns, golf tees, field hockey and soccer pitches.

Chemical control

On turf known to be liable to attack by the fungus, preventive applications of fungicides should be made before the disease appears. It is much easier to prevent the disease appearing than to clear it up once patches have developed. Patches take a long time to recover during the winter and they provide a source of inoculum for the spread of the disease. From local records it should be known whether particular turf areas are liable to attack, and if so, when the disease is most likely to appear. The number of applications and dates of these vary from region to region, but where autumn attacks are usual the first application should be in late summer or early autumn followed by at least one more in late autumn or early winter before the development of a permanent snow cover. In mild, snow-free climates, winter and spring applications may be needed. Surrounds to playing areas should be included, especially the aprons and collars of golf greens which are often neglected. These surrounds may be colonized first with Poa annua because of weakening attacks of the disease and this species then spreads to the greens.

The most reliable and persistent fungicides (and among the most expensive) against fusarium patch and pink snow mold are those based on mixtures of mercurous and mercuric chlorides.

They are still permitted for professional use in some countries, but have been withdrawn from registration in others because of environmental considerations. If used as dry powders, the equivalent of approximately 46 g/100 m^2 metallic mercury may be given per applications. Up to four applications of this type of material may be needed in autumn, winter and early spring to prevent fusarium patch disease on susceptible turf in a mild winter climate. On sites where attacks occur only occasionally mercurous/mercuric chloride mixtures may be used in a curative fashion at increased dosage (up to 90 g/100 m^2 metallic mercury) to check an attack as soon as it is seen. Overall application and not just 'spot treatment' should be carried out because initial spots may not be obvious and may be missed. Wettable powder formulations of mercurous/mercuric chloride mixtures are also available, including those with the mercurous chloride in "microfine" form (Smith, 1957b). Effective control of M. nivale is possible with these at lower mercury dosage than with dry powders. Organo-mercury fungicides are also effective in controlling infections before the development of permanent winter snow covers, but are usually not so persistent. Of these phenyl mercury acetate sometimes shows phytotoxicity at effective dosages. There are often good substitutes for mercury fungicides for the control of M. nivale, notably quintozene, chlorothalonil, anilazine, mancozeb, maneb, benomyl, thiophanate-methyl, benzimidazole, cadmium compounds, dichlorophen, iprodione, triadimefon, which may be registered for usage in some countries. Chloroneb, although intended for the control of Typhula spp. and other basidiomycetes, sometimes controls M. nivalis. It is good practice to ring the changes with these fungicides, especially if systemic materials are included in a program and several applications are made during the season, to reduce the chance of resistance to a particular fungicide developing. Since M. nivale often occurs in complexes with other snow molds alternative materials should be effective against other components of the complex.

In North America, fungicides based on mixtures of mercurous and mercuric chlorides have been the standard materials for the control of snow mold for many years (Boyce, 1932; Broadfoot, 1936; Meiners, 1955; Monteith & Dahl, 1932). Howard et al. (1951) reported, that against pink snow mold, applications of mercurous/mercuric chloride mixtures at 200-400 g/100 m^2 before snowfall and again during a mid-winter thaw appeared to be the most satisfactory treatment for the disease. Meiners (1955), in experiments on the control of snow mold due to either or both M. nivale and Typhula incarnata, found that phenyl mercury fungicides gave the greatest reduction in the incidence of the disease. These were followed, in order of decreasing control effectiveness, by a mercurous/mercuric chloride mixture and cadmium succinate. A fungicide based on thiram and another containing chlorothalonil were not effective when M. nivale was the dominant organism. Gould (1957) found that a fungicide based

on phenyl mercury acetate, applied every 2 weeks in autumn, was much more effective against fusarium patch than if applied at 3- to 4- week intervals. He also found that mercurous/mercuric chloride fungicides gave effective control of the disease. In further work Gould et al. (1961) showed that organic mercurials gave faster and better control of this disease than inorganic mercurials, cadmium compounds and thiram. However, mercurous/mercuric chloride mixtures have remained the standard by which the effectiveness of other snow mold fungicides is judged in some jurisdictions in Canada and the United States where their professional use on turfgrasses is still permitted (in 1983) (Gould, 1965; Gould et al., 1977; Jackson, 1983; Smith, 1976).

In British practice, inorganic mercury fungicides applied as powders, usually bulked with a carrier, were in use on turf in the 1920's. Bennett (1933a) introduced malachite green (with Bordeaux mixture as a sticker) which was effective against fusarium patch but required repeated applications in wet weather and incurred the risk of turf toxicity from the build up of soil copper. Effective control was also obtained with a commercial fungicide based on phenyl mercury acetate. Until 1953, these were the only fungicides in use against fusarium patch in Britain (Smith, 1953). Griseofulvin (Smith, 1956) cadmium (Jackson, 1959; Smith, 1953, 1957, 1957a) but now banned in some countries, and quintozene (Jackson, 1961, 1962) were also shown to be effective. Quintozene (PCNB) at 140 g a.i./100 m^2 was found effective as a protectant and/or eradicant. The persistency of this fungicide under a snow cover was noted (Jackson, 1962). Anilazine was found effective as a preventive (Halcrow, 1965) and eradicant (Handoll, 1966) of fusarium patch at 125 g a.i./100 m^2. Systemic fungicides such as benomyl, thiophanate-methyl and thiobendazole at 90, 90 and 60 g a.i./100 m^2, respectively (Woolhouse, 1971), proved effective and increased the range of alternatives to the contact-type materials and gave longer protection than anilazine, chlorothalonil and quintozene, which are of the latter type (Woolhouse, 1972). Thiobendazole was effective in controlling fusarium patch disease on Poa annua turf at 15 and 25 g/100 m^2 in Sydney, Australia (Siviour, 1975). In Continental Europe, in particular in Scandinavia, the effectiveness of quintozene and some organo-mercurials was shown in the control of overwintering diseases of grasses, among other crops, caused by several different low-temperature tolerant fungi including M. nivale (Jamalainen, 1960). Ylimaki (1972) reported that the resistance of lawns to overwintering fungi and winter damage could be improved by the application of quintozene as a spray or powder at 4-5 kg/ha. In the Austrian Tyrol (Kock, 1976), where both M. nivalis and Typhula spp. were concerned (Dr. L. Kock, pers. comm. 27 Feb. 1981), the order of fungicide effectiveness was anilazine, benomyl, chlorothalonil, quintozene, mancozeb, chlorothalonil, benomyl and thiobendazole. Dithianol, thiophanate-methyl and methoxyethyl mercury silicate were

ineffective in controlling disease and improving turf colour. In Sweden, Bjorklund (1971) found that there were no clear differences in effectiveness between quintozene (125 g a.i.), maneb (160 g a.i.), mancozeb (106 g a.i.) per 100 m^2, respectively, and two experimental materials in the control of M. nivale. Six applications at 2-week intervals reduced infection to almost zero.

In Ontario, Canada, benomyl at 30 g a.i. gave more effective control than at 60 g a.i. in one test, but this was reversed in another where there was more disease. Chloroneb, which is not usually regarded as effective against M. nivale, was as effective as benomyl when applied at 182 g a.i./100 m^2 (Fushtey, 1975). Chloroneb and benomyl, in combination and at lower dosage than when applied separately, gave effective control. Chlorothalonil, at the highest dosage of 265 g a.i./100 m^2, was effective in one test but not in another. Five experimental materials were ineffective. In Saskatchewan, Canada, a heavy autumn attack of fusarium patch was prevented by benomyl (100 g a.i.), dichlorophen (100 g a.i.), thiophanate-methyl (98 g a.i.), quintozene (152 g a.i.) per 100 m^2 and by two experimental systemic fungicides (Smith, 1976). The most effective control of fusarium snow mold was obtained with a wettable powder formulation of mercurous/mercuric chloride (33 g Hg) followed by phenyl mercury acetate (7 g a.i.), chloroneb (97 g a.i.) and quintozene (146 g a.i.) per 100 m^2. Gould et al. (1977), in tests in eastern Washington and Idaho, found that combinations of the following fungicides gave the best control of M. nivale (often in complex with Typhula incarnata).

	Early autumn	Late autumn	Spring
1.	Benomyl - (60*)	chlorothalonil (175)	benomyl (60)
2.	Benomyl - (60)	chlorothalonil (90)	benomyl (60)
3.	Benomyl - (60)	chlorothalonil (90) +chloroneb (145)	benomyl (60)
4.	Mancozeb - (200)	mancozeb (200) +chloroneb (145)	mancozeb (200)

* grams active ingredient/100 m^2

Most treatments gave effective control of M. nivale. Gould et al. (1977) emphasized the need for an early autumn application of fungicide to control M. nivale and found that the substitution of a late heavy application did not compensate for an early light treatment. Smith (1974) reported that in the prairies of western Canada where the fungal spectrum of snow mold complexes is different from that in Washington, effective control of M. nivale in snowy regions is probably dependent on fungicidal prevention of the disease which develops before the permanent snow cover comes. The control of M. nivale may be expected with systemic fungicides such as benomyl, or thiophanate-methyl provided that repeated applications are made during autumn, winter and spring. Contact fungicides such as cadmium compounds,

quintozene, chloroneb, chlorothalonil and mercurials are also likely to be effective. Where only one pre-niveal application of fungicide is possible, inorganic mercury chlorides, phenyl mercury acetate, quintozene, and chloroneb have given good control on lawns and golf greens. Residual effectiveness, persisting into the autumn of the second season in some tests on turf of A. stolonifera, was shown by benomyl (30 g a.i./100 m^2) and iprodione (50 g a.i./100 m^2) but there was significantly more on quintozene-treated plots than all other treatments. Residues of quintozene may suppress other fungi favouring M. nivale (Smith & Reiter, 1975). Current fungicide control recommendations by Jackson (1983) for fusarium patch in Rhode Island are thiophanate-methyl or benomyl at 30-60 g a.i. and quintozene at 125-175 g a.i./100 m^2. Tolerance to iprodione has been reported of M. nivale by Chastagner & Vassey (1982).

Taylor (1974, 1975, pers. comm. 16 Apr. 1979) in British Columbia found that single early-fall applications of benomyl or thiophanate-methyl were not effective in controlling either early or late-autumn attacks of M. nivale in the interior of British Columbia. Additional late autumn applications of other materials such as mercurous/mercuric chlorides, quintozene and chloroneb were needed to secure adequate overwintering control. Fertilizer containing granular quintozene gave as effective control as spray applications at the same fungicide dosage. The disease was less effectively controlled on the Old Shaugnessy cultivar than on Penncross Agrostis stolonifera turf with the same dosage of fungicide.

In Saskatchewan, M. nivale frequently occurs in complex with the LTB phase of Coprinus psychromorbidus. Smith and Mortensen (1981) tested fungicides against both pathogens singly, and in combination of three materials. In the combinations, benomyl, which is often used for late summer application against M. nivale, was applied first. When M. nivale was dominant, but when the LTB was present, combinations of three materials were generally more effective than three applications of one of the materials alone in controlling the disease complexes. The exceptions were thiophanate-methyl and triadimefon. There was almost as much disease on plots given three applications of benomyl (26%) as on untreated controls (31%). These results illustrate the complications which have been noted when combinations of fungicides with different spectra of activity are applied to complexes of snow molds.

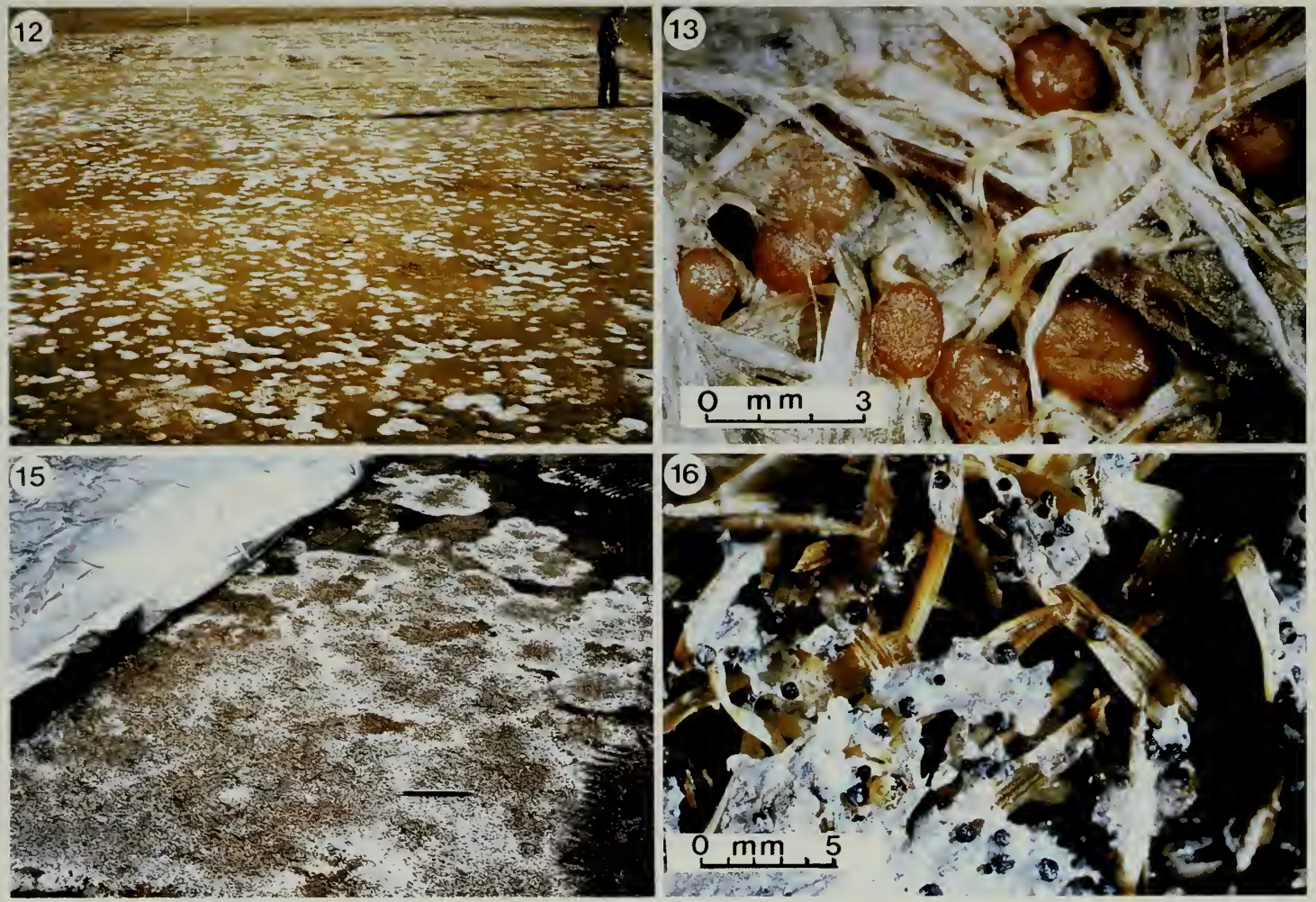

Fig. 12. *Typhula incarnata* snow mold on a bentgrass green in northern British Columbia. Fig. 13. Sclerotia of *T. incarnata*. Fig. 15. Mycelium on disease patches caused by *T. ishikariensis* var. *canadensis*. Fig. 16. Mycelium and sclerotia of *T. ishikariensis* var. *canadensis*.

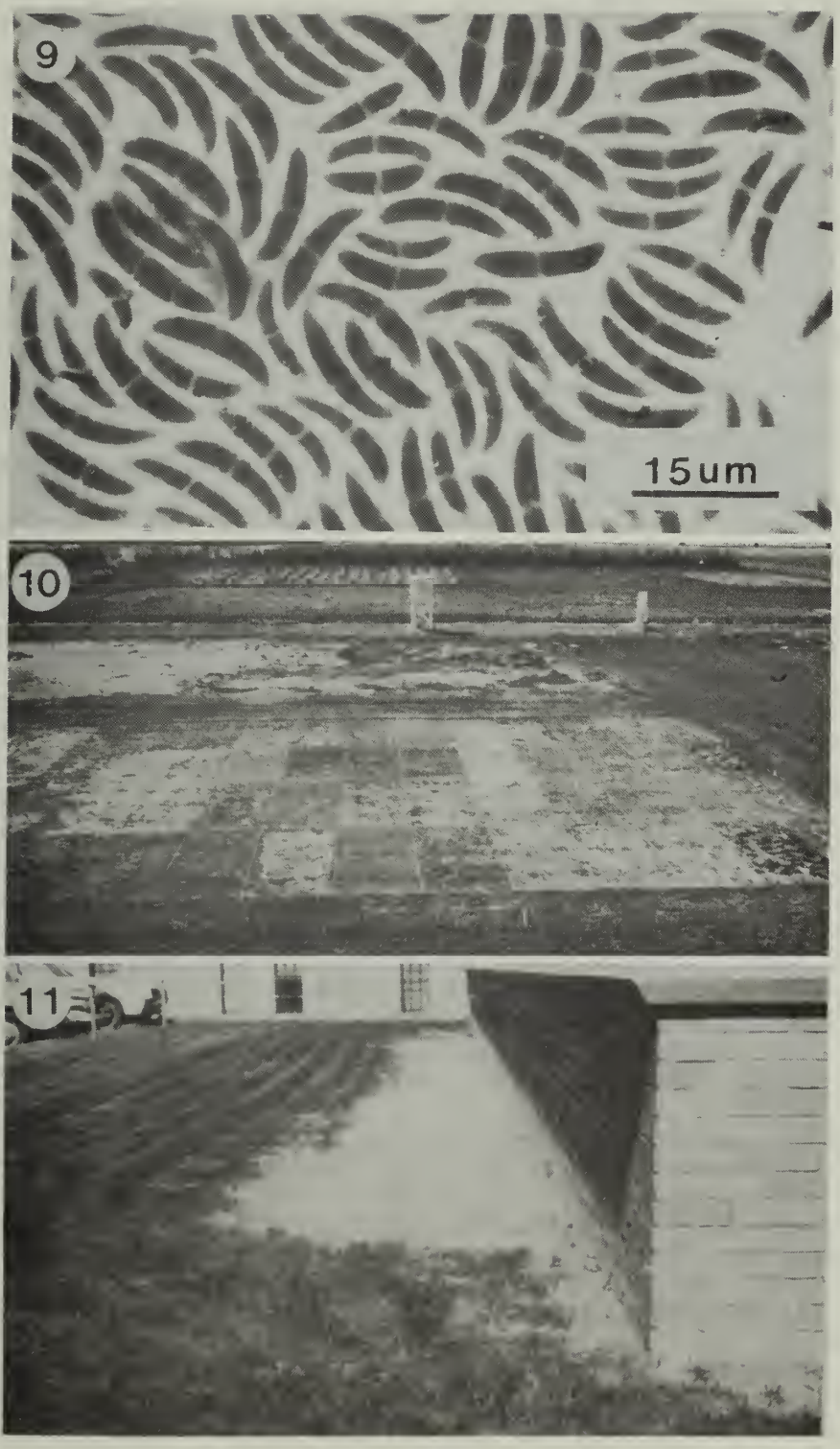

Fig. 9. Conidia of *Microdochium nivale*. Fig. 10. Differential resistance in *Agrostis* cultivars to pink snow mold. Fig. 11. Pink snow mold on *Poa pratensis* turf adjacent to the wall of a heated building.

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GREY OR SPECKLED SNOW MOLD OR TYPHULA BLIGHT Typhula incarnata Lasch. ex. Fr.

Several Typhula species cause disease at low temperatures on overwintering plants. T. incarnata, T. ishikariensis and its varieties ishikariensis, idahoensis and canadensis (Arsvoll & Smith, 1978; and see below) are important snow molds of forage and turf grasses and winter cereals from the cool temperate to the boreal regions of the northern hemisphere (Arsvoll, 1975; Arsvoll & Smith, 1978, 1979, 1979a; Dahlsson, 1973; Ekstrand, 1955, 1955a; Fushtey, 1980; Gould et al., 1977; Gulaev, 1948; Howard et al., 1951; Imai, 1929, 1930, 1936; Jackson, 1962, 1963; Jackson & Fenstermacher, 1969; Jamalainen, 1957, 1978; Jensen, 1970; Kock, 1976, and Dr. L. Kock, pers. comm. 27 Feb. 1981; Kristinsson & Gudleifsson, 1976; Lebeau & Cormack, 1959; Matsumoto & Araki, 1982; Matsumoto & Sato, 1982; McDonald, 1961; Meiners, 1955; Potatosova, 1960; Remsberg, 1940, 1940a; Roed, 1969; Schmidt, 1976; Skipsna, 1958; Skirde, 1970; Smith, 1974, 1974a, 1975, 1976, 1978, 1981; Stienstra, 1980; Tasugi, 1935; Taylor, 1974, 1975; Taylor & Fushtey, 1977; Tomiyama, 1955; Vaartnou & Elliott, 1969; Vang, 1945; Vanterpool, 1944; Volk, 1937; Wernham, 1941; Ylimaki, 1972). While T. incarnata may cause some injury in the absence of a snow cover, more severe damage in turfgrasses requires 2-3 months to develop (Jackson & Fenstermacher, 1969; Matsumoto & Araki, 1982; Smith, 1978). T. ishikariensis vars. need 4-5 months snow cover to cause severe injury to grasses (Arsvoll, 1975; Smith, 1978). This snow cover requirement, their temperature optima, the osmotic pressure tolerance of their mycelium (Bruehl & Cunfer, 1971; Tomiyama, 1955) fit the Typhula snow molds into different "epidemiological niches" in relation to Myriosclerotinia borealis which needs colder winters with a longer snow cover and Microdochium nivale which has a higher optimum temperature and which may cause severe damage in the absence of a snow cover. The distribution of these pathogens in North America is not a simple latitudinal one as suggested by Ekstrand (1955) for Scandinavia and Lebeau & Cormack (1961) for western Canada (Smith, 1974). Regional climates may be considerably modified by topography, aforestation and windbreaks which change the precipitation amounts and/or snow depth and duration which can determine the incidence or dominance of a particular Typhula sp. In northeastern Saskatchewan (Lat. 54°N) for instance, T. ishikariensis var. canadensis is a common turfgrass pathogen and it is important in the Peace River region of northern Alberta and British Columbia. On the other hand, T. incarnata, which can cause snow mold in the absence of a snow cover, is very uncommon in the Canadian Prairies. However, T. incarnata, T. ishikariensis vars. canadensis and/or isikariensis are important turfgrass pathogens in southern Ontario (Fushtey, 1980), Minnesota (Stienstra, 1980; Sweets &

Stienstra, 1976) and northern Wisconsin (Dr. G. Worf, 1977 & pers. comm. April 1980, and Smith, unpublished). During a survey of golf courses in northern Idaho and northeastern Washington in March 1976 (Smith, unpublished) the only Typhula sp. found on snow mold patches was T. incarnata, although T. ishikariensis is reported to be an occasional problem in the region (Gould et al., 1977). In western Washington only T. incarnata is common (Gould et al., 1978). Bruehl and Cunfer (1975) reported T. idahoensis Remsberg (T. ishikariensis (Imai) Arsvoll & Smith) dominant on winter wheat on former grasslands or grass-sagebrush lands and T. ishikariensis Imai (T. ishikariensis var. ishikariensis (Imai) Arsvoll and Smith, 1978) dominant on former forest lands in the Pacific Northwest of the USA. Bruehl and Machtmes (1980) continue to recognize T. idahoensis and T. ishikariensis as separate species with a continuum of cultural types between them, denying recognition of the idahoensis, ishikariensis and canadensis varieties of T. ishikariensis proposed by Arsvoll and Smith (1978). T. ishikariensis Imai sensu Arsvoll and Smith has a circumpolar distribution in Asia, Europe, and North America (Arsvoll & Smith, 1978; Imai, 1936; Matsumoto & Araki, 1982; Matsumoto et al., 1982; Masumoto & Sato, 1982). T. idahoensis Remsberg was described from Idaho and Montana (Remsberg, 1940, 1940a). It has a wider distribution than this e.g. in the Pacific Northwest of North America (Bruehl et al., 1978, Bruehl & Machtmes, 1980), but its distribution limits are uncertain because of earlier taxonomic confusion (Arsvoll & Smith, 1978; Jamalainen, 1957; McDonald, 1961). Arsvoll and Smith (1978) have found a morphologically distinguishable variety, T. ishikariensis Imai var. canadensis Smith & Arsvoll widely distributed on grasses in Canada from British Columbia to Ontario and parts of the northern USA. Biotypes of T. ishikariensis similar to var. canadensis are found in Hokkaido, Japan (Matsumoto & Sato, 1982). To accommodate the Canadian variant Arsvoll & Smith (1978) proposed that the prior specific name T. ishikariensis should be used with ishikariensis, idahoensis and canadensis as varieties. This was a compromise since there were enough differences between Canadian isolates of var. canadensis and the other two varieties to consider it as a separate species yet sufficient geneti compatibility to retain T. ishikariensis as the specific name. Recent work on cultural variation within T. idahoensis Remsberg and T. ishikariensis by Bruehl and Machtmes (1980) have not satisfactorily solved the taxonomic problem since the genetic relationships of var. canadensis isolates and North American vars. idahoensis and ishikariensis have not been adequately studied. The three varietal names in T. ishikariensis will be used here.

GREY SNOW MOLD OR TYPHULA BLIGHT CAUSED BY Typhula incarnata Lasch ex Fr.

Snow mold of turfgrasses caused by T. incarnata has been reported from Scandanavia (Arsvoll & Smith, 1979; Jensen, 1970; Smith, 1975; Ylimaki, 1972), the Netherlands (de Leeuw & Vos, 1970), Germany (Skirde, 1970), Austria (Kock, 1976, and Dr. L. Kock, pers. comm.), Switzerland (Smith, unpublished), the British Isles (Jackson, 1964), Japan (Hosotsuji, 1977; Shimanuki et al., 1981), western, central and eastern Canada (Fushtey, 1980; Smith, 1973, 1978; Vaartnou & Elliott, 1969), northwestern United States and Northern Great Plains (Evans, 1973; Gould et al., 1977; Meiners, 1955), the mid-west and eastern United States (Jackson & Fenstermacher, 1969; Remsberg, 1940; Wernham, 1941), and New England (Dr. J.M. Fenstermacher, pers. comm.).

The disease may develop in a mild form in winter in the absence of snow, appearing first as patches of yellow-brown grass, 2-5 cm in diameter. Under a snow cover the patches may coalesce and increase in size, up to 0.5 m (Fig. 12). On emergence from the snow, the bleached grass leaves are sometimes covered with a fairly sparse to dense white or greyish-white mycelium. The grey colour is probably due to the atmospheric pollutants in the snow (Jackson & Fenstermacher, 1969). Orange or faintly pink sclerotia, up to 5 mm in diameter may be found on or within the infected tissues at this time, often in the crowns of the plants. Where snow cover has been light and/or of short duration, plant injury is often superficial, but this fungus may cause severe, deep-seated damage in the plant under a long, deep snow cover. Under such conditions other snow mold fungi may be present in adjacent patches.

The fungus

Typhula incarnata Lasch ex Fr.

Corner (1950) considered that T. itoana from Japan (Imai, 1929) and T. incarnata from Europe, as redescribed by Donk (1933) (from Rabenh. Fung. Europ. No. 1313) were identical. T. incarnata Lasch ex Fr. (Fries, 1836) has priority over T. itoana Imai (Corner, 1950; Lehmann, 1964).

There is considerable confusion in the literature between T. incarnata and T. graminum (Lehmann, 1964; McDonald, 1961; Remsberg, 1940, 1940a; Tasugi, 1935; Vang, 1945; Volk, 1937) and other sclerotial fungi such as Sclerotium rhizoides Auersw. (Hungerford, 1923) and S. fulvum Fr. (Young, 1937). This is largely due to the failure to find fertile sporophores (Volk, 1937) and reliance on inadequate descriptions of the sclerotia (Remsberg, 1940, 1940a). However, from Tasugi's (1935) description of the T. graminum Karst. with which he was working it is almost certain that the fungus was T. incarnata Lasch ex Fr. Imai (1936) showed that T. incarnata from Japan and T.

graminum (not Karst) from CBS, Baarn, Holland were very similar in cultural characters and sporophore formation, but considered that T. incarnata was not the same as T. graminum Karst. Tomiyama (1955) showed that T. incarnata was heterothallic. Roed (1969), however, showed that isolates of T. graminum Karst. from CBS, Baarn, T. itoana from Canada (originally from Remsberg) T. incarnata Lasch ex Fr. from Japan (from Tomiyama) and three T. itoana isolates from winter rye and Phleum pratense were interfertile. He regarded these fungi as strains of T. incarnata Lasch ex Fr.

Berthier (1974) places T. incarnata Lasch ex Fr. ? in a new sub-genus Microtyphula in the Genus Typhula (Fries emend. auct. Syst. Mycol. 1821 1:494).

Sclerotia of T. incarnata from plants are brown or faintly pink and smooth when young, darkening to reddish-brown or dark-brown and wrinkling as they dry (Fig. 13). They are variable in shape from subglobose to elongated, flattened or irregular, 1-5 X 0.5-3 um. There is no stalk as in T. phacorriza Fries. In fall, 1-3 sporophores develop from each sclerotium. These are often pubescent with hairs clasping the sclerotium. They may be straight, slightly curved, simple or less commonly with branched stipes and clubs, 5-30 mm tall (Fig. 14a). The white stipes are 0.5-1 mm and the rose-coloured clubs 0.2-3 mm in diameter. Basidia forming the hymenium on the clubs are tetrapolar, and broadly clavate, 5-8 X 27-35 um with ovoid basidiospores flattened or incurved on one side above an apiculum 3-5 X 5-10 um (Arsvoll & Smith, 1978, 1979; Imai, 1936; Remsberg, 1940; Smith, 1981; Sprague, 1950).

On culture media mycelial growth is white, radial, sometimes stranded and the hyphae are dikaryotic with clamp connections. The optimum pH range for mycelial growth on PDA according to Tomiyama (1955) is 5-8. Unlike Myriosclerotinia borealis, frozen agar will not support the growth of T. incarnata, probably because it requires a higher water potential than M. borealis. The difference in response to temperature observed by Tomiyama (1955) and confirmed by Bruehl and Cunfer (1971) may be related to osmotic pressure tolerance. Sclerotia are produced in concentric rings or piled centrally, and they are often irregular, or lobed. The optimum temperature for mycelial growth is usually 9 to 15°C and the range from below -6 to 21°C (Arsvoll, 1975; Arsvoll & Smith, 1978; Smith, 1980 and Table 2 below). It is not always easy to distinguish single dark sclerotia of T. incarnata from those of T. ishikariensis vars., but those of T. incarnata are resilient when turgid and those of T. ishikariensis are brittle when pressure is applied with a needle point. The cortical cells of T. incarnata sclerotia are lobate, interlocking like a jigsaw puzzle (Arsvoll & Smith, 1978).

Table 2. Temperature ranges for *Typhula* snow molds in artificial culture (°Celsius)[1]

Species/variety	Temperature Minimum	Optimum	Maximum	Authority
Typhula incarnata Lasch Ex Fr.	< 0	9-12	18	Remsberg, 1940
	<-5	10-15	22-23	Ekstrand, 1955
	<-5	8-15	22-23	Tasugi, 1935
	0	10	25	Remsberg & Hungerford, 1933
	0	8	25-30	Volk, 1937
		10		Vang, 1945
	-6	9-12	21	Arsvoll, 1975
	<-6	12-15	21	Arsvoll & Smith, 1978
	-7	7-15		Tomiyama, 1955
	-10	7	22	Lehmann, 1965, 1965a
T. ishikariensis Imai var. *ishikariensis* Arsvoll & Smith	<-6	9-12	20	Arsvoll & Smith, 1978
="*T. borealis*" Ekstrand	-5	10	21	Ekstrand, 1955
T. ishikariensis Imai var. *idahoensis* Arsvoll & Smith	6	9	18	Arsvoll, 1975
	6	9	18	Arsvoll, 1975
	<-6	9-12	18-20	Arsvoll & Smith, 1978
	< 0	9-12	18	Remsberg, 1940
	<-5	5	20	Dejardin & Ward, 1971
T. ishikariensis Imai var. *canadensis* Smith & Arsvoll	<-6	6-9	21	Arsvoll & Smith, 1978
= "*T. hyperborea*"	-5	5-10	15-20	Ekstrand, 1955
Ekstrand	< 0	10	16	Potatosova, 1960

[1] From Smith (1969) - revised

Isolation of the fungus and pathogenicity tests

Allow the sclerotia to imbibe water and then surface sterilize them with 70% ethyl alcohol or hypochlorite solution (1% chlorine) for 2 min and then wash 5-7 times in sterile water. Slice the sclerotia with a sterile scalpel and plate out on malt or potato-malt agar (Smith, 1981). Incubate at 10-15°C.

For pathogenicity tests in controlled environments culture the fungus in PDA broth at 10-15°C, macerate the mycelium and apply to run-off on test grasses with an atomizer. Only one isolate should be used because mixing different ones may reduce pathogenicity (Arsvoll, 1976; Smith & Arsvoll, 1975). Incubate plants in the dark in moist chambers of the kind as suggested by Blomqvist & Jamalainen (1968), Arsvoll (1975) or Smith (1981) for up to 10 wk at 1-2°C. Place plants on a greenhouse bench to recover before rating. An inoculum of macerated mycelium gives more reliable infection than sclerotia.

Host range and susceptibility to T. incarnata

T. incarnata is a weak, unspecialized pathogen (perthophyte) on the Gramineae (Bruehl, 1967, 1967a; Cormack & Lebeau, 1959; Hirai, 1956; Imai, 1929; Lehmann, 1965a; Tomiyama, 1955; Werham & Chilton, 1943). It is a very successful saprophytic colonizer, even in the presence of other microfungi from 0-10°C (Matsumoto & Sato, 1982). Its sclerotia may form on dead or moribund parts of other plants. Host resistance is assumed to be non-specific (Lehmann, 1965) and quantitative, governed by several genes each possessing an additive effect (Arsvoll & Smith, 1979). There are considerable differences in resistance between species and cultivars of grasses to T. incarnata (Arsvoll, 1977; Gould et al., 1978; Schmidt, 1976; Vargas et al., 1972; Wernham, 1941). It is probable that the fertility conditions of the turf when attacks develop and the duration of the snow cover have as much effect on damage severity as inherent resistance of grasses (Jackson & Fenstermacher, 1969). Tomiyama (1955) showed that although older leaves (of wheat) were more resistant to the spread of T. incarnata than younger ones the latter were more susceptible to infection. The effect of plant age on the winter-hardiness of grasses (including susceptibility towards snow molds) has been reviewed by Arsvoll (1977). Usually first-year grasslands are more severely damaged by snow molds than older ones. Young plants were found to be more susceptible to snow molds, including T. incarnata in pot experiments than older, well-established ones (Arsvoll, 1977). Winter-hardiness was found to be greatest (in wheat) in the 4- to 6-leaf stage, but Bruehl & Cunfer (1971) noted that these plants were highly vulnerable to snow mold. Lehmann (1965a) noted that there was an interaction between winter-hardiness and susceptibility, but Tomiyama (1955) found that winter-hardiness was not related to T. incarnata resistance in wheat and that leaves of early-sown

plants suffered severe injury while those of later-sown ones generally did not (but see Hardening, Page 5). Winter-hardiness and snow mold resistance are both increased by reserves of carbohydrates and prolonged, deep snow cover subjects the plants to nutrient exhaustion and reduces disease resistance (Bruehl & Cunfer, 1971; Tomiyama, 1955, and see Hardening). Pre-hibernal fertilization and management greatly influence carbohydrate reserves, dormancy, hardening and subsequently freezing tolerance and snow mold resistance of grasses. Arsvoll & Larsen (1977) found that increasing supply of nitrogen was correlated with increasing susceptibility to T. ishikariensis as well as M .nivale and M. borealis in grasses although they did not test T. incarnata.

Hirai (1956) found that T. incarnata produced a cytotoxin which provoked a hypersensitive reaction in resistant plants. This was a simple acidic substance.

Disease development

Although T. incarnata will make mycelial growth at temperatures from -7 to 21°C (Arsvoll, 1975; Lehmann, 1965; Smith, 1980a), the optimum for most isolates, 9-15°C, is a little higher than for the other graminicolous Typhula spp., but a little lower than M. nivale (Smith, 1980a). Bruehl and Cunfer (1971) found that T. ishikariensis var. idahoensis was virulent at temperatures as low as -1.5°C on wheat, but that neither M. nivale nor T. incarnata caused snow mold at this temperature. Tomiyama (1955) found that lesions caused by T. incarnata on wheat leaves spread most at 5°C and ceased at -5°C. The mycelium of T. incarnata grows abundantly at 0-3°C at a time when grass vigor is low and plants are susceptible. Although grass plants may be damaged from late fall to early spring in wet, cold climates when there is little or no snow cover, more often the fungus is active under deep prolonged snow (Sprague & Rainey, 1950), especially when the turf surface remains unfrozen (Jackson & Fenstermacher, 1969; Koch, 1976).

Sclerotia form on and in diseased tissues as mycelial spread declines. This change may start before snow melt since immature sclerotia can be seen then. These oversummering structures are resistant to high temperatures and desiccation and remain dormant until autumn. Desiccation is necessary before germination will occur (Lehmann, 1965).

Potatosova (1960a) found that sclerotia would germinate in 4-6 weeks if kept moist at low temperatures (1.4-13.5°C). In nature, sclerotial germination takes place in cool moist weather in autumn either by production of vegetative mycelium or of sporophores. In laboratory tests in sand, the optimum temperature for myceliogenic germination was 2-10°C, and high substrate moisture and atmospheric humidity above 70% RH was necessary. Light is not necessary for mycelial germination (Lehmann, 1965), but for basidiocarp production short wave ultra-

violet light is needed (Lehmann, 1965; Remsberg, 1940; Tasugi, 1935).

Basidiospores are discharged during rainy or foggy weather (Sprague & Rainey, 1950) by wind and rain. The optimum temperature for their germination is 9-20°C according to Tomiyama (1955), but this was 12-17°C for Lehmann (1965) and they remained viable after 4 months storage at -10 to -15°C. Basidiospores germinate to produce a haploid mycelium which is less pathogenic than when it is dikaryotized (Lehmann, 1965a).

Weather conditions largely determine the incidence and time of development of sporophores. In Rhode Island this varies from late September to early November (Jackson & Fenstermacher, 1969). In England in 1963, in the heaviest recorded outbreak of the disease, sporophores developed in late September (Jackson & Fenstermacher, 1969). In the USSR sclerotia germinated from September to October (Potatosova, 1960a). Sprague & Rainey (1950) reported that sclerotia germinated in October and spores were trapped until 21 December in Washington. In Central Norway sporophores are freely produced in October, although sclerotia commence to germinate in September (Smith, unpublished). The extreme climate of the Canadian prairies is very unfavourable for sporophore production of graminicolous Typhula spp. (Smith, unpublished). Basidiocarps are not common on turf in western Washington (Gould et al., 1978). While some outbreaks of the disease probably result from basidiospore infection (Sprague & Rainey, 1950) mycelium produced by direct germination of sclerotia is more likely to start the disease (Sprague & Rainey, 1950; Tasugi, 1935; Tomiyama, 1955). Lehmann (1965a) considered that basidiospores were unessential as sources of infection in winter wheat and Tomiyama (1955) failed to infect wheat leaves with a basidiospore suspension.

Dikaryotic isolates of T. incarnata may show mutual antagonism which reduces the pathogenicity of mixtures of virulent strains (see T. ishikariensis below).

Control

There have been few studies on the effect of fertilization on the incidence and severity of snow mold caused by T. incarnata in turf. Tyson (1936) found that plots of bentgrasses fertilized with inorganic nitrogen, i.e. ammonium sulphate, urea or sodium nitrate suffered less from snow mold than those where the same amount of nitrogen supplied by cottonseed meal, dried blood or dried digested sewage was given[1]. Practical experience suggests that, whatever the cause, snow mold damage to turfgrass is aggravated by excessive or unbalanced nitrogenous fertilizer,

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[1]The causal agent of the snow mold was not specified, but from illustrations it appears to have been largely T. incarnata, a common cause of snow mold in Michigan (Vargas & Beard, 1971).

especially if applied late in the growing season which produces lush, unseasonable herbage growth (see earlier in text). High and unbalanced nitrogen increased susceptibility of forage grasses to T. ishikariensis (Arsvoll & Larsen, 1977; and see T. ishikariensis). The general turf management methods used for the cultural control of other snow molds should be used. See in particular, control of Microdochium nivale and T. ishikariensis.

Published information on the control of grey snow mold often does not specify the causal organism. This makes the evaluation of the effectiveness of the disease control given by resistant cultivars difficult. Regional evaluation of these is necessary as is the case with other snow molds (Smith, 1980). Unhardy cultivars, poorly adapted to the local environment should be avoided (Arsvoll & Smith, 1979). Vargas et al. (1972) found that most of the common cultivars of Poa pratensis were susceptible in Michigan, but Adorno, Monopoly and some selections from New Jersey showed fairly good resistance. T. incarnata and M. nivale often occur in fungal complexes, with T. ishikariensis under snowier conditions, in turfgrasses (Fushtey, 1975, 1980a; Smith, 1973, 1978) and winter cereals (Bruehl, 1967; Smith, 1978). Bruehl (1967) found that there was a strong correlation between the resistance of winter wheat lines to the three pathogens. Arsvoll (1977) found that there was a correlation between freezing resistance, a component of winter-hardiness, and susceptibility to these snow molds in two forage grasses. This may also be the case with some turfgrasses (Vargas et al., 1972), but this requires further testing.

The bentgrasses (Agrostis spp.) which are common components of fine turf, are often severely damaged by T. incarnata (Smith, 1978; Wernham, 1941). Gould et al. tested a large number of cultivars and strains of bentgrasses in turf for resistance to T. incarnata in western Washington. Plots were inoculated with cultures of the fungus isolated from golf greens. None were immune to T. incarnata, although some were more resistant than the older cultivars such as Highland and Astoria (A. tenuis Sibth.). The same strains were tested for resistance to M. nivale in western Washington (Table 3).

Table 3. Disease ratings for snow mold caused by T. incarnata in western Washington and M. nivale in eastern Washington on the same lines of Agrostis spp. in golf green turf. (Data from Gould et al. 1978).

Species	No. of entries	Average rating* T. incarnata	M. nivale
Agrostis tenuis Sibth.	33	3.97	1.43
A. stolonifera L.	73	1.74	3.34
A. canina L. ssp. canina Hwd.	9	2.67	2.44
A. gigantea Roth.	3	5.00	1.33

*The rating was on a scale of 1-5, where 5 was least disease.

A. gigantea (redtop) lines were most resistant to T. incarnata; few of the creeping bentgrasses (A. stolonifera) showed much resistance and on average were the least resistant to T. incarnata while several of the browntop bentgrasses (A. tenuis) showed considerable resistance. The velvet bentgrasses were intermediate. Against M. nivalis redtop and browntop were most resistant but the correlation between resistance to T. incarnata and M. nivalis was very weak except in the case of the velvet bentgrasses.

Chemical control of snow mold caused by T. incarnata

Where T. incarnata causes yearly problems fungicides are needed for effective control, especially on fine turf of golf and bowling greens composed of Agrostis spp. and Poa annua.

Mixtures of mercurous and mercuric chlorides (2:1) were considered the most reliable fungicides for the control of T. incarnata in the mid-west and eastern USA (Noer, 1944; Wernham & Kirby, 1943). In eastern Washington, cadmium succinate, phenyl mercuric acetate and thiram fungicides were more effective than the inorganic mercurials (Meiners, 1955). In Rhode Island Jackson and Fenstermacher (1969) confirmed the effectiveness of phenyl mercuric acetate, mercurous and mercuric chloride mixtures and cadmium succinate fungicides in preventing this snow mold. Vargas and Beard (1970) found that a granular formulation of chloroneb was more effective than a wettable powder formulation of the same material and mercury chloride mixtures. In Idaho, where T. incarnata was the dominant snow mold, Gould et al. (1977) obtained best control of the disease with early autumn, late autumn and spring treatments with (1) benomyl and chlorothalonil (2) mancozeb, mancozeb and chloroneb (3) mancozeb, benomyl and chloroneb and (4) mancozeb, thiophanate methyl and chloroneb formulations. In Michigan Vargas and Beard (1971) found that single applications of chloroneb or mercurous + mercuric chloride fungicides made 1 month before the development of a permanent snow cover were almost as effective as those made just before the snow cover developed. Taylor (1974) obtained as effective control of T. incarnata snow mold in the interior of British Columbia with one application of chloroneb as with mercurous + mercuric chloride. A wettable powder formulation of quintozene gave good control when applied at high dosage. Quintozene was also effective applied dry with a fertilizer. In New York State good control of T. incarnata was obtained with thiram and cadmium fungicides or combination products containing either of these materials. Combinations of thiophanate + thiram, chlorothalonil + thiram and of thiram + chlorothalonil + cadmium succinate were the most effective (Marion et al., 1979). In New Hampshire iprodione, quintozene, cadmium succinate, chlorothalonil + chloroneb and triadimefon were very effective against T. incarnata on bentgrasses (Nutter et al., 1979). Other materials reported effective are iprodione, oxycarboxin + thiram

+ carboxin and triadimefon (Jackson, 1983; Smith, 1980b), applied as preventatives before development of a fall snow cover.

SPECKLED SNOW MOLD CAUSED BY Typhula ishikariensis Imai AND VARIETIES

Many published records of typhula snow mold on turfgrasses do not specify whether T. incarnata or T. ishikariensis was involved. T. ishikariensis snow mold develops where winters are longer and more severe than is the case with T. incarnata (Arsvoll, 1973; Ekstrand, 1955; Jamalainen, 1974; Kristinsson & Gudleifsson, 1976; Smith, 1980a). Since the snow cover develops earlier and remains longer in these regions, turfgrasses probably suffer more because of the greater depletion of energy reserves (Tomiyama, 1955) than under the short-duration snow cover necessary for T. incarnata. However, there is also evidence from infection experiments that T. ishikariensis is more aggressive towards grasses than T. incarnata (Arsvoll, 1976, 1977; Cormack & Lebeau, 1959; Wernham & Chilton, 1943).

Imai (1930) described T. ishikariensis on wheat, grasses and red clover in Japan and Remsberg (1940) described the similar graminicolous T. idahoensis in North America. Roed (1956) and Jamalainen (1964) in Scandinavia and McDonald in Canada considered them synonymous. Ekstrand (1955) described two "new species", T. borealis on winter cereals and grasses and T. hyperborea on cereals and grasses only from northern Sweden, Norway and Finland (Arsvoll, 1975; Ekstrand, 1955). McDonald (1961) suggested that these were also T. ishikariensis. No type material of these "species" can be found (Arsvoll & Smith, 1978). Smith (1973, 1974) found a "new" graminicolous Typhula sp., temporarily called "FW", in western Canada. Arsvoll & Smith described this as T. ishikariensis Imai var. canadensis and at the same time reduced T. idahoensis Remsberg to a variety of T. ishikariensis. In 1973 T. ishikariensis Imai was identified from winter cereals in Idaho and Washington (Bruehl et al., 1975), but Bruehl and Machtmes (1980) consider this and var. idahoensis as separate species, and do not recognize var. canadensis.

T. borealis of Ekstrand (1955) and T. ishikariensis Imai var. ishikariensis Arsvoll & Smith (1979) should be regarded as the same fungus although genetic evidence is not available. Morphological descriptions are very similar. T. borealis and var. ishikariensis have a host range wider than the Gramineae (Arsvoll, 1975; Ekstrand, 1955). T. hyperborea (Ekstrand, 1955) and T. ishikariensis var. canadensis appear to be similar morphologically and in host range (Arsvoll & Smith, 1978), being confined to grasses and cereals. An isolate of T. borealis from tulips received from Dr. Tchackchenko at the Moscow Botanic Gardens in 1980 (Smith, unpublished) was of the var. ishikariensis.

T. ishikariensis Imai sensu Arsvoll & Smith (1978) has been reported causing snow mold of turfgrasses in northern Japan (Hosotsuji, 1977), northern Scandinavia (Arsvoll & Smith, 1979; Jensen, 1970; Smith, 1979; Ylimaki, 1972), Canada (Allen et al., 1976; Arsvoll & Smith, 1978; Ekstrand, 1955a; Fushtey, 1975,

1980; Lebeau & Cormack, 1959; Smith, 1976; Vaartnou & Elliott, 1969; Vanterpool, 1943), northern United States (Gould et al., 1977; Smith unpublished; Sweets & Stienstra, 1976; Wernham, 1941; Dr. G.L. Worf, pers. comm.). It may be expected on turfgrasses in other regions such as the northern parts of the USSR and China and Alaska where it has been found on forage or wild grasses, cereals or other plants (Kristinsson & Gudleifsson, 1975; Lebeau & Logsdon, 1958; Potatosova, 1960; Protcenko, 1967; Schmidt, 1975) although the variety concerned is often not known.

The fungi

Typhula ishikariensis Imai var. ishikariensis Arsvoll & Smith (1978)
= T. ishikariensis Imai (1930)
? = "T. borealis" Ekstrand (1955)

Sclerotia are erumpent, readily detached from the host, globose to subglobose or slightly flattened, light brown to almost black (0.3-) 0.5-1.5 (-2) mm diam., surface smooth to rough. Rind cells are fairly regular in outline, moderately lobate, rarely digitate (Årsvoll & Smith, 1978). Sporophores are 3-20 mm in length. In autumn, 1-3 sporophores are produced from each sclerotium; they are erect, straight or curved, rarely branched, fusiform, occasionally flattened and ramose (0.3-) 0.5-1 (-3) mm broad, grayish-white to light brown, stipe filiform, slender, darker than the fertile portion (Fig. 14b). Basidiospores are similar in shape to those of T. incarnata, ovoid, ellipsoidal, flattened to incurved one side above a pointed apiculus (5-) 6-8 (-11) µm. On agar media aerial mycelium is often sparse, sclerotia single, scattered or in concentric rings, on the surface or submerged in the agar (Arsvoll & Smith, 1978).

Ekstrand's description of T. borealis (1955) is very similar to the above, except that the sclerotia are slightly smaller, up to 1.5 mm and the basidiospores are slightly longer and narrower, 5.5-13.25 X 2.0-4.5 um. The host range on winter cereals and grasses, clover, winter rape and beets is similar to that of T. ishikariensis var. ishikariensis (Ekstrand, 1955; Imai, 1930; Tomiyama, 1961; Ylimaki, 1969). The cardinal temperatures for T. borealis were:- minimum, -5; optimum, 10; maximum, approximately 20°C (Ekstrand, 1955). The optimum pH for mycelial growth in culture for T. ishikariensis Imai is 5-8 (Tomiyama, 1955) and 5-7 (Sweets & Stienstra, 1976).

Typhula ishikariensis Imai var. idahoensis (Remsberg) Arsvoll & Smith (1979)
= T. idahoensis Remsberg (1940)

Sclerotia are erumpent, less easily detached from the host than those of var. ishikariensis, globose to subglobose or slightly flattened, brown to almost black, 0.5-2 mm diam., surface often rough and ridged; rind cells are irregular in outline. On sclerotia germinated outdoors in Norway, lobate, often digitate sporophores were 3-14 mm tall, clavulae elongate, fusiform, rarely ramose, sometimes inflated, 0.5-1.5 mm broad, greyish-white to light brown, stipe filiform, slender, darker than the fertile portion. Basidiospores are ovoid to ellipsodial, flattened or incurved on one side above a pointed apiculus (6-) 7-9 (-13) X (2.5-) 3-5 (-8) µm (Årsvoll & Smith, 1978). Bruehl and Cunfer (1975) give measurements of collections of both vars. of idahoensis and ishikariensis. Dimensions given by Remsberg (1940) for T. idahoensis are similar to those above.

On agar culture media mycelium is usually sparse, but occasionally abundant and floccose in some isolates. Sclerotia are single, clustered or tending to form in concentric rings, on the surface and submerged in the agar. Var. idahoensis is restricted to hosts in the Gramineae (Bruehl & Cunfer, 1975; Remsberg, 1940). Potatosova (1960) in the USSR considered on the basis of morphology that T. idahoensis Remsberg was synonymous with T. graminearum Gulaev (1948), T. humulina A. Kuznetzova (1953) and T. borealis Ekstrand (1955). While the general description given by Potatosova (1960) agrees with that of Remsberg (1940) dimensions are different: sclerotia, 0.3-2 mm; clavulae, 0.5-5.0 X 0.3-1 mm; stipae, 3-7 X 0.1-0.5 mm; basidia, 27-31.5 X 5.8-7.7 µm; basidiospores, 9.7-10.6 X 4.6-5.2 um.

Typhula ishikariensis Imai var. canadensis Smith & Arsvoll (1978)
? = "T. hyperborea" Ekstrand (1955)

Sclerotia are readily detached from the host or from abundant wefts of mycelium spanning the leaves (Fig. 15 & 16); they are globose to subglobose or elongate-oval, light brown to almost black (0.2-) 0.3-0.8 (-1.6) mm diam., surface smooth, often with attached hyaline hyphae when fresh, rind cells fairly regular in outline to very irregular, lobate, sometimes digitate (Arsvoll & Smith, 1978). Sporophores are (1-) 3-6 (-11) mm tall, clavulae elongate-fusiform, sometimes inflated, (0.2-) 0.4-0.8 (-1.4) mm broad, greyish-white to light brown, stipe erect, slender, darker than the fertile portion (Fig. 14c). Basidiospores are ovoid to ellipsoidal, flattened or incurved on one side above a pointed apiculus, (5-) 6-8 (-11) X (2-) 3-4 (-4.5) um. On agar media aerial mycelium is abundant, floccose; sclerotia are formed in the aerial mycelium, usually on the surface, but occasionally submerged in the agar, scattered or in

radial rows rather than in concentric rings. The hosts are grasses, winter cereals and occasionally forage legumes in northern North America (Arsvoll & Smith, 1978) and Japan (Matsumoto & Sato, 1982).

T. hyperborea described by Ekstrand from northern Scandinavia (1955) has similar sclerotia to the above, up to 1.5 mm in diameter with sporophores darker in colour than T. borealis. Basidiospores were 5.5-11.0 um long X 2.75-5.75 um wide. The aerial mycelium was often fluffy in culture and the hosts were gramineous species only. The description fits var. canadensis. The cardinal temperatures for T. hyperborea were:- minimum, -5; optimum, 5-10; maximum, 15-20°C (Ekstrand, 1955). This is similar to those for var. canadensis (Årsvoll & Smith, 1955). The Typhula sp. reported by Vanterpool in 1932 from turf in Saskatoon (1944) with sclerotia 0.5-1.0 mm in diameter was probably the var. canadensis.

Symptoms and diagnosis

In all varieties of T. ishikariensis, field symptoms are similar to those caused by T. incarnata (q.v.) except that sclerotia and sporophores of all vars. of T. ishikariensis are never red or pink as in T. incarnata and sclerotial characters are quite different. Compared with the orange brown or pinkish sclerotia of T. incarnata those of T. ishikariensis range from light umber to very dark brown or almost black when dry. The sclerotia of the latter are never gelatinous or "rubbery" like those of T. incarnata when swollen with water. The rind of T. incarnata sclerotia is much more difficult to remove than that of T. ishikariensis vars. In T. ishikariensis vars. sporophores vary in colour in different collections of the same variety from greyish-white to light brown. The stipe base is always darker than the clavula.

It is very difficult to determine the variety of T. ishikariensis if only a few sclerotia are available (Bruehl & Machtmes, 1980; Smith, 1980a). While there are differences in basidiospore morphology the fruiting stage may not be available for comparison. Ekstrand (1955) used basidiospore size and shape successfully to separate the "species" "T. borealis" and "hyperborea" but basidiospore characters do not provide a convenient diagnostic tool. The slightly lower optimum temperature of var. canadensis and more abundant scattered sclerotia in culture distinguishes the variety from the other two. Probably the best single diagnostic feature is rind character (see text, Table 4). Using a needle, the rind may be separated from the medulla of a sclerotium with a needle when fully swollen in water and softened for 15-30 min in a nearly-boiling 10% aqueous solution of potassium hydroxide. As a final resort, cultural characters and then the di-mon mating technique may be used to separate vars. idahoensis and canadiensis from var. ishikariensis (Bruehl et al., 1975; Smith &

Arsvoll, 1978). Bruehl et al. (1983) described a pigmentation reaction which has been used to distinguish between T. ishikariensis Imai and T. idahoensis Remsberg.

Matsumoto & Sato (1982) have collected T. ishikariensis sensu lato biotypes from various parts of Hokkaido in Northern Japan. They found that they comprised two genetically different groups which did not mate with each other. Biotypes in Group A were identical with T. ishikariensis and T. ishikariensis var. ishikariensis. The other group comprised biotypes B and C which were differentiated culturally. Type B was not identical with T. idahoensis or T. ishikariensis var. idahoensis, but biotype C was similar to T. ishikariensis var. canadensis. Matsumoto and Sato (1982) considered that the T. ishikariensis complex includes several populations differing in genetics and morphology in North America, Norway and Japan.

Bruehl and Machtmes (1980) who do not recognize var. canadensis, consider that there is a continuum of variation in cultural characters between T. idahoensis Remsberg and T. ishikariensis Imai. They suggest the use of T. ishikariensis sensu Arsvoll & Smith (1978) where there was no need to take the classification further. This is agreed. However, it is pointed out that the vars. canadensis and idahoensis which showed the greatest morphological differences were closest genetically.

Table 4. Morphological and cultural characters useful in differentiating among *Typhula ishikariensis* varieties

Character	var. *ishikariensis*	var. *idahoensis*	var. *canadensis*
Sclerotial attachment to plant tissue	Usually firmly attached	Tend to be superficial	Quite superficial, easily detached
Sclerotial diameter in mm	0.3-2	0.5-2	0.2-1.6
Sporophore height in mm	4-20	3-14	1-11
Aerial mycelium on BASM agar at 6°C	Usually little	Usually little	Abundant
Sclerotial arrangement in culture on BASM agar at 6°C	Sclerotia in concentric rings	Sclerotia in concentric rings or in a pile	Sclerotia scattered, abundant and suspended in aerial mycelium
Superficial appearance of rind of imbibed sclerotia	Often rough, but rarely ridged or wrinkled	Often wrinkled, ridged or with splits in surface	Ridges rare, but with superficial mycelium attached
Rind cell characters	Digitate cells rare, cell outlines smoother than var. *idahoensis*; cells spherical in some isolates	Cells more irregular in outline than var. *ishikariensis* digitate and lobate cells sometimes present; cell disjunctions and ridges	Rind cells resemble var. *ishikariensis* but less rounded than the former and not so lobate as the latter Rind ridges not common

Host range and susceptibility to Typhula snow molds compared

T. ishikariensis is considered to be a more aggressive snow mold than T. incarnata. Vars. ishikariensis and canadensis are reported to have wider host ranges than var. idahoensis (Arsvoll & Smith, 1978; Bruehl & Cunfer, 1975; Ekstrand, 1955; Imai, 1930; Masumoto & Sato, 1982; Ylimaki, 1969). Monokaryons are less pathogenic than dikaryons in var. idahoensis (Kiyomoto & Bruehl, 1976). There was no differential virulence to host cultivars although dikaryons showed great differences in virulence on a particular wheat cultivar. This is also the case with var. canadensis on winter rye cultivars (Smith, unpublished). Most grasses are susceptible to T. ishikariensis (Arsvoll, 1975; Kristinsson & Gudleifsson, 1975; Lebeau & Logsdon, 1958; Remsberg, 1940a; Schmidt, 1976; Smith & Arsvoll, 1975; Wernham & Chilton, 1943), including turfgrasses in Agrostis spp., Poa pratensis L., Festuca rubra L., Lolium perenne L., Festuca pratensis Huds. and Phleum pratense L. Grass cultivars and strains from northern regions are usually more resistant than those from the south (Andersen, 1966; Ekstrand, 1955; Jamalainen, 1974). Vaartnou and Elliott (1969) found considerable differences in susceptibility to T. ishikariensis (probably var. canadensis) in cultivars of F. rubra and P. pratensis in forage and seed tests in northwest Canada. Older stands were more severely damaged suggesting inoculum buildup. While none of the grasses were killed some were severely damaged, such as Golfrood, Duraturf and the Olds cultivars of F. rubra and Merion and Nugget in P. pratensis. Park P. pratensis tolerated the fungus while there were several tolerant clones in the Common sort. Highly- resistant clones were found in Reptans and Boreal F. rubra. Methods for large scale screening of grasses for resistance to T. ishikariensis vars. in controlled environments have been developed (Arsvoll, 1977; Arsvoll & Larsen, 1977; Cormack & Lebeau, 1959; Smith, 1981; Wernham & Chilton, 1943).

Disease development

Sprague (1952, 1959) and Sprague and Rainey (1950) suggested that infection by basidiospores was possible. Ekstrand (1955) considered that infection by T. borealis and T. hyperborea took place mainly by basidiospores through the leaf and shoot system. From there the fungi penetrated to the whole plant. This claim appears to have been based on observational rather than on experimental evidence. It seems more likely that mycelium from sclerotia is the main source of inoculum in T. ishikariensis as well as in T. incarnata (Cunfer & Bruehl, 1973). In var. idahoensis, after sporulation is complete, hyphae may emerge from the spent sporophores and grow like hyphae from sclerotia (Cunfer & Bruehl, 1973).

Matsumoto and Sato (1982) found T. ishikariensis had very

poor competitive saprophytic abilities on unsterile grass leaf blades, but isolates had very high virulence compared with T. incarnata. The latter was compensated with high competitive saprophytic ability (see Snow mold complexes and competition).

Sporophores of T. ishikariensis var. canadensis have not yet been found in the field in western Canada, on sclerotial inoculum of different isolates of the fungus applied to turfgrasses or winter cereals which subsequently developed severe snow mold (Smith, 1975a; Smith & Reiter, 1976). Nor were they found on sclerotia of different isolates applied to marked plots of turf in summer or on golf greens with a history of heavy attacks, in northeastern Saskatchewan. Many attempts to induce sporophore development on sclerotia of T. incarnata and T. ishikariensis vars. sown in pots in late summer and placed outside have always failed at Saskatoon in the Canadian Prairies (Smith, unpublished). This method of induction is reliable in eastern Washington (Christen, 1979; Cunfer & Bruehl, 1973) and in eastern Norway (Smith & Arsvoll, 1978). Sporophore production by both the above species is common in pastures in autumn in eastern and central Norway (Arsvoll, 1975; Smith, unpublished). Basidiospores probably have a greater role to play in infection in such more temperate climates than in areas with more extreme climates, such as the Canadian Prairies. Christen (1979) noted that secondary sclerotia would form in the sporophores in crosses of var. idahoensis X var. ishikariensis which had remained infertile. The secondary sclerotia, if produced in the field, would produce a further survival mechanism; they remained viable for 11 months at 10°C.

According to Potatosova (1960a) sclerotia of T. ishikariensis germinate in October at 1.4-4.6°C and do not need a dormancy period, just low temperature and moisture. Those of var. canadensis will occasionally germinate in moist chambers in a cool greenhouse but generally there is a requirement for exposure to short wave uv light from mercury vapor tubes or to daylight. The var. idahoensis is most virulent in the temperature range 1.5 to -1.5°C (Bruehl & Cunfer, 1975). This is perhaps related to its lower temperature optimum for mycelial growth.

Sclerotia are smaller and are produced in greater numbers in T. ishikariensis vars., particularly in var. canadensis than in T. incarnata and are more freely shed. The smaller size of the propagule is compensated for by greater numbers, perhaps an evolutionary modification favouring sclerotia as inoculum. The sporophores of T. ishikariensis are much shorter than those of T. incarnata and more sheltered in the turf which would be advantageous when the microclimate was extreme, but disadvantageous for wind dispersal. It seems likely that the basidiospores' main function is likely to be in the exchange of genetic material, especially in T. ishikariensis vars. The small size and ease of detachment of the sclerotia from leaves in var. canadensis, in particular, may be a modification in response to

very extreme climates allowing them to escape from very exposed positions on leaves to comparatively sheltered places in the sole of the turf. The smaller size and ease of detachment would also allow some short-range wind dispersal. Sclerotia of Typhula spp. are long-lived structures, retaining viability for many years in cold storage. Bruehl et al. (1966) observed that 8 years was not long enough to eradicate var. idahoensis or T. incarnata in wheat crops. McKay and Raeder (1953) and Bruehl et al. (1966) noted that snow mold increased with each successive wheat crop. Vaartnou and Elliott (1969) found field evidence for inoculum buildup in T. ishikariensis as the age of grass stands increased. Survival of var. idahoensis sclerotia buried in soil was little affected by storage at 1-2°C except in the presence of clover, but considerably reduced at 24°C when in the rhizospheres of pea and clover. Depth of burial had little effect on sclerotial survival, but germination was more rapid in deeply-buried sclerotia than those in the soil surface. There are psychrophilic soil-borne bacteria on sclerotial surfaces which can inhibit sclerotial germination, but do not antagonize growing mycelium. They may provide a mechanism of biological control operating within a specific crop rotation (Huber & McKay, 1968).

Arsvoll (1975b) and Smith & Arsvoll (1975) found that when macerated mycelium of dikaryotic isolates of T. ishikariensis var. ishikariensis from different geographic regions in Norway were mixed and used as inoculum against Phleum pratense L. in pathogenicity tests, significantly less severe disease was produced than when isolates were used separately. A similar reduction in aggressiveness was found in isolates of var. canadensis from different locations in Canada when these were mixed together. Mutual antagonism was demonstrated in culture in these two vars., but not between different dikaryotic isolates of T. incarnata. In a more critical study, Arsvoll (1976) showed that isolates of T. ishikariensis, and T. incarnata from different regions of Norway, aggressive when used singly, lost much pathogenicity when isolates of each species were mixed. Considerable intra-specific antagonism was shown between isolates obtained from the same field and even between isolates from the same sq. m in T. ishikariensis, although this mutual antagonism was not shown in all cases. In one field, all isolates from the centre of a 1 metre quadrat, and in another, two isolates from a 10 metre quadrat showed no antagonism suggesting that the isolates in the mixtures were of the same origin. It seems likely that intra-specific antagonism between Typhula isolates is due to the production of metabolites or staling substances which are mutually inhibitory to the different isolates. Variability in pathogenicity of the isolates does not seem to explain the effect of mixing isolates on reduction in disease severity. The mixing of isolates in these Typhula spp. and vars. is contra-indicated unless critical studies show that mutual antagonism is not involved (Arsvoll, 1976; Smith & Arsvoll, 1975). The genetical and epidemiological implications of mutual antagonisms

are unclear.

Ekstrand (1955) considered that adequate phosphorous, and to a lesser degree, potassium increased resistance to T. borealis. Lime applications had the effect of increasing resistance mainly by making the phosphorous available. Bruehl et al. (1966) found that moderate applications of ammonium nitrate and ammonium phosphate in autumn had no appreciable effect on the disease, but adequate nutrition aided recovery. Arsvoll & Larsen (1977) in experiments in controlled environments found that attacks by T. ishikariensis on Phleum pratense increased with increasing nitrogen and were severe at low phosphorous concentrations, decreasing in severity with increasing phosphorous. Equivocal results were obtained with potassium. Acclimation improved plant resistance except with high nitrogen and low phosphorous. The effect of excessive nitrogen applications is to delay the onset of dormancy and winter acclimation (hardening-off), which in cool temperate, continental and boreal regions, where snow molds such as T. ishikariensis, T. incarnata, Myriosclerotinia borealis and Coprinus psychromorbidus cause problems, is signalled by a "browning-off" of the turf (Smith, 1975b, 1981).

Tronsmo (1982) showed that resistance to freezing was positively correlated with resistance to T. ishikariensis var. ishikariensis in artificially hardened grass plants, but it was not in unhardened ones. Resistance to the pathogen was positively correlated between acclimated and unacclimated plants of the same genotype, but freezing resistance was not. While resistance to freezing in artificially-hardened plants of Phleum pratense decreased considerably after dehardening, resistance to T. ishikariensis showed no decrease after 2-weeks dehardening.

Control

General snow mold control methods should be used: (See also control of T. incarnata)

(1) Avoid unbalanced or excessive nitrogenous fertilizer, particularly towards the end of the growing season, to allow the grasses to "harden off". Moderate applications of balanced fertilizer may be applied when the turf has reached near-dormancy, especially if fertility has declined, as indicated by soil analysis, to encourage rapid growth recovery in spring.

(2) Improve soil and air drainage, and in snowy regions, regulate snow drifting with ventilated fences placed to drop the snow clear of the turf, or if that is not possible, to promote an even snow cover. Remove snow drifts with a snow blower in spring, if feasible, but take care not to cause mechanical injury by wheeled or tracked vehicles. More rapid snow melting in spring may be promoted by the application of dark material such as soot, dark sand, or topdressing when the sun becomes stronger.

(3) Use known resistant cultivars, or if not available, those which are well adapted to the regional climate. These are usually of northern origin.

(4) Apply suitable fungicides. These are indicated below. T. ishikariensis is less amenable to control by fungicides which are effective against T. incarnata, and several applications may be needed in fall and early winter. Since T. ishikariensis may occur in complexes with other snow molds, a fungicide programme should be chosen which will control all pathogens (see Complexes).

Chemical control

Mercurous and mercuric chlorides, singly and incombination were used to control snow molds of turfgrasses before Typhula spp. were known causes, from the late 1920's (Dahl, 1934; Evans, 1973; Monteith, 1927; Noer, 1944; Smith, 1965; Tyson, 1936). Dosages of up to 125 g/100 m^2 of the salts were used. Some organo-mercurials, notably phenyl mercuric acetate, and thiram (Meiners) (1955) were found effective. Quintozene (PCNB) was used to control snow molds of winter cereals in Germany (mainly T. incarnata and M. nivale) by Pichler (1957) and its use was extended to Scandinavia where T. ishikariensis was also involved. Jamalainen (1958) found phenyl mercuric acetate and phenyl mercuric salicylate more effective than quintozene against cereal snow molds, including T. ishikariensis, but in this case seedling infections with M. nivale were also involved. Dosages of active ingredient (Hg) were 75-212.5 g/ha and 110.5-221 kg/ha for the mercurials, but 5-10 kg/ha for quintozene. Andersen (1966) found methoxymethyl mercuric chloride more effective than captan, quintozene, or methyoxymethyl mercuric salicylate on a susceptible Phleum pratense cultivar attacked by T. ishikariensis. However, these materials were less effective when applied to a less susceptible cultivar. Ylimaki (1972) reported effective control of snow molds of turfgrasses in Finland, including T. ishikariensis, with quintozene applied in autumn. In Saskatchewan, Smith (1976) found that quintozene, mercury chlorides, chloroneb, carboxin, oxycarboxin + thiram + carboxin at 202, 108, 195, 150, and 215 g a.i./100 m^2, respectively, were the most effective materials where T. ishikariensis var. canadensis was the predominant pathogen in natural outbreaks of snow mold. Only one application of these materials was made in these tests and this is insufficient to give practical control in most cases. Where T. canadensis was in complex with Coprinus psychromobidus, quintozene and mercury chlorides were most effective. On turf of the Seaside and Penncross cultivars of A. stolonifera inoculated with a culture of T. ishikariensis var. canadensis on sterile grain, quintozene (275 g a.i./100 m^2) consistently gave effective control. In another test chloroneb (159 g a.i.), oxycarboxin + thiram + carboxin (224 g a.i.),

iprodione (100 g a.i.) and, unexpectedly, benomyl (31 g a.i.) gave a practical level of control with two autumn applications (Smith & Reiter, 1975). In Manitoba, Allen et al. (1976) reported excellent control of snow mold on golf course turf with quintozene + borax and generally good control with chloroneb, carboxin, carboxin + thiram and thiobendazole, all in combination with borax. T. ishikariensis var. canadensis was found in all tests and was the principal pathogen at two of these. However, at two locations, C. psychromorbidus was the dominant pathogen, causing severe damage. Borax is particularly effective against the latter pathogen (Lebeau et al., 1961; Smith & Mortensen, 1981) but is liable to be phytotoxic to turfgrasses. Taylor (1974) in tests on putting green turf in the interior of British Columbia against snow mold caused by complexes of borax T. ishikariensiss var. canadensis, T. incarnata and M. nivale found that the non-mercurials, chloroneb (179 g a.i.) and quintozene (112.5 g a.i./100 m^2) would effectively control mild outbreaks of snow mold with one application. To control severe cases, up to 214 g a.i./100 m^2 was used without turf damage. Applications of chloroneb and quintozene in granular form at the same dosage gave similar results to the spray application (Taylor, 1975). In southern Ontario, Fushtey (1975) found that on turf of Agrostis stolonifera L. fungicides containing benomyl or related compounds failed to give satisfactory control where Typhula spp. T. ishikariensis var. incarnatas T. ishikariensiss var. ishikariensis and T. ishikariensis var. canadensis, (Fushtey, 1980; Smith, unpublished) were involved. Fungicides containing chloroneb or chlorothalonil were effective. Late autumn application, probably not before 1 November, was suggested. In later studies in southern Ontario (Fustey, 1980) where the three Typhula spp. or vars. were complexed with M. nivale, fungicides containing inorganic mercury chlorides, and phenyl mercuric acetate gave the best overall control. Where there was little disease (controls less than 7%) all fungicides gave acceptable control. Where T. ishikarensis var. canadensis was the dominant snow mold and the disease was very severe (controls averaged 85%) quintozene (131.25 or 178.75 g a.i.), mercurous + mercuric chlorides (72 g Hg salts), an experimental wettable powder (Baymeb 6447, 60 g a.i./100 m^2), a quintozene granular material at twice the standard dosage (16.9% quintozene) and a granular fungicide containing 0.68% phenyl mercuric acetate + 4.65% thiram at twice normal dosage gave excellent control. Conflicting results were obtained at two other sites. On one, where T. ishikariensis var. canadensis was also severe (controls averaged 95%) only chlorothalonil (194.4 g a.i.), mercurous + mercuric chlorides (75 g a.i.) and the granular phenyl mercuric acetate + thiram gave practical control. On the other, where T. ishikariensis var. ishikariensis was moderately severe (controls averaged 65%) only benomyl and iprodione (at the highest dosage 60 g a.i./100 m^2) failed to give effective control. In Japan, Shimanuki et al. (1981), effective control

of Typhula blights of Agrostis turf was previously obtained with mercurial fungicides such as phenyl mercuric acetate and with pentachlorophenol. The use of these has been abandoned because of toxicity. Recently mepronil (3-isopropoxy-2-methyl benzanilide) and oxine-copper [bis(quinoline-8-olato)copper] have been found effective and are registered for use against Typhula blights.

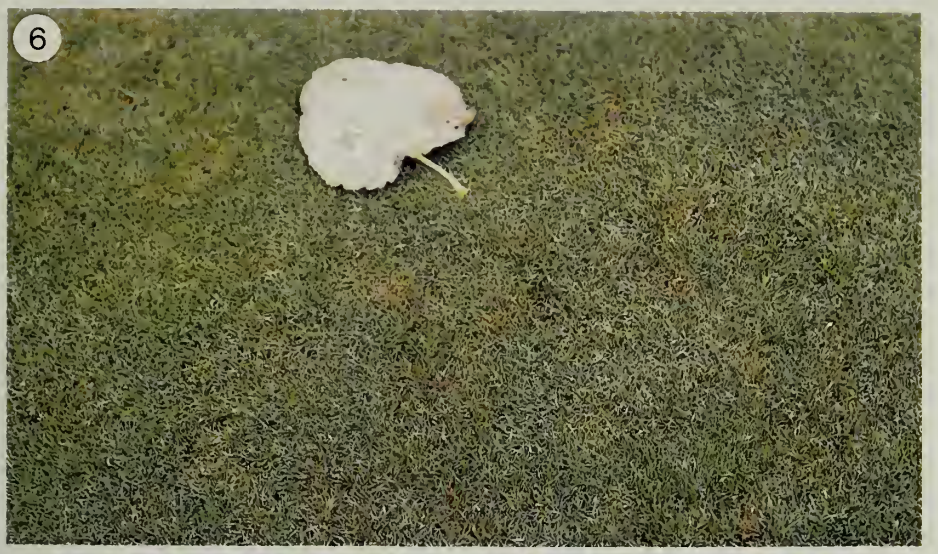

Fig. 6. Very early symptoms of fusarium patch disease. Fig. 7. Fusarium patch disease - late fall symptoms. Fig. 8. Pink snow mold symptoms after snow melt.

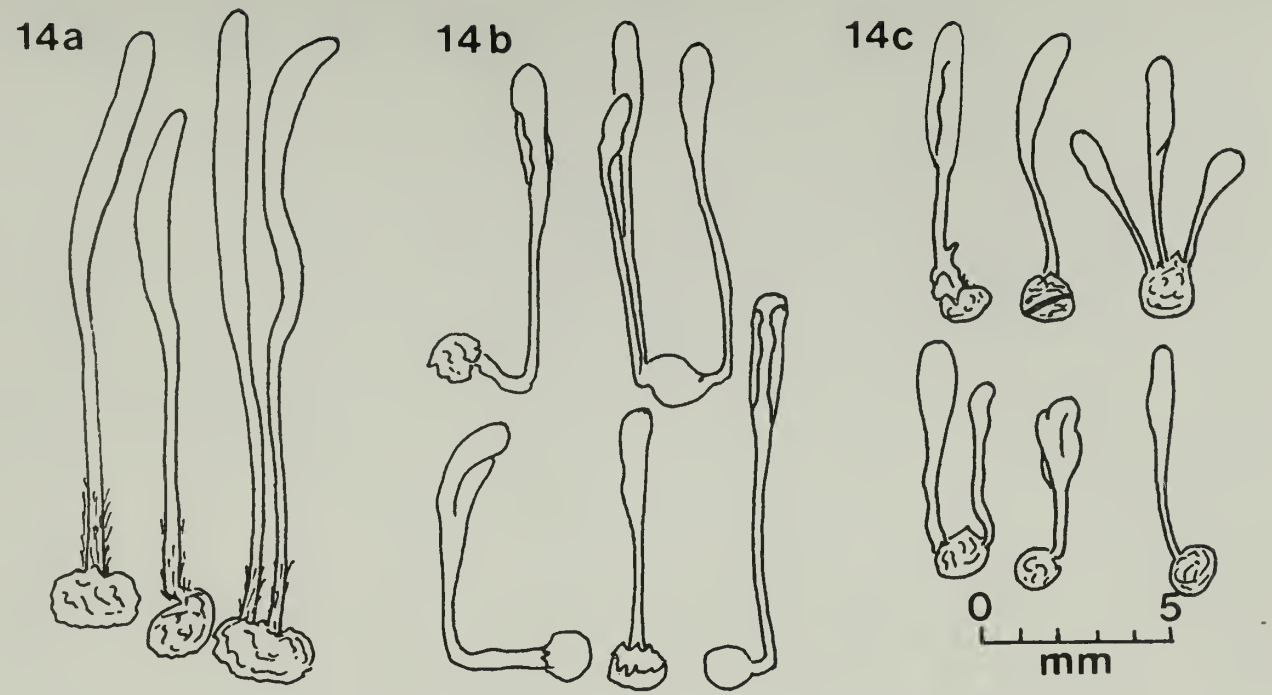

Fig. 14. Sporophores of (a) *Typhula incarnata*, (b) *T. ishikariensis*, and (c) *T. ishikariensis* var. *canadensis*.

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Grey or speckled snow mold or typhula blight

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SCLEROTINIA SNOW MOLD OR SNOW SCALD

Sclerotinia snow mold causes damage to winter cereals and perennial grasses in Scandinavia, Finland, northern Europe and Asiatic parts of the USSR and the Ukraine, northern Japan, Alaska, some northern states of the USA and Canada (Adachi et al., 1976; Andersen, 1960; Arsvoll, 1973, 1976; Cormack & Lebeau, 1959; Ekstrand, 1955; Eleneff, 1926; Groves & Bowerman, 1955; Jamalainen, 1949; Khokhyrakoff, 1935; Lebeau & Logsdon, 1958; Pukhalski, 1937; Roed, 1960; Sakuma & Narita, 1963; Shavrova, 1972; Smith, 1975; Solkina, 1939; Sprague et al., 1971; Tomiyama, 1955; Tupenevich, 1965; Tupenevich & Shusko, 1939; Ulander, 1910; Vleugel, 1917).

Reports of the disease on turfgrasses are much less numerous than those on winter cereals and forage grasses. However, in regions where forage grasses are damaged by the fungus, turf attacks may be expected. The disease has been reported on turfgrass in Norway, Sweden, Finland, Alaska and northwest Canada, the Prairie Provinces, southern Ontario and Minnesota (Allen et al., 1976; Groves & Bowerman, 1955; Hansen, 1969; Jensen, 1970; Kallio, 1966; Smith, 1971, 1973, 1974, 1978; Stienstra, 1974, 1980; Vaartnou & Elliott, 1969; Ylimaki, 1972).

Symptoms

When deep, long-duration snow covers melt in spring, patches of grass up to 15 cm approximately across, with water-soaked leaves and sparse grey mycelium appear. Several patches may coalesce (Fig. 17a). The infected leaves bleach almost white and wrinkle up to become thread-like on drying and exposure to light, but later darken with the growth of saprophytic fungi. Plants bearing sclerotia are almost always dead. Sclerotia are at first cream to putty-coloured or even faintly pink when broken across, globular, elongate or flake-like, sometimes arched, with the remains of plant vascular tissues attached. They are found in sheaths, crowns, leaf axils on the surface of or within the leaves. Their size varies according to the size and nutrition of the host and its organs on or in which they were formed (Jamalainen, 1949; Sakuma & Narita, 1963), but they are commonly up to 7 or 8 mm long and up to 3 or 4 mm wide when fresh (Fig. 17b & c, Table 5). When mature they have distinct black rinds and, when the host tissues have dried, readily become detached from the plant. On drying they become furrowed or wrinkled.

The fungus

Myriosclerotinia borealis (Bubak & Vleugel) Kohn (1979) syn.; Sclerotinia borealis Bubak & Vleugel (Vleugel), (1917); Saccardo Syll. Fung. 24, Sect. 2 p 1179, 1928).

Jamalainen (1949), McDonald (1961), Roed (1960) and Sprague et al. (1961) considered that S. graminearum Eleneff ex Solkina (Solkina, 1939) is very similar, if not the same, fungus. The latter name has been retained by Russian workers (Shavrova, 1972) and was used for many years in Japan (Tomiyama, 1955). S. borealis has priority over S. graminearum because the latter was not fully described (in 1919) by Eleneff (Eleneff, 1926; Solkina, 1939). Kohn (1979) transferred the fungus to the genus Myriosclerotinia. Ulander (1910) first observed the fungus causing damage to Dactylis glomerata L. and other grasses near Lulea in northern Sweden.

Sclerotia may germinate in the autumn to produce one or more stalked, cup-shaped apothecia (Fig. 17c) varying in colour from pale yellow, fawn, buff, or even faint pink to pale brown. Dimensions of fungal structures are given in Tables 5 & 6. The stalks are usually about 1 mm in diameter at the base. The upper surface of the disc of the mature apothecium bears asci and filiform, hyaline paraphyses, the latter slightly swollen at the free end and about 2 um in diameter. The asci are cylindric, tapering to a slender stalk, inoperculate with an apical pore (Table 6). Ascospores are uniseriate, one-celled, hyaline, often unequal in size, elliptical to oval sometimes slightly pointed. They are discharged forcibly into the air. No macroconidia have been found. Groves & Bowerman (1955) found spermatia 2.5-3.5 um, globose, hyaline, only in culture. They were produced endogenously in flask-shaped phialides borne in clusters on the mycelium. Solkina (1939) found microconidia in S. graminearum. These were globose, hyaline, uniguttulate, 2 µm in diameter are borne on irregularly branched conidiophores.

Table 5. Dimensions of sclerotia of *M. borealis* from grasses in mm.[1]

Authority	Length (major axis)	Breadth (minor axis)	Thickness
Groves & Bowerman (1955)	3-8	1-4	
Jamalainen (1949)	2-6	1-3	0.6-2
Roed (1960)	3-5	1-2	
Sakuma & Narita (1963)	1-10	0.2-4	
Arsvoll (1975)	2-8	1-4	

[1]Sakuma & Narita (1963) found that the shape and size of sclerotia were related to the species of grass on which they developed. There was almost no difference in the size of the apothecia, asci or ascospores produced by these sclerotia.

Table 6. Number of apothecia per sclerotium, dimensions of apothecia, asci and ascospores of *Myriosclerotinia borealis* Bub. & Vleug. and *S. graminearum* Elen. ex Solkina

Authority	No. apothecia/ sclerotium	Apothecial width/mm	Apothecial stalk length/mm
M. borealis			
Vleugel (1917)	1-3	up to 6	up to 25
Groves & Bowerman (1955)		2.5-5.5	2-6
Tomiyama (1955)		up to 6 mm	1-15
Sprague et al. (1961)	(lab. induced)	up to 4	up to 9
	(natural)	0.5-6	up to 10
Årsvoll (1975)	1-6 (1-3)	1-8 (2-6)	1-15 (2-6)
Sakuma & Narita (1963)		1.5-5	1-18
Jamalainen (1949)	(young)	0.5-2.1	3.3-9.6
	(older)	2.5-5.5	
S. graminearum			
Solkina (1939)		2.5-6	up to 15

Authority	Asci Length (um)	Asci Width	Ascospores Length (um)	Ascospores Width
M. borealis				
Vleugel (1917)	190-210	9-13	19-28	7-11
Jamalainen (1949)	180-250	8-14	9-22	
Groves & Bowerman (1955)	175-200	11-14	17-21	6-8
Tomiyama (1955)	202-266	11-17	18-23	8-10
Roed (1960)	170-220	8-13	14-28	7-12
Sprague et al. (1961)			17-24	8-11
Sakuma & Narita (1963)	180-274	11-17	17-28	7-12
Arsvoll (1975)	150-220	8-13	12-22	6-8
Smith (unpubl.)[1]	146-179	9-12	12-16	6-7
S. graminearum				
Solkina (1939)	175-300	10-14	16-23	7-10

[1] From *Agrostis stolonifera*

Table 7. Cardinal temperatures for vegetative growth of *S. borealis*

Authority	Minimum	Optimum	Maximum
Ekstrand (1955)	<-5	5-10	20
Tomiyama (1955)	-7	7-15	
Sprague et al. (1961)		4	
Sakuma & Narita (1963)		8	18-20
Ward (1966)	<-7	0	15
Arsvoll (1976)	-6	3-6	18

Several workers found that the naturally produced sclerotia needed a ripening period before they would germinate and produce fertile apothecia (Pukhalski, 1937; Solkina, 1939; Sprague et al., 1961; Tupenevich & Shirko, 1939; Yakovleff, 1939). Yakovleff (1939) considered a period of exposure to sunlight to be needed also. Groves & Bowerman (1955) found that for sclerotia of Canadian isolates produced on sterile grain, the following treatment would induce fertile sporophores:-

Two months on moist, sterilized quartz sand in the dark at 0°C.
Spermatization followed by maintenance in the dark at 5°C for 1 month.
Transfer to shaded greenhouse at 10-5°C for 2 months.

Since fertile sporophores appeared on selfed, crossed and unspermatized cultures held under the same conditions, it was concluded that M. borealis is homothallic and self-fertile, but this treatment failed to induce fertile sporophores in a Swedish isolate. Sprague et al. (1961) in Washington induced fertile sporophores by maintaining naturally-produced sclerotia on wet sand under lights, including 2 h/day "hard" uv, for about 5 weeks at 5-6°C. With sclerotia of four Norwegian grass isolates cultured on sterile wheat grains, Årsvoll (1976) found that the following conditions were optimum for ascospore production:-

Pre-treatment freezing of sclerotia at -6°C.
Excess water in sand in a covered germination container.
Sand medium buffered at pH 5-6.
Twelve h/day light from fluorescent "daylight" tubes at 2000 lux plus additional nuv light.

Arsvoll (1976) found that under these conditions sclerotia germinated without a resting period and that they maintained some germinability for more than 3 years. Under optimum conditions, germination of the sclerotia started after 3-4 weeks and the first mature apothecia were formed in another 3-4 weeks. Smith (unpublished) has followed precisely the methods given by Arsvoll (1976) with sclerotia of Canadian isolates of M. borealis from natural sources and cultures on grain with indifferent success. Usually only infertile sporophores developed, although a collection from Agrostis stolonifera in southern Saskatchewan yielded fertile sporophores in the laboratory. These structures have been recorded in the field in southwest Alberta (Dr. L. Piening, pers. comm. 1980).

The optimum temperature for ascospore germination in S. graminearum is 3-16°C. The spores will not germinate at 30°C. Freezing at -3°C did not kill the spores (Solkina, 1939). In M. borealis, ascospore germination occurred between -6 and 21°C with optima above 9°C in the presence of free water drops on the surface of water agar. Glycerol was added to the agar to prevent

freezing. The storage of apothecia sealed in polyethylene for 3 years at -23°C did not affect ascospore germinability. Ascospores germinate from one or both ends and will do so even when still contained in the ascus (Årsvoll, 1976).

The fungus may be isolated from fully imbibed sclerotia after surface sterilization with 70% ethyl alcohol and/or 1% chlorine water for 2 min, followed by washing in several changes of sterile water. The sclerotium is then sliced with a sterile scalpel and the pieces planted on potato-dextrose agar, or on malt-yeast-glucose agar (Ward, 1966).

M. borealis grows better than most other snow molds at low temperatures (Table 7 and see other snow molds). Ward (1966, 1968) in critical studies on the relationship between temperature and growth and respiration found that the optimum temperature for vegetative growth was 0°C, although the initial growth rate was slightly higher at 5, 10 and 15°C. Ice crystals retarded, but did not prevent, mycelial growth on agar medium. The lethal temperature was 30°C after 2 days exposure, which is the same upper limit found for ascospore germination by Solkina (1939) in S. graminearum. Tomiyama (1955) found that S. graminearum was able to develop more quickly on frozen media than on unfrozen ones at the same temperature. Sclerotium formation occurred from 15-0°C with an optimum at 10°C. At temperatures above 15°C respiration continued to a maximum at 25°C, but was not coupled to growth which had stopped. For vegetative growth pH optima were 3-6 and there was little growth at pH 8 and 9 (Ward, 1966). This is almost the same as the pH range for apothecial production of 3.5-7, with an optimum of 3-6 found by Arsvoll (1976). Yakovleff (1939) found that the fungus developed most profusely on acid soils and Demidova (1961) found that no sclerotia germinated at pH 8. Tomiyama (1955) found the pH optimum for mycelial growth to be 3-6. The different cardinal temperatures for growth noted by workers (Table 7) may have arisen from differences between isolates from widely differing geographical sources and hosts and different cultural techniques and should not be regarded as good evidence for speciation within S. borealis.

Pathogenicity tests

Tomiyama (1955) was unable to obtain infection of wheat by soil inoculation with M. borealis. Cormack and Lebeau (1959) found that infection did not develop well in cold-acclimated grasses when inoculum was placed on the soil surface below them and incubation was at 2 or 5°C. No sclerotia were produced on the plants. Sprague et al. (1961) obtained only slight lesioning at 2-3°C when mature apothecia from the field were used as the source of ascospore inoculum. Sakuma and Narita (1963) used ascospores produced by apothecia on sclerotia from different grass species in successful reciprocal infection studies. They found almost no differences in pathogenicity of the different

isolates. Årsvoll (1976) obtained infection of detached leaves of Phleum pratense in 2 weeks at 0°C when ascospore inoculum was applied. Arsvoll (1977) and Arsvoll and Larsen (1977) used macerated mycelial cultures grown on potato-dextrose broth and ascospores successfully to obtain infection in pathogenicity tests with grasses at 0°C. Ascospore suspensions were probably more effective as inoculum than macerated mycelium. 0°C was a more effective incubation temperature than 2°C. Smith (unpublished) found infection poor at -2°C using mycelial suspensions.

Host range and susceptibility

Most of the commonly used cool-season turf grasses are reported susceptible to Myriosclerotinia borealis (Cormack, 1952; Ekstrand, 1955; Groves & Bowerman, 1955; Hoshiro & Hirashima, 1978; Jamalainen, 1949, 1974; Kallio, 1966; Lebeau & Logsdon, 1958; Mastumoto & Araki, 1982; Nissinen & Salonen, 1972; Roed, 1960; Sakuma & Narita, 1963; Smith, 1972, 1974; Tupenevich & Shirko, 1939; Vleugel, 1917). Jamalainen (1949) found that M. borealis was most severe in Poa trivialis, Phleum pratense, Festuca rubra and Lolium perenne, but that there were great differences in resistance between strains of species. Generally grass lines from northern regions of Fenno-Scandia were more resistant in Sweden, Norway and Finland to M. borealis than more southerly lines (Ekstrand, 1955; Jamalainen, 1974). Southern Swedish and Danish varieties of Festuca pratensis were susceptible in northern Norway. Lolium perenne is generally so susceptible to all snow molds and frost damage that it does not overwinter well in Finland (Jamalainen, 1974; Nissinen & Salonen, 1972). Festuca rubra was also extremely susceptible, even in southern Finland. Kallio (1966), in Alaska, found that Common Kentucky Poa pratensis was the most resistant to M. borealis, followed by Park and Newport. Merion was least resistant. The Norwegian cultivar Holt was the most resistant Poa pratensis in northern Finland (Jamalainen, 1970). Although some cool-season grasses show resistance to M. borealis, if conditions are particularly favourable for the fungus it will decimate even the most resistant. Sakuma & Narita (1963) reported that perennial ryegrass and fescues are so easily damaged by the disease that they are not grown much in eastern Hokkaido. Grass cultivars which were not frost-hardy or became depleted in nutrients under a snow cover were nearly all susceptible to M. borealis (Hoshiro & Hirashima, 1978). In a severe outbreak in southern Saskatchewan in 1971 (Smith, 1972), Seaside Agrostis stolonifera L. was less severely damaged than the Penncross cv. First-year stands of agricultural grasses have been reported to be more susceptible than older ones (Andersen, 1960; Arsvoll, 1975, 1976b, 1977; Blomqvist & Jamalainen, 1968; Jamalainen, 1968; Thorn, 1967).

Epidemiology

Most workers agree that the most severe damage from M. borealis occurs in years with deep, prolonged snow covers, particularly if that cover develops on unfrozen or lightly frozen soil (Adachi et al., 1976; Arsvoll, 1973; Ekstrand, 1955; Elenev, 1926; Hoshiro & Hirashima, 1961; Jamalainen, 1949, 1974; Kallio, 1966; Khokhryakov, 1935; Lebeau & Cormack, 1961; Sakuma & Narita, 1974; Smith, 1974; Sprague et al., 1971). Ozaki (1979), in Hokkaido, considered that a snow cover more than 40 cm deep was necessary to maintain an optimum temperature for the high activity of the pathogen. These findings contradict those of Tomiyama (1955) who noted that M. borealis was common in Hokkaido in northern Japan when there was comparatively little snow. He also found that it was the prevalent pathogen under snow when the soil was frozen for a long time. Roed (1960), in Norway, also found that more severe cases of S. borealis damage occurred after winters with rather thin snow cover on deeply frozen soil. He commented that deep or long duration snow covers on frozen or slightly frozen soil also favoured the development of other snow mold species. Tomiyama (1955) showed that the mycelium of M. borealis was able to grow more quickly on frozen agar media than on unfrozen (supercooled) ones at the same temperature.

Soil inoculation with sclerotial or mycelial cultures was not effective in establishing infection with S. borealis (Tomiyama, 1955). Poor infection resulted when mycelial inoculum of the fungus was applied to the soil surface (Cormack & Lebeau, 1959). Arsvoll (1975) was unable to obtain infection with mycelial plugs. Slight infection only (2-5%) resulted from soil inoculation in winter cereals when sclerotia of M. borealis grown on sterile rye grain were used. This compares with 40-94% for Typhula ishikariensis var. canadensis (Smith, 1975). No apothecia of M. borealis or basidiocarps of the Typhula were seen. On the other hand, moderately heavy (up to 29% of the plot area) to very heavy infection (up to 99%) resulted from turfgrass inoculation with sclerotial cultures grown on sterilized rye grain (Smith, 1976; Smith & Reiter, 1976) although apothecia were not observed[1]. Tomiyama (1955) failed to obtain infection with ascospores, although he had difficulties in maintaining suitable low temperatures for incubation for a sufficiently long time. Arsvoll (1975) obtained heavy infection of Agrostis tenuis, Festuca pratensis, F. rubra, Lolium perenne, Phleum pratense, Poa pratensis and other grasses at 0°C after 12 weeks incubation using ascospores as inoculum. Matsumoto & Araki (1982) found that M. borealis developed in the first half of winter on grass plants which had been injured by cold and subsequently were snow-

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[1]These may have developed unnoticed since Smith was abroad in autumn of 1975 when apothecial development was expected.

covered. Attacks by Typhula incarnata follow as host tissues degenerate (Tomiyama, 1955). Ascospore infection in autumn appears to be the most likely means of primary infection in the field. Spread from host plants under snow seems to be through contact of infected leaves with healthy ones (Sakuma & Narita, 1963). Entry of mycelium into leaves is via wounds (Tomiyama, 1955) or through stomata or between cells (Årsvoll, 1976).

Saturated soils and temperatures between 6 and 12°C, approximately, favour sporulation. Higher temperatures (greater than 15°C) inhibit sporulation. In regions with long periods of humid, cool or cold weather in autumn, which generally shade off into winter apothecia are often not difficult to find in all types of grassland in some regions of Norway (Arsvoll, 1973; Roed, 1960; Smith, unpublished). Where the seasonal temperatures change more rapidly and autumn is often dry, as in the Central Prairies of Canada, sufficiently long periods of favourable weather for apothecial production are rare. Perhaps the criteria which govern ascospore production and infection may be more critical in some regions in determining disease occurrence and severity than subsequent temperatures under the snow and snow depth (Årsvoll, 1976; Ekstrand, 1955; Fokin, 1939; Khokhyrakov, 1935). M. borealis is a slow-growing pathogen without high competitive saprophytic ability. Once infection is established, an extended period of deep snow cover, when host nutrient reserves are declining may lead to further weakening of host resistance to invasion (Bruehl & Cunfer, 1971; Hoshira & Hirashima, 1978; Tomiyama, 1955). The lower optimum incubation temperature in pathogenicity tests of 0°C for M. borealis (Arsvoll & Larsen, 1977) suggests that it would have some competitive advantage over Typhula spp. with optima at 2°C approximately (Årsvoll & Larsen, 1977) and over Microdochium nivale with an optimum above 3°C (Lebeau, 1964).

Årsvoll (1973) found that the fungus caused significant injury in grassland in Norway only when there were more than 170-180 days of snow cover, including 90 ice days. Where there were more than 180 days of snow cover, including 110-120 days during winter, the damage was more severe than that from T. ishikariensis (q.v.). These meterological criteria usually restrict the occurrence of M. borealis to certain regions of higher land away from the coasts south of lat. 60°N. Where M. borealis commonly occurred, its incidence increased with increasing elevation, i.e. the opposite of abiotic winter injury.

Damage to grasses caused by M. borealis is reported to be usually more severe in the eastern and northern than in the western part of Hokkaido, the principal northern island of Japan (Sakuma & Narita, 1963).

Lebeau & Cormack (1961) considered that M. borealis was confined to extreme northern regions of North America such as Alaska (lat. 60°N) and the Prince George area (lat. 54°N), British Columbia. However, severe damage on Agrostis turf occurred in southern Saskatchewan, 50°N in 1971 (Smith, 1972).

S. borealis has also been reported from Agrostis golf turf in southern Manitoba (Allen et al., 1976), in southern Ontario, at lat. 44°N on Lolium perenne L. turf in 1979 (Smith, unpublished) and on golf green turf in northern Minnesota at lat. 47°N (Stienstra, 1974, 1980). Under favourable conditions it may be expected occasionally to cause problems in turf north of the Canada/USA border. In western Canada two epidemic seasons were noted in 1971/1972 and 1973/1974 (Smith, 1972, 1975, 1978). While the fungus has a boreal and sub-boreal distribution the climatic effects of continental land masses and altitude on temperature and snowfall may extend its range southwards, as for example to northern Washington (lat. 40°N) where it was found at 1200 m (Sprague et al, 1961), and Minnesota (Stienstra, 1980).

The increased incidence of disease due to M. borealis on peat compared with mineral soils (Arsvoll, 1973; Demidova, 1960; Ekstrand, 1955; Jamalainen, 1949; Yakovlev, 1939) may be largely due to pH effects on mycelial growth or sporophore development (see section on fungus and below).

Phosphorous nutrition considerably influences resistance to M. borealis. Tomiyama (1955) found that P deficiency increased severity of injury. Ekstrand (1955) noted that P applications increased resistance and Årsvoll and Larsen (1977) found that resistance to M. borealis increased significantly with increasing P. Tupenevich and Shirko (1939) noted that liming increased resistance while Ekstrand (1955) suggested that this effect may be due to release of P by the lime. Adequate N is needed to maintain plant vigor and encourage recovery of overwintering grasses from M. borealis (Jamalainen, 1957; Nissinen & Salonen, 1972; Sakuma & Narita, 1973). However, Årsvoll and Larsen (1977) found that resistance significantly decreased with increasing N. Ekstrand (1955) found that K occasionally increased resistance but Tomiyama (1955) and Årsvoll and Larsen (1977) found no consistent effect. Sakuma and Narita (1963) found that the relationship between the disease and nutrient status depended on soil type.

Control

The disease is likely to be a major problem in turfgrass only in continental North America north of the Canada/USA border and in northern parts of Norway, Sweden and Finland and in high snowfall regions or years. Its distribution in the USSR is likely to be similar to that in continental North America while in Japan only the heavy snow regions of Hokkaido appear to be at great risk.

No turfgrass species are completely resistant to M. borealis and there is little specific information on the comparative resistance of cultivars. However, there is good evidence that cultivars from higher latitudes are more resistant to winter injury, including snow mold damage than those from further south (Andersen, 1960; Jamalainen, 1974). The northern types should be

used where the disease is common. Poa pratensis is the preferred species for lawn turfs in northern regions of North America and some differences in resistance have been noted between cultivars. Except in the most severe outbreaks there is usually good recovery. Festuca rubra is susceptible and should not be used north of lat. 54° N in western Canada. This species, used in road verges in northern Canada, is often severely damaged. Lolium perenne appears to be susceptible, but its lack of cold hardiness eliminates its use in northern regions. No resistant Agrostis spp. or cultivars appear to be available although Colonial bent (A. tenuis Sibth.) seems less susceptible than Seaside or Penncross [A. palustris Huds. = A. stolonifera L., Smith (1974)]. Where the disease is common, fungicidal protection is essential on bentgrasses.

Although turfgrass of low vigor will be slow to recover from the disease, excessive N fertilization in the late summer and fall should be avoided. Soil P levels should be adequate. Where bentgrass turf has become very acid or is grown on peaty soil the low pH may encourage the disease. However, where the turf is acid the benefit of raising the pH with lime above neutrality to discourage M. borealis should be weighed against the possible risk of encouraging fusarium patch and take-all patch diseases.

Do not use snow fences around golf greens to trap snow where the fungus is common and attempt to speed the rate of drift melting by snow removal or the use of dark material spread on the drifts.

After a severe attack on bentgrass turf reduce the carry-over of sclerotial inoculum to the next season. Let the grass dry, thoroughly scarify the patches and pick up detached sclerotia with a vacuum sweeper. Repeat until few sclerotia are collected.

The use of quintozene (pentachloronitrobenzene) for the control of M. borealis in forages was pioneered by Finnish workers (Jamalainen, 1970; Ylimaki, 1955). This material was also effective in the control of the fungus on grasses in Japan (Sakuma & Narita, 1963) and Norway (Hansen, 1969). Kallio (1966) found quintozene (190 g a.i./100 m^2), thiram + phenyl mercuric acetate (112 g + 4.5 g a.i./100 m^2) and thiram (190 g a.i./100 m^2) applied to S. borealis-infected Poa pratensis turf resulted in percentage survival of 90, 80 and 78, respectively, while that untreated turf was 29 percent. Smith (1976) and Smith and Reiter (1976) found that benomyl (33 g), mercurous-mercuric chlorides (78 g), thiophanate-methyl (8 g), phenyl mercuric acetate (7 g) and quintozene (200-400 g a.i./100 m^2) were effective against slight to moderate infections with one or two applications in early and/or late autumn. Against very heavy infections (up to complete coverage with patches) the most effective materials were benomyl (31 g), thiram + carboxin + oxycarboxin (92-183 g), carbathiin (118 g) and quintozene (207 g a.i./100 m^2). The first application was made in September and the second in October. Chlorothalonil (107 g a.i./100 m^2) also gave effective control.

Stienstra (1974) found quintozene in combination with thiophanate-methyl effective against disease in golf green turf in Minnesota. In later studies Stienstra (1980) found that neither mercurous/mercuric chloride mixtures nor chloroneb controlled M. borealis. Where complexes with Typhula were concerned tank mixtures of chloroneb and quintozene, chloroneb and mercurous/mercuric chlorides, and quintozene with mercurous/mercuric chloride gave the best control.

Biological control of M. borealis with an Acrostalagmus species has been demonstrated experimentally, but practical biological control measures have not been developed (Pohjakallio et al., 1956).

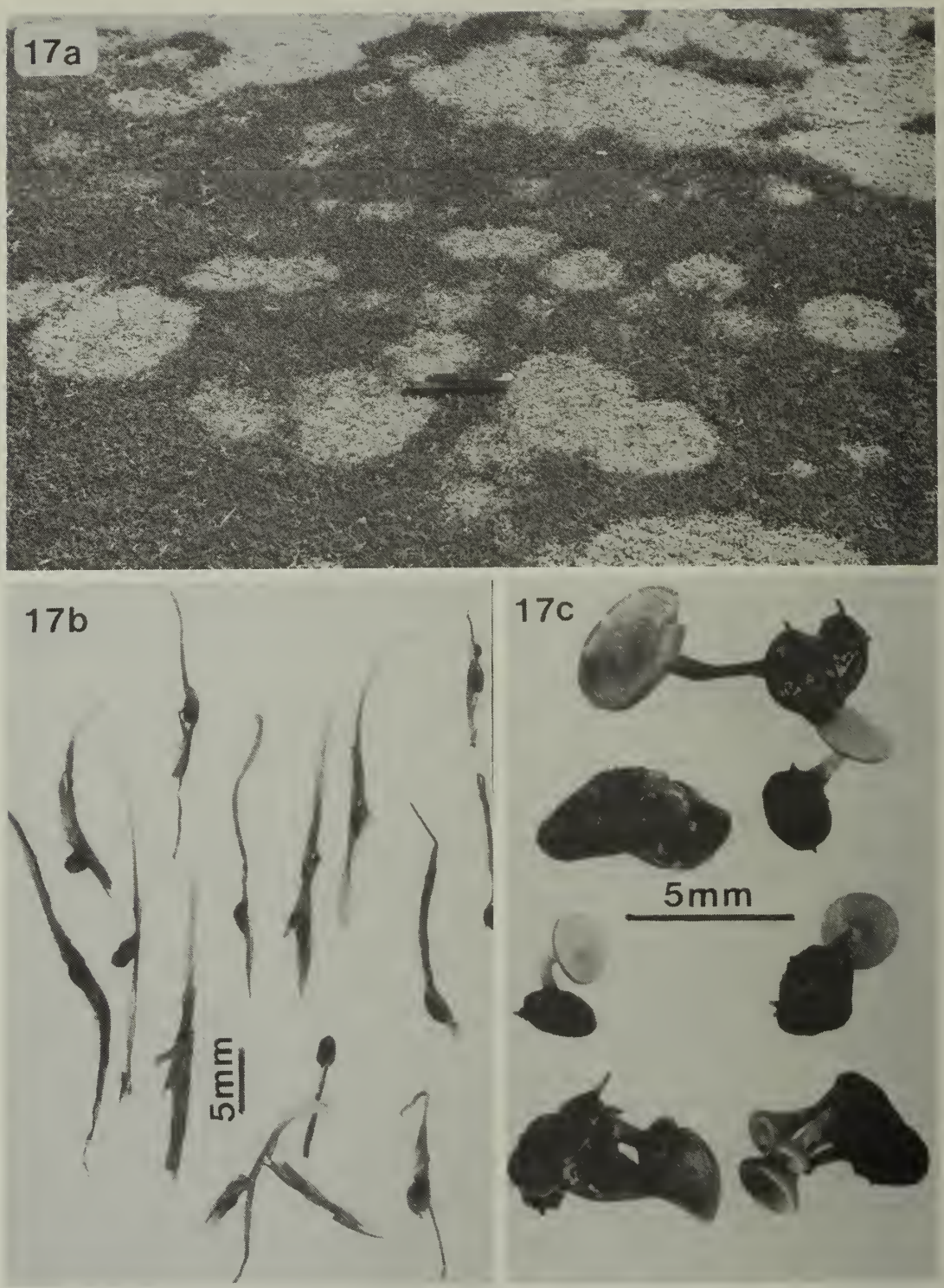

Fig. 17a. Bleached, coalescent patches caused by *Myriosclerotinia borealis* on *Poa pratensis* turf. Fig. 17b. Sclerotia of *M. borealis* attached to leaves of *Agrostis stolonifera.* Fig. 17c. Apothecia produced by sclerotia of *M. borealis.*

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Sclerotinia snow mold

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COTTONY SNOW MOLD - LOW-TEMPERATURE BASIDIOMYCETE (LTB) AND SCLEROTIAL LOW-TEMPERATURE BASIDIOMYCETE (SLTB)

In 1931 an unidentified low-temperature-tolerant basidiomycete (LTB) was found associated with a Fusarium sp. and a Rhizoctonia sp. causing snow mold of golf greens at Edmonton, Alberta (Broadfoot, 1936, 1938; Broadfoot & Cormack, 1941). Vanterpool (1944) isolated the same fungus from lawn turf in Saskatoon, Saskatchewan in 1932. Later, it was found on turf and forage grasses in Alberta and Saskatchewan (Cormack, 1948) and in Alaska (Lebeau & Logsdon, 1958; Sprague, 1962). A fungus closely resembling the LTB was isolated from winter rye at Whitehorse, Yukon Territories in 1964 (Traquair et al., 1983). Platford et al. (1972) and Allen et al. (1976) reported that the fungus was involved in snow mold disease in amenity turfs in southern Manitoba. It is the common cause of snow mold on all classes of sports turf and lawns, particularly in the lower snowfall regions of western Canada (Lebeau, 1960; Smith, 1969, 1972, 1973, 1974a, 1975, 1978, 1980, 1980a, 1980b), but it also causes damage in the higher snowfall regions in the Canadian Prairies. The pathogen, considered to have a geographical range restricted to western Canada and Alaska, is probably more widespread than this. It is an unspecialized pathogen with a wide host range on winter cereals, grasses, forage legumes, and weeds (Cormack, 1948; Lebeau, 1969) and causes pear decay in cold storage (Spotts et al., 1981). The fungus has two vegetative phases which are conspecific with Coprinus psychromorbidus (Redhead & Traquair, 1981; Traquair, 1980; Traquair & Smith, 1982) but which differ morphologically and pathogenically and are designated as the low-temperature basidiomycete (LTB) or the sclerotial low-temperature basidiomycete (SLTB).

COTTONY SNOW MOLD - Low-temperature basidiomycete (LTB) phase

Symptoms

The disease is first seen at snow melt in spring as bleached patches of turf. If there are few patches, these are often almost circular in outline and up to 1 m in diameter (Fig. 18a). Abundant mycelium is often present with a more luxurious fringe at the patch edge or the mycelium may be quite sparse and greyish-white (Fig. 18a). There are no sclerotia as with Typhula spp. and Myriosclerotinia borealis. The mycelium often disintegrates quickly on exposure. Where patches are crowded together, particularly on turf of Poa annua, they often do not coalesce and a green strip of undamaged grass is left between the patches giving a "crazy paving" effect (Fig. 18b). In mild cases, the disease may go unnoticed at snow melt, only becoming apparent when spring growth recovery of the turf takes place. Affected patches fail to "green up" like the remainder of the lawn. The significance of this is discussed below and under Typhula snow molds. Mycelium of the fungus, outlining patches of the disease, may often be seen after snow melt, on turf which was topdressed with soil or compost in the autumn. Under humid conditions, saprophytic fungi, notably Alternaria alternata Kiessler and Cladosporium herbarum Link, may colonize leaf tissues killed by the LTB giving a grey appearance to the patches.

Patches of Agrostis, Poa annua, and Festuca rubra turf, severely attacked by the LTB fungus, usually do not recover and the space left often fills in with Poa annua and broad-leaved weeds from seed lying dormant in the turf surface. In all except the most susceptible cultivars of Poa pratensis (Smith, 1980) recovery usually takes place, but it is sometimes as late as August before patches have healed up completely.

The causal fungus

Coprinus psychromorbidus Redhead & Traquair (Traquair)

By the use of a 'di-mon' mating technique (Breuhl & Kiyomoto, 1975) Smith (1977) showed that the LTB was probably not one of the common graminicolous Typhula spp. of the Prairies which had lost its ability to produce sclerotia. Many other (unpublished) attempts have been made by workers in western Canada to find a fruiting state of the fungus. Traquair (1980) found a small agaric of the Coprinus urticicola complex of the section Herbicolae on the necrotic crowns of alfalfa (Medicago sativa L.), dug from the field, subjected to a freezing test and allowed to recover in a greenhouse. By means of the 'di-mon' mating technique (Bruehl & Kiyomoto, 1975) he showed that the LTB and the Coprinus sp. were conspecific. The same fungus was found

on crowns of potted alfalfa and on plants in the field. In 1983, this fungus was found on sheaths of a potted plant of Agrostis stolonifera L. in a greenhouse at Saskatoon (Smith, unpublished; Fig. 18c). The species had been named Coprinus urticicola (Berk. & Broome) Buller (Hanna, 1939) and had been found in Manitoba in the Canadian Prairies on leaf sheaths of Marquis wheat (Triticum aestivum L.) and decaying stems of nettle (Urtica dioca L.). However, the name was incorrectly applied (Redhead & Traquair, 1981) and the fungus from alfalfa (Traquair, 1980) and the Agrostis stolonifera (Smith, unpublished) is C. psychromorbidus.

The caps of the fruits (basidiocarps) of C. psychromorbidus are 7-12 mm wide, conical to flat when mature with a narrow central boss (Fig. 18c). The edges of the cap are finely furrowed and when older they split and curl up. The cap surface is felted with curled back, orange-yellow to yellow-brown scales, the remains of a universal veil. There is a characteristic fungal smell. The gills are narrow, attached to a ring at the top of the stalk, grey at first, then blackening and deliquescing. The stalk (stipe) is 40-70 mm long and 2-3 mm wide when mature (Fig. 18c), usually slightly enlarged at the base with a hollow centre and watery, white fibrous flesh. The surface of the stalk has a fine, silky to faintly frosted surface. Buttons are at first pure white with brown scales developing later. When mature and deliquescing, HCN is evolved by the basidiocarps. The spore print is chocolate-brown to brownish-black. Basidiospores (6.4-) 7.2-8.8 (-9.6) X 4.8-5.6 (-6.4) um are pale brown or yellow-brown to blackish- brown when mounted in KOH or H_2O, smooth, elliptical to broadly elliptical in profile and generally elliptical or ovate in face view. They are thick-walled with an apical pore. Basidia are shortly club-shaped to almost sac-like, thin-walled, hyaline, 12.0-25.0 X 8.8-10.0 um and four-spored. There are short sterile basidial elements (brachybasidioles) surrounding the pleurocystidia bridging the gills and cheilocystidia at their edges. Clamp connections are present throughout the ground hyphae of the basidiocarp tissue. (From Traquair, 1980, modified).

In culture, isolates from spores or hyphae from basidiocarps are mesophilic, with optimum temperatures of 22°C. Growth is rapid at this temperature. The edge of the colony consists of the radiating mycelium which may be sparse, adpressed to the surface or cottony. The aerial mycelium is white, woolly or cottony, becoming resupinate or felted as it ages with white hyphal knots. The mycelium has a strong fungal smell. No fruiting has been seen in culture. HCN is produced on soybean agar after 3 weeks (Traquair, 1980). Hyphae are hyaline, thin-walled with clamp connections, mostly 1.6-4.0 µm in width. The hyphal knots consist of compactly interwoven, swollen hyphae 4.0-8.0 µm wide with oil droplets. The outer hyphae of these knots are thick- walled and narrower, 1.2-4.8 µm wide.

The fungus may be isolated from mycelium on leaves, crowns,

and shoots of turfgrasses by plating unwashed infected fragments onto potato-dextrose or potato-malt-yeast extract agar (BASM agar, Smith 1981) at 1°C. Alternatively, isolates may be obtained from mixed inocula on turf plugs by the process of eliminating other low temperature-tolerant fungi. These have sclerotia, as in Typhula spp. and Myriosclerotinia borealis or, in the case of Microdochium nivale the characteristic spores and pink mycelium develop when exposed to light (q.v.). The turf plug may be incubated in a glass or plastic moist chamber at circa 4°C under fluorescent or nuv light and then examined for a non-sporing, white basidiomycete with abundant clamp connections and cottony growth. Hyphae are similar in dimensions to those of mesophilic isolates from basidiocarps of C. psychromorbidus, that is, finer than in most Typhula spp., M. borealis and M. nivale, but the density of aerial growth is very variable. There are no sclerotia or spores and rarely rudimentary basidia are produced in culture (Smith, 1981).

Ward, Lebeau and Cormack (1961) grouped isolates of the LTB into types A, B, and C on their cultural appearance, supported by their reaction to temperature, pH, tolerance of antibiotics, their ability to produce HCN in culture and hosts, and their pathogenicity. Type A isolates were slow-growing, produced HCN in large quantities in plants and none in culture; they were moderately pathogenic on grasses and produced stroma-like bodies in culture (Smith, 1981; Ward, 1964a). Type B were more rapid growing than type A and produced abundant, fluffy, aerial mycelium and released much HCN in culture and were of equivalent pathogenicity to type A towards grasses. Type C was a heterogenous group of very fast-growing isolates which did not produce HCN and were not pathogens. The LTB isolate used by Smith in field fungicide and turfgrass resistance studies was highly pathogenic on grasses, winter cereals, and alfalfa (Smith, 1975, 1976, and 1980a). It was a type B LTB.

Broadfoot and Cormack (1941) found that the cardinal temperatures for the LTB to be -4, 15, and 26°C, and Smith (unpublished) noted that the optimum temperature for most turfgrass isolates was about 15°C. Ward et al. (1961) found that the optima for type A and B isolates was 12.5°C and for type C 12.5 and 17.5°C, but response to temperature was variable for types B and C. All isolates grew at 0 and 25°C.

HCN and pathogenesis

Lebeau and Dickson (1953) found that an isolate of the LTB produced sufficient HCN on natural and artificial culture media to kill buds and crown tissue of alfalfa. The production of HCN over a wide range of temperature suggested that it was produced during active mycelial growth rather than by autolysis of its own hyphae as with other basidiomycetes (Robbins et al., 1950). However, Ward & Lebeau (1962) later showed that while a type A isolate produced HCN during active growth, a type B isolate

appeared to produce HCN by autolysis. Lebeau and Cormack (1956) showed that turfgrasses infected with the LTB gave positive tests for HCN, whereas uninfected controls and turf samples taken in spring contained no toxin. Lebeau et al. (1959) found that alfalfa plants damaged by the LTB contained HCN in concentrations proportional to the severity of damage in the host. Ward (1964) showed that the LTB accumulates a cyanogenic compound in growing mycelium before free HCN could be detected and this compound breaks down chiefly during autolysis. Ward and Thorn (1966) found that HCN could be produced throughout the mycelial growth of the fungus from L-glycine. Apparently HCN is a normal metabolic product of many basidiomycetes (Bach, 1956; Loquin, 1944, 1947).

HCN is toxic to alfalfa buds and seedlings (Lebeau & Dickson, 1953) and it will also damage grass seedlings if present in sufficient concentration (Filer, 1964). However, it is uncertain whether it initiates pathogenesis in the LTB. Although Lebeau et al. (1959) stated that "mycelium was not present in crown bud tissues until positive tests were obtained for HCN" they also admitted that it was difficult to prove that plant tissues had absorbed HCN before invasion by the fungus. Their data also showed that HCN production was highest at the end of a winter's growth when autolysis would also be high (Lebeau & Cormack, 1956; Lebeau et al., 1959).

Ward et al. (1961) noted that type C isolates were highly compatible with other isolates in culture, but that in type A and B isolates "zones" were formed between adjoining colonies. These "zones", where ribbons of green grass show as boundaries between adjacent diseased patches, are often noticed in attacks of the LTB in turf and are often a useful diagnostic character (Fig. 18b). They appear to be due to mutual or intra-specific antagonism (Lebeau, 1975; Smith & Arsvoll, 1975). It seems highly unlikely that they are directly due to HCN. In addition, to its toxic effects on plants, HCN has been shown to inhibit the growth of several fungal species (Robbins et al., 1950). If the HCN produced by the fungus during growth was responsible for the initiation of pathogenesis and the death of the patches, the occurrence of the green ribbons would be unlikely since around patch margins the fungus is very active metabolically. It seems possible that the fungal attack on the green ribbons is prevented by a water-diffusible mutual inhibitor to LTB growth (Smith & Arsvoll, 1975). Grass in patch centres may have been killed by HCN resulting from autolysis of LTB mycelium.

Host range and cultivar resistance

The LTB has a wide host range which includes legumes, grasses, winter cereals, pears and some small fruits, vegetables, non-woody ornamentals, and weeds (Cormack, 1952; Spotts et al., 1981; Vanterpool, 1944). Cormack (1952) found most species of Bromus, Elymus, and Agropyron, including A. cristatum (L.)

Gaerten., often used in low-maintenance lawns in the Canadian Prairies, very resistant to resistant. Of the other turfgrasses, Poa pratensis varied from high to moderately resistant, but Poa trivialis L. was susceptible. Festuca rubra L. spp. rubra was often more severely damaged than F. elatior L.; F. rubra L. var. commutata Gaud. and F. ovina L. were susceptible. Phleum pratense was moderately susceptible. Agrostis spp. and Poa annua are often used in fungicide tests because of their susceptibility (Smith, 1976). However, some cultivars of creeping bent (A. stolonifera L.), e.g. Northland (Lebeau, 1967) are less susceptible than other Agrostis spp. Smith (1975, 1980, 1980a) found considerable differences in susceptibility to LTB in cultivars of P. pratensis, F. rubra, F. ovina, and F. duriuscula in field tests. A type B isolate (Ward et al., 1961), grown on sterile rye grain, was used as inoculum. Poa pratensis cultivars were generally more resistant than those of F. rubra and F. ovina. Susceptible lines often came from mild climates, where the range of pathogens was different from the Canadian Prairies. Many of the resistant lines came from the USSR or the eastern Baltic and Canada (Smith, 1975, 1980, 1980a). No strains completely resistant to the LTB were found, but selection for early winter dormancy promises to be of considerable value in increasing field resistance to LTB snow mold in Poa pratensis. The cultivar Dormie (Smith & Cooke, 1978) appeared to derive its field resistance from this characteristic.

Epidemiology

It is rare to find LTB snow mold severely damaging turf in the first or even the second spring after sowing (Smith, 1969). It takes at least that period for inoculum to build up. It is not clear in what form the inoculum survives over summer since there is no known sclerotial stage for the LTB, although there is for the SLTB which is conspecific (Smith, 1981; Ward, 1964a). Stromatal cushions are produced in culture by some type A isolates and these may be more resistant to unfavourable conditions than mycelium, but type B isolates which are found attacking turfgrasses are mycelial only. It is possible that the fungus persists as mycelium in plant debris (Lebeau & Cormack, 1961) and that the disease develops from mycelial infection. Once established, the disease appears to be very persistent, returning in the same turf location. Traquair (1980) and Traquair & Smith (1982) found that the cultural characters of isolates of C. psychromorbidus, which is conspecific with the LTB and the SLTB, ranged from the completely mycelial LTB, through forms with mycelial knots and cushions to the SLTB sclerotial form. Some SLTB isolates lose the ability to produce sclerotia on repeated subculturing, but other have retained the sclerotial character through 10 years of repeated subculture on "lean" medium (cornmeal, malt, yeast extract agar, Smith & Traquair, 1982). It may be that some LTB isolates have pleomorphic SLTB

oversummering stages. Turf of Poa pratensis, formed by seeding land that has been fallowed for several years rarely shows LTB injury until after the second winter unless inoculated (Smith, 1975). Cormack (1952) found that the pathogen did not show significant survival after 2 or 3 years when the land was kept free of a susceptible host. Lebeau et al. (1959) noted that the pathogen must be established (in alfalfa) in autumn to incite the disease. Deep snow covers are not so important in disease development as with snow molds such as Myriosclerotinia borealis and Typhula ishikariensis (q.v.). It is not necessary for the soil to be unfrozen for severe disease to develop (Cormack, 1948; Lebeau, 1964). These two factors may be of importance in pathogenesis in relation to the significance of HCN. Shallow snow covers would permit freer ventilation and escape of the toxicant more rapidly than deep ones, but the lower temperatures at the soil surface would probably favour absorption of the HCN by plant tissues (Lebeau & Dickson, 1955). Slow thawing at temperatures near freezing appears to favour disease development (Cormack, 1948). Nevertheless, LTB snow mold on turfgrasses is usually more severe in years when a persistent snow cover develops before the soil is fully frozen (Smith, 1980c; Fig. 3). In such years the likelihood of complexes of two or more snow molds developing is also increased (see Disease complexes). It seems probable that the LTB is a "low-grade" pathogen which needs to develop considerable amounts of mycelium by colonizing the dead host tissues before it can overwhelm living crown buds (Cormack, 1948; Lebeau & Dickson, 1955; Smith, 1980).

Heavy shading by trees and the liberal use of nitrogenous fertilizer and irrigation water increases disease severity (Smith, 1969), probably by delaying the acclimation of the turfgrass. Fall applications of processed sewage sludge and ammonium nitrate, even in combination with the fungicides chloroneb or mercuric chloride, increased susceptibility to LTB snow mold. A slow-release, crotonyl diurea fertilizer with the same fungicides gave excellent control with good spring colour. Untreated control plots had no snow mold but were of poor colour (J. B. Lebeau, pers. comm., 20 Jan. 1980). Applications of ammonium sulphate or a mixture of the latter and superphosphate applied when turf of Poa pratensis and Festuca rubra was nearly dormant in late autumn had no significant effect on the severity of LTB snow mold and plots greened up more rapidly in spring than untreated or no nitrogen plots. However, turf receiving phosphate only, as superphosphate, increased twofold the severity of the snow mold. Since the turf was almost dormant the increase may have resulted from the alteration of turf surface pH by the superphosphate or from a direct nutrient effect on the initial growth or subsequent metabolic activities of the LTB or competing organisms (Smith, 1977a).

Control

Cultural - The use of suitable resistant species and cultivars forms the main basis for cultural control. On minimum care turf in farm yards and on rough lawns crested wheatgrass, Agropyron cristatum (L.) Geartn. is probably the best choice since it is very resistant to LTB. It goes almost completely dormant in late fall but starts growth very early in spring. It may be used in a polystand with Poa pratensis if a denser turf is needed. On irrigated turf of domestic lawns, golf tees, fairways, and green collars, where LTB is prevalent a resistant cultivar of Poa pratensis is suitable. Many of the newer cultivars which are stated to have resistance to snow mold have not been tested for resistance to LTB snow mold. Until such time as they have been proven, resistant cultivars such as Dormie (Smith & Cooke, 1978), Delta and Park should be sown. Fylking, Barkenta, Golf, Sydsport, Cougar, and Merion which are susceptible (Lebeau, 1976; Smith, 1975, 1980a) should be avoided or used in low proportions in blends. There are no Festuca rubra or F. ovina cultivars comparable in resistance to that of the best Poa pratensis available and only Durar F. duriuscula has high resistance (Smith, 1975). Both Penncross and Seaside bentgrasses are severely attacked in golf greens, but the vegetatively propagated Northland A. stolonifera is often less severely damaged (Lebeau, 1967). Bentgrass greens in the Canadian Prairies should always be protected by fungicides against the LTB if invasion of P. annua into patches of bentgrass killed by the snow mold is to be prevented. Poa annua itself is very susceptible to LTB snow mold.

Management practices should be suited to the type of turf involved. Any major or minor nutrient lack is likely to be reflected in poor recovery from the disease. Particular care should be taken to adjust the amount of nitrogenous fertilizer applied after July to the state of grass growth. If inorganic nitrogen, such as ammonium sulphate (23-0-0) or ammonium nitrate (34-0-0), is applied after the end of July, use light applications (0.5 kg 100 m^2) and generally the total seasonal application should not exceed 2.5 kg/100 m^2. Inorganic nitrogen applied in September will delay dormancy and acclimation. Lebeau (1976) has found that the use of the slow-release, organic nitrogen fertilizer, crotonyl-di-urea (19-11-11) applied in combination with mercuric chloride or chloroneb fungicides resulted in good spring colour and good control of LTB snow mold. By making applications of nitrogenous fertilizer when lawn turf has gone "dormant" in early winter it is possible to elicit more rapid spring green-up. Ammonium sulphate (23-0-0) alone at 0.125 or 0.25 kg N/100 m^2 or in combination with superphosphate (20% soluble phosphoric acid) at 0.15 or 0.30 kg P 100m^2 did not significantly increase severity of LTB snow mold. However, phosphate alone should not be used since it doubled the severity of the disease (Smith, 1977).

There is no published experimental evidence on the effect of soil moisture on the severity of LTB snow mold. However, field

observations suggest that as in alfalfa (Cormack, 1948) the disease is less severe on turf watered deeply (to 15 cm) in autumn than on shallowly watered or on unwatered turf. Since LTB occurs in regions where desiccation injury is also a major problem in spring, adequate soil moisture, but not waterlogging, resulting in a deep root system is essential if the risk of poor recovery from snow molds and abiotic winter injury is to be reduced. Although the LTB will cause severe disease under shallow (10-15 cm) snow covers, long-lying snow drifts increase injury. Snow fences may sometimes be used to control the formation, position, and depth of snow drifts (Darby, 1971). It is sometimes expedient to remove snow mechanically from prone locations in late winter or spring or to dust black or dark coloured soot or fine fly ash to increase the rate of snow melt at that time (Dewey & Nielson, 1971; Sakuratani & Ishiguro, 1976). Soil heating with electrical resistance cables has been used effectively to control LTB (and M. nivale) attacks on very susceptible turf of Agrostis tenuis (Lebeau, 1964, 1967). Plots with minimum temperatures controlled at -3°C or unheated were damaged by the LTB, those controlled at 3 or 6°C were infected with M. nivale, but those maintained at a minimum of 0°C were unaffected by snow mold. The cost of installation and operation limits the use of the method.

Polyethylene and other synthetic plastic sheeting and netting and brush, compost, straw, or soil have been used to give protection to turf from cold injury and desiccation (Evans, 1973; Lebeau, 1964; Watson, 1969) in the northern USA and Canada. In southern Alberta, polyethylene covers increased the effectiveness of inorganic mercurial fungicides, allowing a much lower dosage to be used to obtain effective control of LTB snow mold. Topdressing appears to have little effect on disease.

Damage from LTB snow mold may be so severe on susceptible species and cultivars that it is necessary to returf or reseed the killed areas.

Fungicidal - Broadfoot (1936) obtained effective control of snow mold, considered to be caused by a complex of the LTB, a Fusarium sp. (probably M. nivale) and a Rhizoctonia sp. (probably a secondary organism) with mercuric or mercurous chlorides, singly or in combination. A single application of 400 g a.i./100 m^2 or greater gave effective control. Lebeau et al. (1961) also found mercury chlorides the most effective materials. No difference in efficiency was noted between mercuric or mercurous chlorides.

Where a very susceptible cultivar, such as Merion Poa pratensis is used, combinations of the inorganic mercury chlorides are still the most effective material (Allen et al., 1976; Smith, 1976). Wettable powder formulations of these are available in addition to dry powders, usually formulated in the proportion of 2:1 mercurous:mercuric chloride. The wettable powders usually give as effective control at lower dosages (80-110 g a.i./100 m^2) as the dry powders, but more than one

application is advisable in fall and early winter (Smith, 1976, 1980b).

Quintozene (275 g a.i./100 m^2), chloroneb (160 g a.i./100 m^2), and thiram + carboxin + oxycarboxin (275 g a.i./100 m^2) as wettable powders are very effective substitutes for inorganic mercury, except on very susceptible cultivars. At least two applications are required, in autumn and early winter (Smith, 1976, 1980b; Smith & Mortensen, 1981). Selection of a fungicide for LTB control should take into account complexes with other snow molds. Smith and Mortensen (1981) obtained effective control of LTB snow mold complexed with fusarium patch disease on bentgrass turf with three applications of triadimefon (62 g a.i./100 m^2), quintozene (200 g a.i./100 m^2), mercuric/mercurous chloride (47 g a.i./100 m^2), fenarimol (62 g a.i./100 m^2), thiram + carbathiin + oxycarbathiin (183 g a.i./100 m^2), and borax (375 g a.i./100 m^2). Combinations of three fungicides, even those regarded as effective against the LTB (see above) gave conflicting results. In another test, where the dominant snow mold was M. nivale with the LTB also present, combinations of three fungicides were generally more effective than single materials applied thrice.

COTTONY SNOW MOLD - Sclerotial low-temperature tolerant basidiomycete (SLTB) phase

An unidentified, sclerotial, low-temperature tolerant basidiomycete, designated as the SLTB, was found associated with snow mold damage to turfgrass from southeastern Saskatchewan to the Peace River region of British Columbia (Smith, 1972, 1974). It has also been found in spring, after snow melt, on diverse dying and dead hosts and on leaf and twig debris lying on the ground (Smith, 1981; Smith & Piening, 1980; Traquair & Smith, 1982).

Symptoms

Patches of snow mold-damaged turf are similar in shape to those caused by the LTB, but they are often smaller and injury less severe (Fig. 19a). The mycelium is white, cobwebby, stretching from leaf to leaf in longer turf, but particularly in that of close mown swards of golf greens or aprons, much of the mycelium is dense white or grey knots formed on leaves and litter (Fig. 19c). These cream- or tan-coloured knots develop into thick-walled brown or black sclerotia, irregularly-shaped, mostly 0.25-1.5 mm in size which are found mainly on the side of host tissues or plant debris nearest to the ground. Honey-coloured exudates may ooze from the sclerotia.

The fungus

Coprinus psychromorbidus Redhead & Traquair (1981)
(Sclerotial phase Traquair & Smith, 1982)

Methods of isolation

Fully imbibed sclerotia are surface-sterilized with 70% ethyl alcohol, rinsed in several changes of sterile water, sliced with a sterile scalpel and plated on cornmeal-malt-yeast extract agar (Smith, 1981) and incubated at 1°C in sealed containers until mycelium is produced. Subculture hyphal tips and grow on CMMY agar at 15°C. Cultural success is often low and is reduced further by more active surface sterilisants. If bacteria are troublesome contaminents, add 5% glycerol to BASM agar (Smith, 1981) and incubate sclerotial slices at -3 to -4°C until mycelium is produced.

Rate of growth in culture

Growth on BASM agar in growth tubes is more rapid (1.3-3.3 mm/d) than on PDA or malt agar (radial growth 0.67-1.7 mm/d). For most isolates the optimum growth temperature is from 10-15°C, but some isolates have a very low optimum of 0-5°C with a depression of growth rates between 3 and 5°C (Traquair & Smith, 1982).

Cultural characters

On CMMY agar (Smith, 1981) the colony margin is even, narrow, low-growing or uneven and with lanose mycelium. The aerial mycelium may be either sparse or lanose and felted. Growth is slower than that of the LTB. Compact hyphal knots are produced mainly on the surface appear from *c.* 4 weeks at 6°C. At first, these are cream or buff, changing as they develop to brown or black, hard, rounded or elliptical sclerotia up to *c.* 3 mm sometimes with pale brown exudates. At temperatures above 15°C a brown diffuse colour appears in the agar becoming more intense as the temperature increases to the maximum for growth of *c.* 25°C. On CMMY medium some isolates have continued to produce sclerotia for 10 years of successive transfers, but on other media isolates may lose this ability.

Anatomical characters of mycelium and sclerotia

In culture, hyphae are hyaline and thin-walled with clamp connections, frequently branching at the clamps from 1.8-5.0 um wide becoming tightly interwoven when lanose, submerged hyphae may be contorted, up to 5.5 um wide. Cystalline hyphal incrustations and intercalary swellings may be associated.

Sclerotial initials have hyaline mycelium with thin or only

slightly thickened walls. Clamps occur at cross-walls. Hyphae are frequently branched, tightly interwoven, fused or coalesced. Internal hyphae inflate and become irregularly shaped up to 8.0 um wide. As the sclerotial cortex darkens the hyphae of the medulla inflate further up to 15.0 um, rarely more, to take on an elliptical or irregular shape. Cell contents are oily. Cortical hyphae are 5.6-8.0 um wide, hyaline to yellowish, thick-walled, and angular in the layer nearest the medulla. The outer cells of the rind are angular and thick-walled, more compressed and compact than those of the rest with thick, dark-brown walls, but devoid of cell contents. When turgid and pricked under water the oily contents of the medullary cells are discharged into the water as a milky cloud. This is a good diagnostic feature for the SLTB as none of the other common sclerotial snow molds show the character.

Conspecificity with C. psychromorbidus

Single-spore, monokaryon isolates of C. psychromorbidus which were dikaryotized by dikaryon LTB isolates were also dikaryotized by SLTB isolates (Traquair & Smith, 1982).

Pathogenicity.

Foliar symptoms of snow mold damage caused by the SLTB are similar to that caused by LTB Coprinus, but there is less mycelium and the fungus is not as aggressive as the latter. The disease may be produced on turf by inoculating field plots with isolates grown on sterilized rye grain during late summer. It also developed on acclimated winter wheat grown in sterile soil in controlled environment cabinets inoculated with cornmeal-soil-sand cultures incubated at 1°C for 3-4 days and then a -3°C for 30 days. Foliar symptoms were similar to those caused by LTB Coprinus.

Although Agrostis spp. turfgrasses appear to be most severely damaged in the field by the SLTB Coprinus there is no detailed information on relative susceptibilities of other species and cultivars.

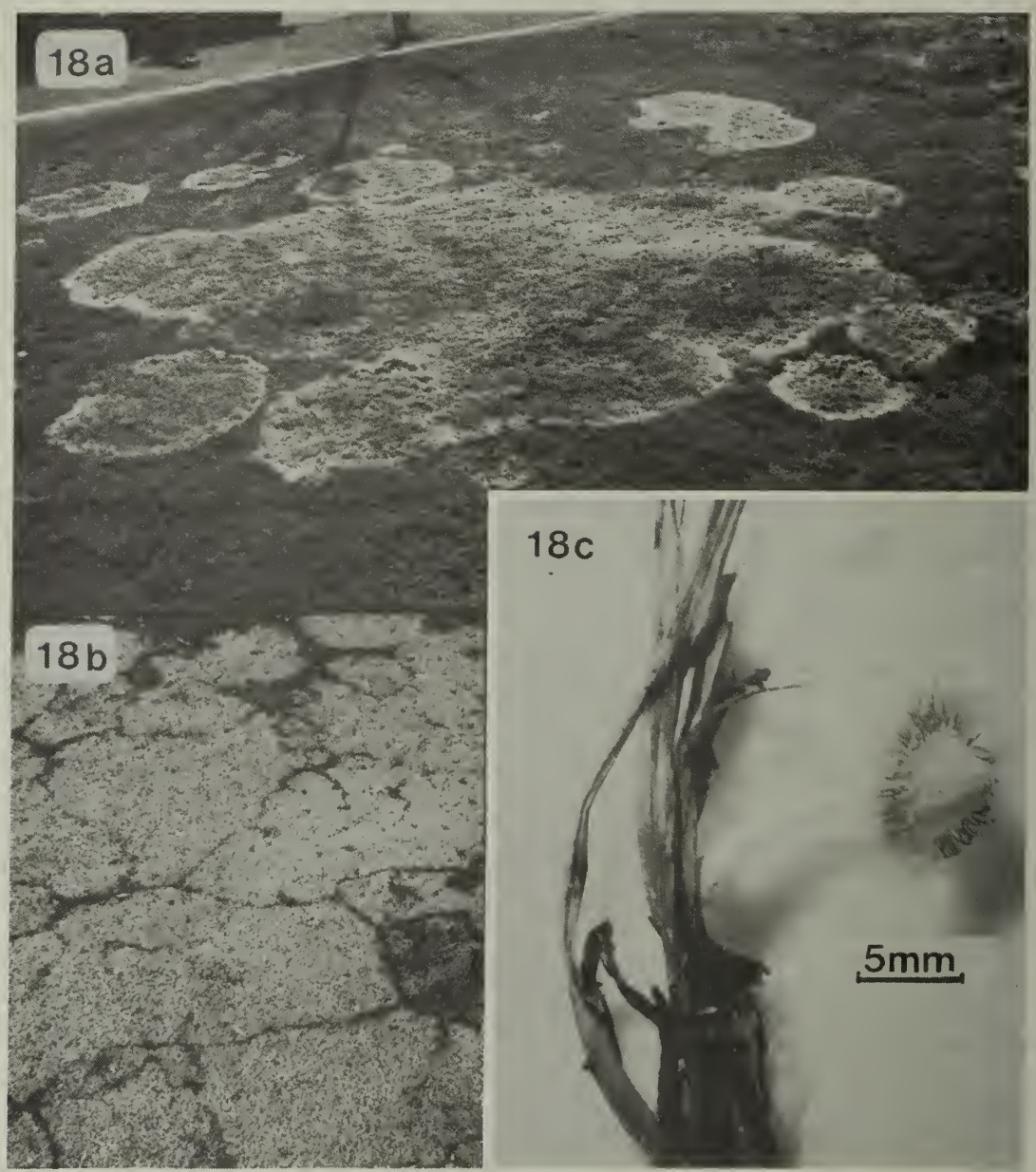

Fig. 18a. Patches of *Poa pratensis* turf after snow melt showing mycelium of the non-sclerotial phase of *C. psychromorbidus* (LTB). Fig. 18b. LTB patches on *Poa annua* turf showing "crazy paving" symptoms with unaffected green ribbons of grass between them. Fig. 18c. Sporophore of *Coprinus psychromorbidus* on a stolon of *Agrostis stolonifera*.

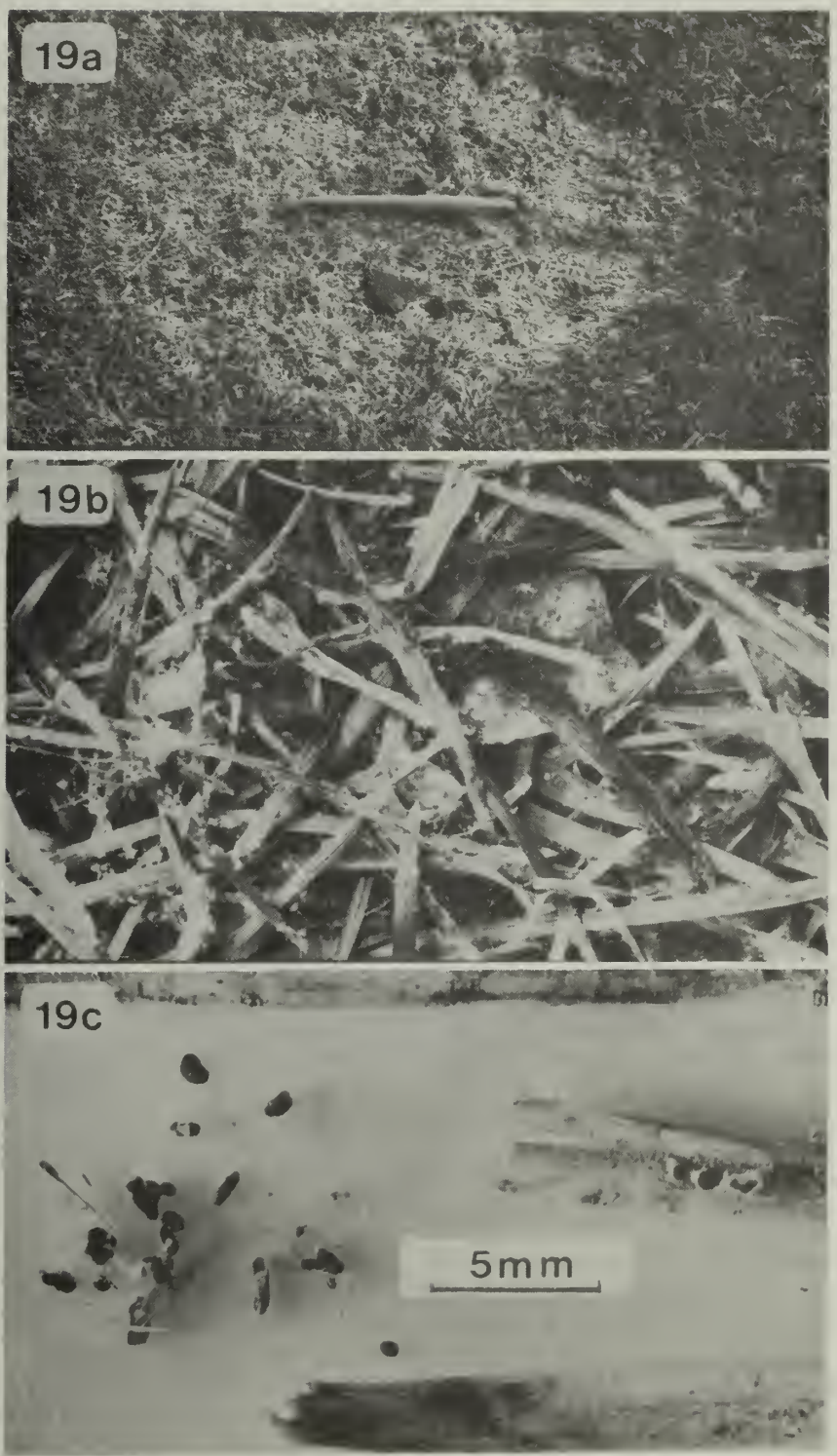

Fig 19a. *Agrostis* turf with the mycelium of the sclerotial phase of *C. psychromorbidus* (SLTB) after snow melt. Fig 19b. Cobwebby mycelium of the SLTB phase and the formation of knots and sclerotial initials on *Poa pratensis* turf. Fig. 19c. Sclerotia of the SLTB phase of *C. psychromorbidus*.

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BROWN ROOT ROT

According to Boerema and van Kesteren (1981) the fungus causing this disease, now named Phoma sclerotiodes (Preuss) ex Sacc., was first described from the decorticated roots and basal stems of an unidentified herbaceous plant from Silesia, East Germany as Plenodomus sp. It has a wide host range on plants from regions with severe winters in continental Europe and North America. It is particularly destructive on herbage legumes, notably sweet clover, Melilotus alba L. and alfalfa, Medicago sativa L. following the winter dormancy period in western Canada, Finland and the USSR (Boerema & van Kesteren, 1981; Cormack, 1934; McDonald, 1955; Mead, 1962; Rodigin, 1935; Salonen, 1962; Sanford, 1933). It may also be pathogenic on other plants, including grasses and cereals which have been exposed to low temperature (Henry & Berkenkamp, 1965; Knowles & Smith, 1981; Lebeau & Logsdon, 1958; Robertson, 1931; Smith, 1981, 1981a; Smith & Piening, 1980). Morochkovski (1933, cited by Boerema & van Kesteren, 1981) found it on sorghum in the USSR and Lebeau & Logsdon (1958) noted it on Festuca rubra L. and Poa pratensis L. in Alaska although they doubted whether it was a major pathogen. It was associated with damage to winter wheat in experimental plots in southern Alberta by Henry and Berkenkamp (1965) and with dead and severely damaged winter wheat and fall rye from Saskatchewan and Alberta in 1979 (Smith & Piening, 1980). Its protopycnidia have been collected frequently from snow mold-damaged turfgrass in western Canada since 1972 (Anon., 1979; Smith, 1981; Smith & Piening, 1980). It was involved in severe brown basal rot of overwintering Dactylis glomerata (Knowles & Smith, 1981; Smith, 1981a).

Symptoms

On dead and dying grasses and winter cereals there is a brown basal rot and root necrosis. Greenish-black, globular pycnidial initials (protopycnidia) up to 0.5 mm diam. are found firmly attached to roots to 10 cm at least below the soil surface. Occasionally these are also found on stems of dead hosts, mainly at or near ground level and on crown tissues. Necrosis is very noticable where protopycnidia are attached.

The fungus

Phoma sclerotiodes (Preuss) ex Sacc.
syn: Plenodomus sclerotiodes Preuss (nomen nudum)
Plenodomus meliloti Markova-Letova
Plenodomus meliloti Dearn. & Sanford
Plenodomus sorghi Morochkovski
Plenodomus karii Petrak

For detailed discussion of synonymy see Boerema & van Kesteren (1981). The fungus was described independently in the USSR and Canada as P. meliloti and then twice again as a new species in the USSR and Finnish Lappland (Boerema & van Kesteren, 1981).

The sclerotia-like protopycnidia (Fig. 20a) (Smith & Piening, 1980) or pycnosclerotia (Boerema & van Kesteren, 1964) are greenish-black, subglobose to conoid, either single or clustered. On roots of grasses they may be slightly elongate and are dished on the adaxial surface where there is a very short attachment process. Maximum diameter of these protopycnidia is approximately 0.8 mm. At this stage there are no locules (Smith, 1981). Mature pycnidia are similar in colour and shape to protopycnidia, but tend to be more hemispheric and they are solitary or confluent, the latter especially in culture, up to 2 mm in groups (Fig 20b). The pycnidial wall, which in protopycnidia is thin and delicate, becomes much thickened as the pycnidia mature. The wall cells are usually 5- to 6-sided, 5-10 um in diam.; cavities of these cells become filled as they age. Locules develop in the ground tissue of the pycnidia at scattered points, the spores being discharged through beaked ostioles up to 200 um long as a cream or yellow cirrus (Fig. 20c). The spores are single-celled, hyaline, elliptical, biguttulate (4-) 4.5-6.5 (-8) X 2-3 (-3.5) µm (Fig. 20d). Conidiophores are cone-shaped and insignificant, formed round the periphery of the locule. In culture the mycelium is greyish-white, darkening to brownish-white with the pycnidia mostly superficial (Boerema & van Kesteren, 1981; Colotelo & Netolitzsky, 1964; Dearness & Sanford, 1930; Smith, 1981).

Isolation and culture of the fungus

Detach protopycnidia from roots with needles or use a blender run at slow speed to remove them from plant base or root tissues. Surface sterilize with 70% alcohol for 30 sec. Wash in several changes of water and plate onto cormeal + malt extract + yeast extract agar and incubate at 1°C. There is no need to split the protopycnidia with a sterile scalpel as the mycelium will grow out from the superficial cells. If bacterial contaminants are troublesome plate the "sterilized" protopycnidia onto BASM agar containing 5% glycerol and incubate at -3 to -4°C. If protopycnidia on pieces of plant tissue are incubated in a moist chamber for several weeks at 15°C, pycnidia and spores will develop. From the latter single cell isolations can be made. The optimum temperature for mycelial growth of most isolates from grasses and cereals is about 15°C. Sanford (1933) found the temperature range for mycelial growth to be 0-27°C with an optimum of 15-17°C. However, most isolates make appreciable growth at -7°C.

Pathogenicity

Pathogenicity on grasses and cereals has not been proven experimentally, although it is often the predominant fungus (Lebeau & Logsdon, 1958). In winter cereals it is often found in complex with the SLTB phase of Coprinus psychromorbidus (Smith & Piening, 1980 and unpublished). Sanford (1933) found that leguminous hosts were susceptible only during winter and spring dormancy stages, although dead Avena sativa L. roots were colonized saprophtically. However, not all grasses and winter cereals bearing protopycnidia of P. sclerotiodes are dead, so susceptibility in these plants may also be related to the dormancy state.

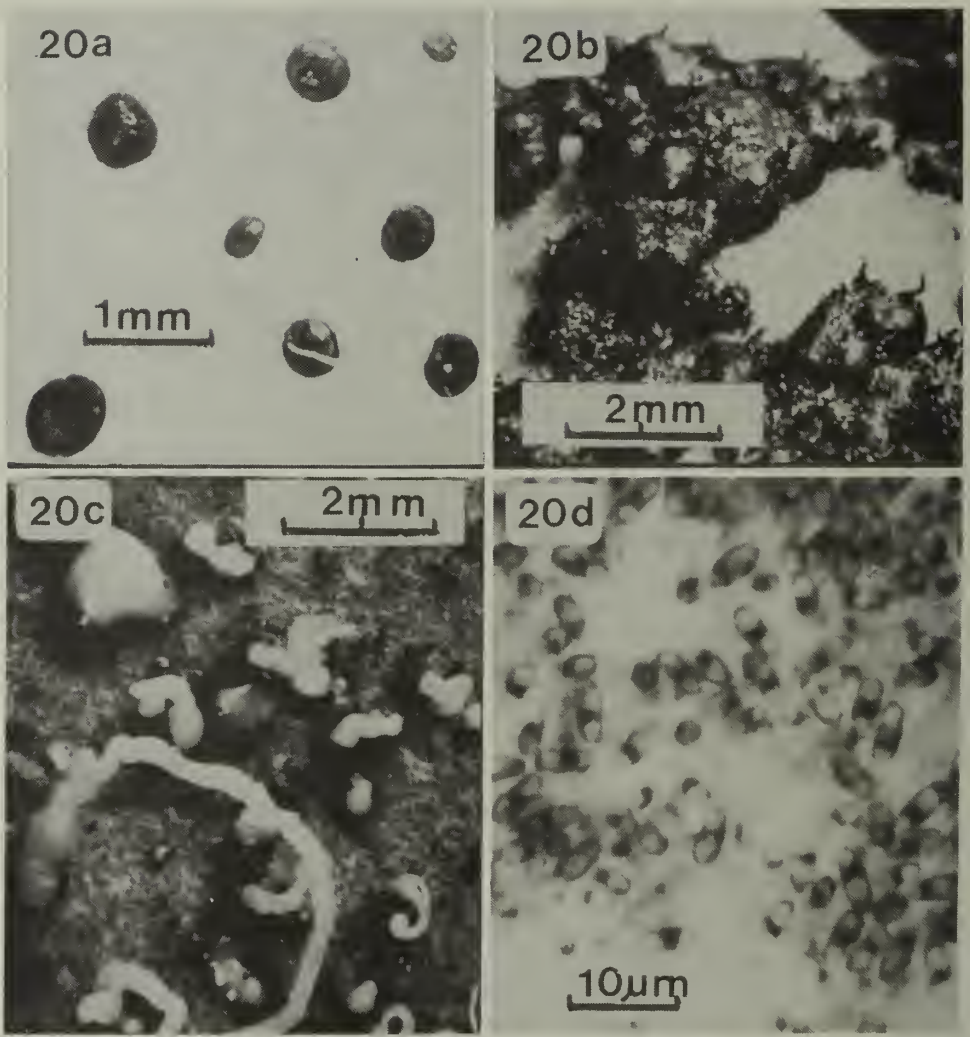
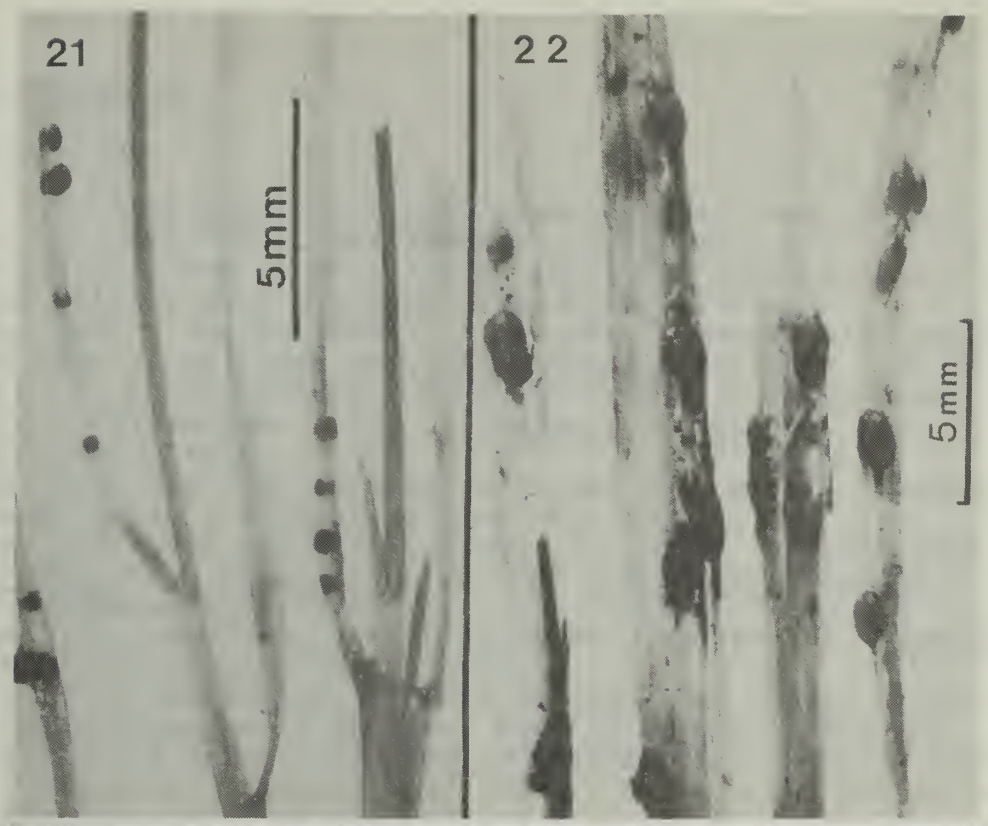


Fig. 20a. Protopycnidia of *Phoma sclerotiodes* detached from turfgrass. Fig. 20b. Pycnidia of *P. sclerotiodes* with protuberant ostioles — from culture. Fig. 20c. Cirri of spores discharged from pycnidia. Fig. 20d. Unicellular, biguttulate spores of *P. sclerotiodes.*



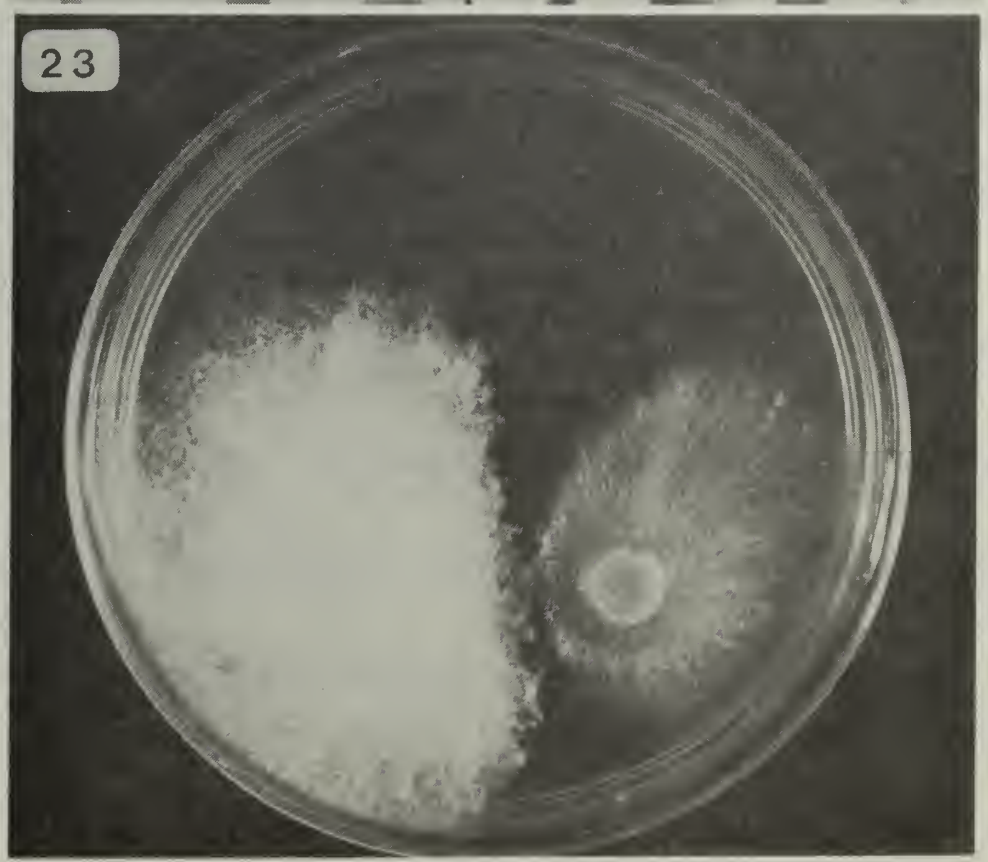


Fig. 21. Sclerotia of a non-psychrophilic fungus, resembling *Sclerotium rhizoides* on leaves of *Festuca longifolia.* Fig. 22. Sclerotia-like stromata of *Acremonium boreale* (*Nectria tuberculariformis*) on grass stems. Fig. 23. Antagonism (barrage effect) between two monokaryotic isolates of *Typhula incarnata.*

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FROST SCORCH, LEAF ROT, TIP BLIGHT, OR STRING-OF-PEARLS DISEASE

This disease has been reported on grasses and winter cereals from northern and eastern Europe. North America and northern Japan (Anon., 1931; Appel, 1933; Baudys, 1928, 1930; Bokura, 1926; Cash, 1953; Davis, 1933; Ekstrand, 1938; Flachs, 1935; Freckmann, 1919; Haskel, 1928; Howard et al., 1951; Hungerford, 1923; Kreitlow, 1942; Matsumoto, 1928; Mikolajska, 1974; Muhle, 1953; Pape, 1926; Remsberg, 1940; Remsberg & Hungerford, 1933; Sampson & Western, 1942; Smith, 1980; Sprague, 1955; Stirrup, 1932; Stout, 1911; USDA Index of Plant Diseases, 1960; van Luijk, 1934; Vang, 1945; von Ottingen, 1934). It may be locally severe.

Symptoms

Infected plants may be stunted. Attacked leaves wilt, curl, or roll, according to species, and bleach almost white. Leaf tips may be narrowed to tendril-like form in species which normally have expanded leaves, and leaf bases may remain green. Sclerotia form in bead-like rows on the withered leaves (Kreitlow, 1942; Stout, 1911).

The fungus

Sclerotium rhizoides Auersw. (Botanische Zeitung 7:294. 1849).

The sclerotia, borne superficially on the leaves may be oval, spherical, or oblong (Ekstrand, 1938; Stout, 1911) from 1-5 mm varying in size according to host. At first they are white or grey, darkening to almost black, becoming rough. While in severe cases they are so abundant as to give a string-of-pearls appearance, in others they may be solitary on the leaves or in leaf axils (Kreitlow, 1942). The mycelium is white or grey, septate, coarse branched, without clamp connections. No fruits have ever been found (Baudys, 1930), naturally or in culture. The fungus grows well at 16°C (Stout, 1911) but three isolates of a similar fungus studied by Smith (unpublished, and see below and Fig. 21) were not psychrophilic. Sclerotia similar to those on grasses form in culture on lima bean agar or on the surface of sterilized bean pods (Stout, 1911). The fungus may be isolated by placing fragments of infected leaves on lima bean agar or potato-dextrose agar (Stout, 1911). Smith (unpublished) isolated a Sclerotium sp. from turf and meadow grasses (see below and Fig. 20) which may be S. rhizoides, by plating slices of surface-sterilized sclerotia (see Typhula spp. for method) onto BASM (potato malt) agar (see Typhula, Pages 80). Attempts to obtain infection of leaves by mycelium were unsuccessful (Stout, 1911). Smith (unpublished) failed to obtain infection of seedlings of Poa pratensis L. and Phleum pratense L. grown in

sterile culture inoculated with mycelium of Sclerotium sp. although sclerotia formed on root and shoot systems (Fig. 20 and see below).

Some of the reports of the disease from Norway and the USA (Davis, 1933; Ekstrand, 1938) appear to have been due to attacks by Typhula spp. (mainly T. incarnata Lasch ex Fr. - synonyms T. graminum auct. non Karst. and T. itoana Imai) according to (Ekstrand, 1938; Remsberg, 1940). Other reports are doubtfully of S. rhizoides (Anon., 1931; USDA Index of Plant Diseases, 1960). T. graminum and Sclerotium rhizoides were regarded by some workers as identical (Haskell, 1928), but this was disproved by Baudys (1930) and Jorstad (cited by Ekstrand, 1938). Stout (1911) found that S. rhizoides from Calamagrostis canadensis in Wisconsin was similar to the fungus from European sources, on a wide range of grass hosts including Poa pratensis. The first description of the fungus appeared in 1849 (reviewed in the Botanische Zeitung 7:294 in 1849) and the Latin description is quoted by Stout (1911). Smith (1980) isolated sclerotial fungi (Fig. 21) resembling S. rhizoides from Festuca longifolia in turfgrass plots at Agassiz, British Columbia, from Poa pratensis in a lawn at Saskatoon and from an unknown grass at Carigill, near Alston in the Pennines in England.

Host range and susceptibility

In turf, Agrostis spp. (Ekstrand, 1938; Flachs, 1935; Kreitlow, 1942; Stirrup, 1932; van Luijk, 1934) Poa pratensis (Kreitlow, 1942; Stout, 1911) and fine-leaved Festuca spp. (Appel, 1933; van Luijk, 1934) are attacked by S. rhizoides. There is no information on specific or varietal susceptibility in turf grasses.

Epidemiology

The mycelium is systemic and perennial. In the USA the disease becomes most conspicuous during April and May. The mycelium develops in the growing points and leaves are successively infected before they expand. The unaffected basal part of the blade becomes flattened out and the area is covered with mycelium just below the point where the next leaf in succession expands. The sclerotia are produced along the infected portion of the leaves or from the mycelium at the leaf base. Sclerotia mature in May and when ripe drop from the leaves with attached short strings of dry mycelium. They may germinate to produce mycelium, but their fate is uncertain. Infected leaves appear at the same time as sclerotia from the previous season are present on the ground. The mycelium is perennial in the underground parts of the plant and may be a soil inhabitant. Infection of the aerial parts of the plant comes from these underground tissues (Stout, 1911).

The disease is said to be favoured by a long snow cover

(Haskell, 1928), but there are conflicting opinions about the relationship of the disease to soil moisture. Oettingen (1934) observed that the disease does not occur on areas subject to regular spring and winter floods. Stirrup (1932) reported that it occurred on Agrostis spp. on an upland meadow where rainfall was usually high. Mikolajaska (1974) found it was an important disease on river meadows needing drainage. Stout (1911) studied the disease in marsh meadows in Wisconsin.

Control

Improve fertility and raise soil pH by liming for it seems probable that the disease is more severe where fertility (Oettingen, 1934; Stirrup, 1932) and soil pH are low (Stirrup, 1932). Howard et al. (1951) suggest collection of clippings when mowing diseased areas to remove inoculum from upper leaves and the use of a mercury fungicide if the disease is severe.

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Nectria tuberculariformis

The sclerotia of the anamorph of this fungus, the hyphomycete Acremonium boreale Smith & Davidson (teleomorph Nectria tuberculariformis - see below) is common on grasses and many other plants in Canada from British Columbia to Ontario. It has been found on dead stems of herbaceous plants (Fig. 22), in the Swiss Alps and also on forage legume debris in Norway (Samuels et al., 1984; Smith, 1972, 1973, 1974a, 1974b, 1981; Smith & Davidson, 1979). The sclerotia of A. boreale are frequently collected with those of other sclerotial snow molds such as the SLTB phase of Coprinus psychromorbidus, Myriosclerotinia borealis, and Typhula spp. from patches of turfgrass damaged by snow molds in western Canada. It is weakly pathogenic towards unhardened grasses at 0 and 3°C (Smith & Davidson, 1979). Its main ecological significance seems to be as an invasive primary saprophyte on a wide range of plant substrates. Its antagonism to snow molds and other fungi suggests that it may play a significant role in determining the nature and intensity of damage in snow mold complexes (Smith & Davidson, 1979). The low-temperature-tolerant Nectriella muelleri Samuels, Rogerson, Rossman & Smith, which has an Acremonium anamorph, another Nectriella sp. with an Acremonium anamorph and Hyponectria sceptri (Karsten) Samuels, Rogerson, Rossman & Smith, anamorph unknown are sometimes associated with Nectria tuberculariformis in the Swiss and Austrian Alps. They also inhibited the growth of some mesophilic plant pathogens (Samuels et al., 1984). Ascomata of the teleomorph, N. tuberculariformis, were found on the surface of typical sclerotia of the anamorph, A. boreale, on Cirsium spinossissimum in the Swiss Alps by Samuels (Samuels et al., 1984) in July 1978. N. tuberculariformis was originally found on the same plant host in the Austrian Alps by Winter (see below) and later reported from North Dakota by Seaver (1909)[1].

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[1]What appeared to be rudimentary ascomata developed on sclerotia of A. boreale collected at Krabol in Norway on forage legume debris in October, 1974 by J. D. Smith. These did not mature.

The fungus

Nectria tuberculariformis (Rehm ex Saccardo) Winter, Rabenh. Kryptogamen Fl. Deutschl. Osterr. und der Schweiz 2 Afl. p 118. 1887. For synonomy see Samuels et al. (1984).
Anamorph: Acremonium boreale Smith & Davidson, Can. J. Bot. 20:2138. 1979.

The sclerotia-like stromata (Fig. 22) are scattered on stems, leaves, bark, and overwintered seeds, 0.1-2.0 mm long X 0.1-1.0 mm wide X 0.5-1.0 mm high in the middle, lenticular in surface view, pulvinate in longitudinal section; white at first, becoming bright orange, finally light brown and hard, occasionally green with superficial algal growth; pale orange internally; subepidermal and remaining so or becoming erumpent by longitudinal splitting of the substrate epidermis; easily removed from the substrate of some hosts, but often firmly attached to subcortical tissues of gramineous hosts; remaining sterile for long periods, composed of compacted textura epidermoidea, cells containing drops of orange pigment, walls 1.0-1.5 um thick. The mycelium in the host or substrate is systemic.

Under cool, moist conditions, a continuous layer of conidiophores develop from portions of the stromal surface; they are erect (7-) 20-30 um long X 1.0 um wide at the tip and 1.5 um wide at the base, hyaline, smooth, and branching. Conidiogenous cells are cylindrical to subulate, orthophialidic, monoblastic, terminal and arising as lateral branches of the main axis, often immediately subtending a terminal phialide and not delimited from the main axis by a septum, 7-16 (-30) um long, hyaline with a smooth, slightly thickened, but not flared collarette.

Cream, pink, orange, or orange-red masses of gloeoconidia may develop on the stromata. Conidia are unicellular with densely staining regions at each end, heteropolar, straight, polysymmetric, oblong to elliptic, sometimes waisted centrally, with or without a protuberant, flattened, basal abcission scar, hyaline, produced in basipetal succession and held in cream to orange slime. They measure (4.5-) 5.0-6.3 (-7.5) X 1.5-2.0 um from natural stromata but with a larger size range from culture (Samuels et al., 1984; Smith & Davidson, 1979).

The perithecial ascomata arise from part of the stromal surface in caespitose groups of 4-15, with bases immersed in stromal tissue. They are white to orange, globose 170-260 um diam., non-papillate with flattened ostiolar areas and covered with fine white hyphae. The ostiolar area appears as a shining bright orange dot against a light, dull background. The ascomatal wall is 30 um wide laterally, of hyphal elements which, in longitudinal section, appear irregular to elliptic in outline with lumena 5-8 X c. 0.2 um. Adjacent cell walls are shared. On the outside of the perithecium the hyphal cells have many free ends, but at the base there are confluent with the stroma cells. The ostiolar canal is periphysate.

Asci are unitunicate, cylindrical to narrowly clavate, (40-) 49-69 (-78) X (5-) 7-9 um, with a refractive ring at the apex. The ascus base is broadly pedicellate with a pore surrounded by a refractive ring at each point corresponding to the septum between the tip, penultimate and basal cells of the crozier. Asci arise in a hymenium on the lower ascomatal wall and are 6 to 8 spored with ascospores biseriate above and uniseriate below or biseriate throughout.

Ascospores are elliptic to elliptic fusiform (8.0-) 8.8-11.8 (-13.5) X (2.0-) 2.5-3.7 (-4.6) um, equally 2-celled, non-constricted at the septum, each cell containing one or more orange drops, wall smooth, hyaline.

Pseudoparaphyses were not seen (Samuels et al., 1984).

Growth in culture. The fungus is very slow growing in culture even at its optimum temperature which, for 5 isolates on cornmeal, malt extract, yeast extract agar, CMMY, ranged from 9-18°C. Appreciable growth in colony diameter was made at -6°C and good growth took place at 0°C. On "lean" media, such as CMMY, the peripheral mycelial is usually submerged in the agar and the colony centre may be waxy or individual sclerotium-like stromata may develop, especially in tube cultures, at temperatures below 10°C. Aerial mycelium is sparse, but occasionally conidia develop on the stromata on conidiophores which are similar to those produced on "natural" stromata. Conidia are produced in basipetal succession and congregate in heads at the tips of free conidiophores or "slime-down" on the stroma.

Sporulation occurs from -3 to 20°C and spores germinate from -2.5 to 20°C, but at the upper end of the range a high proportion of spores become pear-shaped or spherical and fail to produce germ tubes or swell and burst.

Isolation. Use fully imbibed sclerotia as for M. borealis. Plate disinfected slices on CMMY medium and incubate at 0°C in a sealed container until mycelium is produced. Transfer to CMMY and incubate at 6-15°C. Stromata developing on this medium may produce conidiophores bearing conidia from which further cultures can be made.

Pathogenicity tests. For details of pathogenicity tests with this and other low-temperature fungi of low virulence see Arsvoll (1975) and Smith & Davidson (1979).

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OTHER LOW-TEMPERATURE-TOLERANT GRASS PATHOGENS

Bruehl et al. (1966) reported that R. Sprague isolated fungi associated with winter cereals, mainly wheat, during the cold months of December 1959 to April 1960 and December 1960 to April 1961 to determine whether there were any unrecognized winter pathogens of importance. Fifty-five species of fungi were identified; many of these also occur on grasses. Low-temperature isolates of these were tested for pathogenicity in cold chambers at 1-3°C. Although M. nivale, T. incarnata, and T. ishikariensis var. idahoensis were pathogenic under these conditions only a Rhizoctonia sp. of the others showed some pathogenicity. Broadfoot (1936) found that a Rhizoctonia sp. was associated with turf damage during late fall and early winter in central and southern Alberta and Traquair and Smith (1981) collected sclerotia of Rhizoctonia solani from patches of diseased turf in spring in Saskatchewan and northern Alberta. Some of the isolates showed mycelial growth optima at about 20°C, lower than summer isolates.

Arsvoll (1975) determined the temperature/growth responses and the pathogenicity of fungi isolated from grasses suffering from winter injury. Seedlings of Phleum pratense L. raised from sterilized seeds were grown in sterile culture in test tubes on Hoagland's agar. Some were acclimated at low temperature and others were not. The plants were then inoculated with the test fungi in mycelial plugs and incubated at 0 and 3°C and the damage assessed. Those fungi which proved pathogenic were then tested as mycelial macerates of broth cultures on greenhouse-grown grass plants of seven species at the 4-6 leaf stage in pots using the technique of Blomquist & Jamalainen (1968). They were incubated for 2 months at temperatures of 0-5°C (average 3°C) and the damage assessed. In the temperature/growth response study, of the 33 species, 19 grew at -6°C, 10 at -3°C, and 4 at 0°C. At 0°C none of the fungi had a mycelial growth capacity, expressed as percentage of growth at their optimum growth temperature, as high as Myriosclerotinia borealis, which was 80%, Typhula ishikariensis var. ishikariensis, 61%, or T. incarnata, 27%. Microdochium nivalis had only a 6% growth capacity of its optimum, less than that of 13 other fungi which are not recognized as snow molds. At 3°C, 11 of the "non-snow molds" showed over 20% of optimum growth capacity. In the test tube pathogenicity studies, where the disease assessment scale was 0 = no attack to 4 = very severe attack, not even the recognized snow molds caused severe damage after 4 weeks on acclimated plants at 0°C, but Laetisaria fuciformis (McAlp.) Burdsall Corticium fuciforme (Berk.) Wakef., 2.6, Fusarium avenaceum (Corda ex Fr.) Sacc., 2.5, M. nivale, 2.9, T. ishikariensis var. ishikariensis, 2.8, and a sterile hyphomycete, 2.6, caused moderately severe injury on non-acclimated seedlings. After 8 weeks, 14 "non-snow molds" caused moderate to moderately severe damage on acclimated

seedlings. These species included: Ascochyta phleina Sprague, 2.6, Ascochyta sp., 3.5, Cercosporella herpotrichoides Fron., 2.3, Coniothyrium cerealis, E. Muller, 2.0, Corticium fuciforme, 2.4, Epicoccum purpurascens Ehrenb. ex Schlecht., 2.4, Fusarium avenaceum, 3.0, Fusarium culmorum (W. G. Smith) Sacc., 2.4, Fusarium equiseti (Corda) Sacc., 2.5, Hendersonia culmicola Sacc., 2.4, Mortierella hyalina (Hartz) W. Gams, 2.3, Mycocentrospora acerina (Hartig) Dayton, 2.5, Phoma eupyrena Sacc. sensu Wollenw., 2.0 and a sterile hyphomycete, 2.7. T. ishikariensis and M. nivale caused very severe injury, of 4 and 3.9 respectively, but T. incarnata caused only moderately severe, 2.7 and M. borealis, although ascospores were used as inoculum, only slight, 1.4, injury on acclimated seedlings. In the pot test on eight grass species incubated at 0-5°C, Agrostis tenuis, Phleum pratense, Festuca rubra, and Poa pratensis suffered most severely, while Lolium perenne, Dactylis glomerata, and Bromus inermis were least affected by the "non-snow molds". Festuca pratensis was intermediate. On the latter species, C. fuciforme, Dactylaria graminicola Arsvoll, Fusarium avenaceum, Fusarium equiseti, the sterile basidiomycete and hyphomycete were almost as pathogenic as the recognized snow molds.

Although pythium snow rot or snow blight has not been reported on turf or other grasses in North America, damage is caused on winter wheat in northern Japan (Ito & Toykunaga, 1935; Iwayama, 1933; Tomiyama, 1961), Washington State (Bruehl et al., 1966; Lipps & Bruehl, 1977, 1978, 1979), North Dakota (Stack et al., 1979) by Pythium spp. A Pythium sp. was isolated from diseased winter wheat in southern Alberta at snow melt in early April (Smith & Piening, 1980). Although Pythium spp. have not been studied as low-temperature pathogens on turfgrasses, one of them Pythium graminicola Subrm. (syn. P. aristosporum Vanterpool or P. arrhenomanes Drechsl.) with a wide host range on grasses and cereals (Sprague, 1950) has been shown to be pathogenic on winter wheat under snow rot conditions by Lipp and Bruehl (1978). A new species, P. okanagense P.E. Lipps has been isolated from wheat beneath snow in Washington (Lipps, 1980). Pythium snow blight shows a different epidemiological pattern from other snow molds.

Humidity under a snow cover appears to be a most important factor determining the localization of snow molds (Tomiyama, 1961). In ridge cultivation of winter wheat in northern Japan Iwakiri (1946) showed that Microdochium nivale was the most prevalent pathogen on the highest ridges, Typhula spp. on the middle ones and Pythium spp. on the lowest ones. In pythium snow blight, Hirane (1955) showed that infection through leaves was more important than through roots. Lipps & Bruehl (1978) isolated Pythium aristosporum, P. iwayama S. Ito, P. ultimum Trow. and a Pythium sp. at 1°C from wheat plants with rotted leaves and crowns and browned roots from under snow and ice covers in eastern Washington. P. iwayama and P. aristosporum grew more rapidly at 0.5°C than T. ishikariensis var. idahoensis

or *M. nivale*. Both *Pythium* spp. were pathogenic on wheat at 0.5°C, but required different moisture regimes. *P. iwayama* failed to cause rot at 8-15°C, but *P. aristosporum* was highly pathogenic at those temperatures. *P. ultimum* was invasive of roots and leaves but did not incite snow rot and the *Pythium* sp. was associated with ice injury. Lipps and Bruehl (1978) considered *P. awayama* and the *Pythium* sp. as true snow rot pathogens, but *P. aristosporum* and *P. ultimum* were not. The injury to winter wheat by *Pythium* spp. causing snow rot was increased when it was overfertilized with nitrogen (Hirane, 1955; Iwakiri, 1946; Iwayamia, 1936).

There is a need for further studies on low-temperature-tolerant fungi which are not now regarded as snow molds. Although they do not usually cause obvious symptoms like the recognized snow molds under a snow cover, they may cause considerable damage at the low temperatures prevailing in winter before a permanent snow cover develops and in spring at and after snow melt. Since many of them are mesophils they may be able to establish themselves before snow mold attacks develop and become important competitors for space and nutrients with the regular snow molds.

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SNOW MOLD COMPLEXES AND COMPETITION

While it sometimes happens that snow mold disease of turfgrass is caused mainly by one fungal pathogen, often a diverse group of species, such as Microdochium nivale, and/or the LTB and SLTB phases of Coprinus psychromorbidus, Myriosclerotinia borealis, Nectria tuberculariformis, Phoma sclerotiodes, Typhula spp. and vars. occurs in single or overlapping patches of snow mold (Fushtey, 1975, 1980; Smith, 1974, 1976, 1978). Other "non-snow molds" such as Rhizoctonica spp., Cladosporium herbarum, Fusarium spp. and miscellaneous leaf and crown pathogens and saprophytes may also be present (Broadfoot, 1936; Smith, unpublished; Traquair & Smith, 1982). It can often be determined with reasonable certainty which low-temperature fungus is or was dominant if sclerotia are present, but it is often obscure how associated mycelial fungi modified the incidence and severity of the disease. While the "non-snow molds" may not have been involved in the snow mold injury, they may have been important in the fungal succession leading up to the snow mold attack. Where one low-temperature pathogen is responsible for the injury it is probable that it was able to compete successfully for the supply of nutrients, space, moisture, or susceptible plant tissues (Matsumoto & Araki, 1982; Matsumoto & Sato, 1982). This is direct competition. A snow mold pathogen may also compete in a broader sense in having survival structures such as sclerotia, resting mycelium, chlamydospores, or oospores with differing longevity. The organisms may have different tolerances to drought, desiccation, moisture, heat, and chemicals which affect their competitive saprophytic ability (Garrett, 1956). But commensal and symbiotic relationships also occur between snow molds and their associates, such as occur between other soil organisms (Brian, 1960), although little is known about these. One of the mechanisms by which successful competition may be achieved is antibiosis (Clark, 1965; Park, 1960).

Microorganisms compete for possession of plant residues which can be regarded as energy or raw material in different states of vitality (Bruehl, 1975). The more vital, the less likely are the saprophytic organisms able to colonize and take possession. Energy can be passively possessed, stored within resting structures, such as the sclerotia of many snow molds, e.g. Typhula spp. Sclerotium rhizoides and the SLTB stage of C. psychromorbidus, the oospores of Pythium spp. or protopycnidia of Phoma sclerotiodes. Where there are no true dormant structures, there is little energy storage and the fungus dies out when readily available substrate is dissipated. One snow mold, Microdochium nivale, seems to be of the "combination possession" kind (Booth & Taylor, 1976). The mycelium of the fungus attacks seedling tissues, thoroughly penetrating them. It invades aerial parts of grasses, sporulating on dead tissues during moist

periods in the growing season and after snow melt - the "pink snow mold" stage. The spores are quite short-lived and can only be regarded at infective agents (Smith, 1953). However, some recent observations suggest that the fungus may persist as an endophyte (Petrini et al., 1979). Since the pathogen has no chlamydospores other workers have suggested that it persists through unfavourable periods as plectenchymatic aggregates in plant residues (Bennett, 1933) for considerable periods of time (Booth & Taylor, 1976; Colhoun, 1970). In temperate climates the disease may be found as a pathogen on turfgrass at every season of the year (Smith, 1953) so resting structures do not seem too important in survival in this mesophilic snow mold. In continental climates conditions suitable for active disease development are usually presented only in late summer, autumn, early winter, and spring and for "slow disease" under the snow. M. nivalis has "an edge" over its competitors, i.e. the sclerotial snow molds. In cool, moist weather it can cause disease at higher temperatures than they can and so gains possession of the substrate. When temperatures are lower, it is at a competitive disadvantage with, say, T. incarnata, since the mycelial growth capacity of M. nivale at 0 and 3°C is only 8 and 18% compared with 27 and 50% of that of T. incarnata at their respective growth temperature optima (Arsvoll, 1975). Although M. nivalis is a mesophil, it is still capable of slow growth at -6°C. Once the disease is established as fusarium patch in autumn it has established possession of tissues, and using them as a base it persists and develops slowly under the snow.

Matsumoto and Sato (1982) showed that T. incarnata had excellent competitive saprophytic ability, CSA, but that of T. ishikariensis was poor. This low (CSA) of the latter was compensated by greater virulence. Japanese biotypes of T. ishikariensis showed differences in virulence (Matsumoto et al., 1982). Matsumoto and Araki (1982) have produced evidence that M. borealis infects leaves which sustain cold injury and are subsequently snow covered. Hence snow mold tended to predominate in the first half of the winter. By contrast, T. incarnata began to invade the host, starting with the senescing lower leaves and progressing upwards when these younger tissues lost vitality because of tissue respiration under a prolonged snow cover in the latter half of the winter. The pathogenic niche of T. incarnata was secure because of its excellent CSA. Matsumoto and Araki (1982) considered that T. ishikariensis occupied a position between M. borealis and T. incarnata. Whether these conclusions can be drawn for Canadian conditions seems uncertain.

M. borealis, which can cause disease when the tissues of grasses are frozen, has an advantage over Typhula spp. that are slower growing at lower temperatures. At 0°C the mycelial growth capacity of M. borealis is 80% of that at its optimum, that of T. ishikariensis var. ishikariensis and of T. incarnata is 27% (Arsvoll, 1975). The effect of freezing of the plant tissues is to increase the osmotic pressure of the cell sap which M.

borealis tolerates better than Typhula spp. (Bruehl & Cunfer, 1971; Tomiyama, 1955; Volk, 1937). T. ishikariensis var. canadensis probably has an advantage over T. incarnata, with which it occurs in heavy snowfall areas of British Columbia (Smith, 1974) in having a much lower optimum temperature for mycelial growth (Arsvoll & Smith, 1978). This may also account for the infrequent occurrence of disease caused by T. incarnata in Saskatchewan (Smith, 1974, 1978) which has lower temperatures in winter and a lower snowfall than the regions in British Columbia (Potter, 1965) where T. incarnata occurs. The lower snow cover in Saskatchewan results in less modification of the severe winter air temperature (Smith, 1981).

Sclerotia of snow molds such as M. borealis, Typhula spp., the SLTB phase of C. psychromorbidus and the low-temperature antagonist, Acremonium boreale remain dormant during spring, summer, and early autumn. If they did not do so, the energy reserves which give them a competitive advantage over mycelial, low-temperature fungi would be wasted since they are unable to invade plant tissues at temperatures higher than a few degrees above freezing. These fungi require moist conditions and prolonged low temperatures before their sclerotia will germinate. M. borealis and Typhula spp. also require light, and in the case of the latter, light of a specific wavelength (Remsberg, 1940; Tasugi, 1935) before they will produce sporophores freely. None of these fungi have a pronounced dormant period once their sclerotia have parted company from their hosts or matured in culture (Arsvoll, 1976; Arsvoll & Smith, 1978; Smith & Davidson, 1979; Traquair & Smith, 1982). Typhula spp., the SLTB phase of C. psychromorbidus and A. boreale will germinate myceliogenically in the dark if given adequate moisture and prolonged cool temperatures, but M. borealis usually will not. The effect of low temperatures and chilling on oversummering sclerotia has not been examined to the same extent as on overwintering sclerotia (Coley-Smith & Cooke, 1971). The role of associated microorganisms in the "sclerotiosphere" in suppressing sclerotial germination in T. ishikariensis var. idahoensis was shown by Huber & McKay (1968), but the associated bacteria did not antagonize growing mycelium.

Different snow molds may be found colonizing the same plant (Arsvoll, 1975; Ekstrand, 1955) or piece of turf (Smith, unpublished). Ekstrand (1955) noted that infection by M. nivale came from the seed or the soil and the fungus grew upwards and from the upper parts grew laterally. In the case of Typhula spp. or M. borealis, which are active later in the season, infection, he suggested, started mainly from spores on the upper parts of the plant and from these infection points grew downwards to the lower parts. This is doubtful in the case of Typhula spp. since infection in most cases seems to result from mycelium produced by the germination of sclerotia lying on the ground. Matsumoto & Araki (1982) found that M. borealis prevailed on upper leaves, while T. incarnata developed best on lower to upper

leaves on grasses in Sapporo in northern Japan.

Ekstrand (1955) noted that antagonism between snow mold species occurred to a lesser or greater extent when two different ones were inoculated on the same plate of culture medium and incubated at temperatures between -5 and 10°C. Little antagonism was noted between M. nivale and the others since its abundant aerial mycelium overgrew the boundaries between colonies at all temperatures between -2 and 5°C. Antagonism was apparent between M. borealis and T. borealis (T. ishikariensis) from -5 to 10°C, between M. borealis and T. incarnata from -2 to 5°C and between T. borealis and T. incarnata between -5 and 10°C. This was apparently an antibiotic effect. Smith and Davidson (1979) noted similar effects between the snow mold antagonist Nectria tuberculariformis (Samuels et al., 1984) and M. nivale, T. incarnata, T. ishikariensis vars. ishikariensis and canadensis at temperatures of -3 to 10°C. The SLTB phase of C. psychromorbidus was little affected. Isolates of N. tuberculariformis varied in antibiotic activity (Samuels et al., 1984; Smith & Davidson, 1979) but their mycelial growth was not apparently affected by the snow mold pathogens. Tomiyama (1955) reported antagonism between M. borealis and T. incarnata in culture and suggested that this might play an important role in determining their distribution.

Ekstrand (1955) suggested that antibiotic effects noted between snow molds in culture supported the view that primary infection of a plant with one fungus prevented a secondary attack by other fungi. This does not always seem to hold true in the case of turf diseases. For example, M. borealis will overgrow patches of disease caused by T. ishikariensis var. canadensis and the latter fungus will overgrow turf damaged earlier by M. nivale. Intraspecific competition (mutual antagonism) between colonies of isolates of the Ascomycete snow molds M. borealis and M. nivale seems slight, but that between different isolates of the basidiomycetes C. psychromorbidus (LTB phase), Typhula incarnata, T. ishikariensis vars. ishikariensis and canadensis has been found (Arsvoll, 1976; Lebeau, 1975; Smith & Arsvoll, 1975) (Fig. 23). In pathogenicity tests in controlled environments it was shown that when macerated dikaryotic isolates of either T. ishikariensis var. ishikariensis or var. canadensis were mixed and used as inoculum there was significantly less severe disease than when the isolates were used separately (Smith & Arsvoll, 1975). It was later shown that mixtures of highly aggressive isolates of either T. ishikariensis var. ishikariensis or T. incarnata from different geographic regions of Norway were of much lower pathogenicity than when used separately. T. ishikariensis var. ishikariensis showed considerable mutual antagonism between isolates from four grassland fields and even from within one square metre plots in the fields. This showed considerable intra specific and intra varietal variability. On the other hand, no reduction in pathogenicity occurred when four isolates of M. nivale or M. borealis were mixed (Arsvoll, 1976).

When adjacent patches of snow mold caused by the LTB phase of C. psychromorbidus grow towards each other they often do not merge, but remain like "crazy paving" with ribbons of green grass between them. This occurs on both fine putting green turf and on coarser turf of lawns and it appears to be due to mutual antagonism (Smith & Arsvoll, 1975) (Fig. 18b). Lebeau (1975) found that mixing pathogenic isolates of C. psychromorbidus (which also causes winter crown rot of alfalfa) resulted in lower virulence and mycelial growth of the mixture. It has been suggested (Colotelo & Ward, 1961) that the production of HCN in alfalfa plants infected by the LTB of C. psychromorbidus was due to the beta-glucosidase activity of the fungus acting on the cyanogenic substrates in the host. Pathogenesis was described by Lebeau (1966) as being associated with the accumulation of toxic amounts of HCN in host tissues. Strains of the fungus differ in their cultural characters, pathogenicity (Ward et al., 1961), and ability to liberate HCN in culture and host plants:

Type A Highly virulent, slow growers which produce no HCN in culture, but release large amounts of HCN in interaction with plants.

Type B Less virulent than A, grow rapidly, produce large quantities of HCN in culture, but smaller amounts in the host.

Type C Non-pathogenic, rapid growers, produce no HCN in culture or in conjunction with the host.

Although mixing of pathogenic isolates reduced fungal virulence it had no significant effect on the production of beta-glucosidase, so no apparent relationship seemed to exist between pathogenicity and the secretion of the enzyme. However, virulence was restored and production of beta-glucosidase resumed when an avirulent isolate was mixed with a virulent strain (Lebeau, 1975).

The biochemistry of the mutual antagonism shown by Typhula spp. and C. psychromorbidus is not understood. However, antagonism is usually ascribed to staling substances or antibiotics excreted by the fungi into their substrate or volatile products which they elaborate. These metabolites accumulate to the extent that the growth of the fungus producing them stops or is restricted. HCN is an inhibitor of respiratory enzymes (Hutchinson, 1973) which has been shown capable of killing plant seedlings. While it is produced by many fungi, (Bach, 1976; Filer, 1964; Lebeau & Cormack, 1961) Lebeau and Cormack (1961) were unable to show its production by T. ishikariensis var. idahoensis. Mutual inhibition of LTB colonies in turf which is frequently seen is unlikely to have resulted from the action of HCN produced by the adjacent colonies under the snow unless high concentrations of the toxicant were being produced by actively growing mycelia at the patch margins. Under such conditions the death of the intermediate ribbon of green

grass would be expected also. In the reduction in pathogenicity in the studies with the Typhula spp. (Arsvoll, 1976; Smith & Arsvoll, 1975) it seems most likely that the mutual inhibition of the pathogenic isolates was due to a water-diffused inhibitor (Fig. 23). Since macerated mycelium was used as inoculum, competition between cells or groups of cells of the mixed inoculum may have occurred, reducing viability or aggressiveness. This may have been due to the production of metabolites or staling substances, which were mutually inhibitive to the different isolates. It is also possible that one or more of the isolates with lower pathogenicity than the other and with faster growth was able to compete more effectively for living space on the host tissues. Considerable variability in pathogenicity, in rate of growth and antibiotic activity occurs in both dikaryotic and monokaryotic isolates of graminicolous Typhula spp.

At present is it uncertain how the mutual antagonism between isolates of basidiomycete snow molds might be used to control the diseases they cause, as has been possible in the case of fairy rings caused by Marasmius oreades (Smith, 1980a). However, it is possible to start snow molds of turfgrasses on a field scale by inoculation with cultures of the fungi grown on sterile grain (Smith, 1975; Smith & Reiter, 1976). It may be possible to use cultures of isolates of suitable species which will antagonize or outgrow wild strains in regions where the diseases are endemic. The use of mixed isolates of basidiomycete snow molds in pathogenicity tests may produce irregular results because of mutual antagonism (Lebeau, 1975; Smith & Arsvoll, 1975).

The onset of near-dormancy in turfgrasses in autumn or winter is shown by a decrease in leaf and shoot production and the senescence of tissues. In their moribund state they may be colonized by mesophilic primary saprophytes such as Cladosporium herbarum (Pers.) Link, Alternaria alternata (Fr.) Keissler, Epicoccum nigrum Link, Leptosphaerulina and Pleospora spp. Leaf death and chlorophyll degeneration follow. Mesophilic colonists may modify leaf substrates needed by the cold-tolerant snow molds which follow them (Blakeman, 1971) and also antagonize them (Fokkema, 1976). The selection of a turfgrass which becomes dormant or the witholding of nitrogen which may hasten onset of quiescence may improve field resistance through alteration of the ecological succession of organisms on leaves and shoots (Hudson, 1968). A study of these aspects appears to hold promise for the development of biological methods for the control of snow molds of turfgrasses (Smith, 1975, 1980).

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LIST OF ILLUSTRATIONS

produced on sclerotia.

Fig. 18. (a) Coprinus psychromorbidus (LTB or cottony snow mold, non-sclerotial phase). Disease patches after snow melt showing cottony mycelium at margins. (b) LTB patches on Poa annua turf after snow melt showing "crazy paving effect" with intervening ribbons of green, healthy grass. (c) Fruit of C. psychromorbidus on stolon of Agrostis stolonifera.

Fig. 19. (a) A patch of diseased Agrostis turf with the mycelium of the sclerotial phase of C. psychromorbidus (SLTB) after snow melt. (b) Cobwebby mycelium of the SLTB phase and formation of knots and sclerotial initials on turf of Poa pratensis. (c) Sclerotia of the SLTB phase of C. psychromorbidus.

Fig. 20. (a) Protopycnidia of Phoma sclerotiodes detached from turfgrass. (b) Pycnidia of P. sclerotiodes with protuberant ostioles. (c) Cirri of spores discharged from pycnidia. (d) Unicellular, biguttulate, stained spores of P. sclerotiodes.

Fig. 21. Sclerotia of a non-psychrophilic fungus, resembling Sclerotium rhizoides on leaves of Festuca longifolia.

Fig. 22. Sclerotia-like stromata of Acremonium boreale on grass stems.

Fig. 23. Antagonism (barrage effect) between two monokaryotic isolates of Typhula incarnata.

Table 6 Snow molds of turfgrasses - Distribution, Predisposition and Control - Summary

Disease and distribution[1]	Predisposing factors	Cultural control	Fungicidal treatments[2]
Fusarium patch and pink snow mold (*Microdochium nivale*)[1] Boreal, sub-boreal and temperate regions and at higher elevations in warmer regions of Europe, North America, Asia and Australia. The snow mold with the widest climatic (geographical) range because of its mesophilic and psychrophilic characteristics.	Unbalanced or excess nitrogen fertilizer, particularly if applied late in the growing season. Alkaline turf surface or high surface moisture. Cold, humid weather or late autumn/ early winter temporary snow covers. Slow spring snow melt. Turf warming in winter from house basement, pipes, ducts or heating cables. Covers or mulches to protect turf from frost. Susceptible species (or cultivars of generally resistant ones) i.e. *Poa annua*, *P. trivalis* and *Agrostis* spp. *P. pratensis* may be damaged, but seldom killed.	Let turf "harden off" with balanced, but not excessive or late-season nitrogen fertilizer, keep mowing while leaf growth continues. Remove clippings and fallen leaves. Open up hedges and other dense windbreaks. Break up dew by switching or poling. Replace susceptible cultivars or species with more resistant ones. Control establishment or ingress of *P. annua*.	Control disease before growth slows down in autumn or patches may remain until spring. Depending on region, this may require the application of fungicide from late summer onwards. Several applications, preferably of differing materials until a permanent snow cover develops, or where complexes occur to reduce risk of turfgrass pathogens developing resistance. Benomyl, mercurous + mercuric chlorides, phenyl mercuric acetate, phenyl mercuric acetate + thiram, oxycarboxin + thiram + carboxin, chlorothalonil, quintozene, iprodione and methyl thiophanate are generally effective.

Table 3 Snow molds of turfgrasses - Distribution, Predisposition and Control - Summary (cont.)

Disease and distribution [1]	Predisposing factors	Cultural control	Fungicidal treatments [2]
Grey snow mold (Typhula incarnata) Temperate to sub-boreal climates in Europe, Asia, and North America, but less common in more extreme climates in continental interiors, i.e. Prairies of Canada and higher snowfall regions of more northerly latitudes, probably due to competition from other fungi more tolerant of lower temperatures. Often in complex with C. nivalis and/or T. ishikariensis vars.	Excessive or unbalanced nitrogenous fertilizer especially in organic form if applied late in the growing season. Cultivars which are not winterhardy. Most Poa pratensis and Agrostis stolonifera cultivars are susceptible. Poa annua is very susceptible.	Allow turf to harden off in autumn for winter as for M. nivalis. Ensure adequate, balanced but not excessive or late nitrogenous fertilization. Use winterhardy cultivars of northern origin. Control ingress of P. annua.	Fungicides should be applied in fall or early winter before the development of a permanent winter snow cover. In some less snowy regions, i.e. co areas, where T. incarnata is the main snow mold fungus, only one fungicide application is needed. Timing of the application is not critical in places where T. incarnata is the principal component of complexes. Where T. ishikariensis is present in significant amounts one fungicide application is inadequate. Effective fungicides are: mercurous + mercuric chlorides, phenyl mercuric acetate, chloroneb, quintozene, cadium succinate, chlorothalonil, mancozeb and thioallophanate in combination with other materials.

Table 8 Snow molds of turfgrasses - Distribution, Predisposition and Control - Summary

Disease and distribution[1]	Predisposing factors	Cultural control	Fungicidal treatments[2]
Speckled snow mold (Typhula ishikariensis) vars.) ishikariensis - Northern Scandinavia, northern Japan, northern North America. May be expected on turfgrasses in Iceland, higher elevations in northern Europe, Switzerland, the USSR and northern Asia since it has been reported on grasses or other species from these regions. idahoensis - arid grassland areas in Washington, Idaho, Utah and Montana in the USA, northern Japan. Reports on var. from other regions not confirmed - may be vars. ishikariensis or canadensis. Lower snowfall areas than var. ishikariensis or canadensis. canadensis - moderate to heavy snowfall regions in western Canada, particularly with long snow covers, eastwards to Ontario and Minnesota, northern Japan.	Longer and colder winters than favour T. incarnata, but similar fertility conditions to the latter. Most species and cultivars are susceptible, but especially those of southern origin, and when excessive nitrogen and irrigation delays the onset of winter dormancy.	Similar to T. incarnata, but snow drift control is especially important under long snow covers. Remove snow from fine turf or speed melting with dark topdressings. Use cultivars with high winterhardiness especially those derived from local ecotypes. Ensure adequate soil phosphorous status.	T. ishikariensis vars. are less responsive to control by some fungicides effective against T. incarnata and several autumn and early winter applications may be needed. Effective fungicides are: chloroneb, quintozene, phenyl mercuric acetate, with and without thiram, carboxin + thiram + oxycarboxin, mercurous + mercuric chlorides, iprodione and chlorothalonil Results with benomyl and thiobendazole[3] are erratic.

Table 8 Snow molds of turfgrasses - Distribution, Predisposition and Control - Summary (cont.)

Disease and distribution [1]	Predisposing factors	Cultural control	Fungicidal treatments [2]
Sclerotinia snow mold (*Myriosclerotinia borealis*). Regions with high snowfall and long duration snow covers in northern parts of Scandinavia, the USSR and Japan, in Alaska, western and central Canada and north-central USA.	Severe damage occurs only in higher snowfall areas and years when snow cover is prolonged. Epidemics occur even when permanent snow cover develops on frozen ground. Most common grasses are susceptible, but *Agrostis* cultivars are often severely damaged. Disease endemic on native grasses and sown species in ditches and other snow traps adjacent to mown turf which serve as infection reservoirs. Peat soils with low pH. Inadequate P and N.	Spread snow drifts to encourage rapid melting, especially on fine *Agrostis* turf. Control disease on infection reservoirs adjacent. Use cultivars of northern origin rather than southern ones which are less winterhardy. Ensure that the phosphorous status of the soil is adequate, but that there is sufficient nitrogen for good spring recovery. Do not use *F. rubra* north of Lat. 54°N in western Canada. Reduce inoculum by turf scarification and vacuum sweeper in spring.	When autumn is moist and "open" make at least two applications of fungicide before a permanent snow cover develops. Effective fungicides are: quintozene, benomyl, methyl thiophanate, chlorothalonil, thiobendazole, phenyl mercuric acetate, with or without thiram, and carboxin + thiram + oxycarboxin. Mercurous + mercuric chlorides and chloroneb are not reliable against the turfgrass disease.

Table 3 Snow molds of turfgrasses - Distribution, Predisposition and Control - Summary (cont.)

Disease and distribution [1]	Predisposing factors	Cultural control	Fungicidal [2] treatments
Cottony snow mold (Coprinus psychromorbidus) LTB and SLTB (sclerotial) phases of a highly variable fungal species. So far, the the fungus has been found on turfgrasses only in the Canadian Prairies, the Yukon and Alaska.	Shallow to deep snow covers, slow melting of drifts in spring. Unbalanced, late nitrogenous fertilization and unbalanced phosphate fertilization when turfgrasses are quiescent in winter. Previous attacks. Most common turfgrasses are susceptible.	Control snow deposition with ventilated fences and spread show drifts or speed snow melting with dark-coloured topdressing. Use cultivars which show early onset of dormancy and encourage this by greatly reducing nitrogen use in fall and early winter. There are some field resistant Poa pratensis cvs., and Festuca spp., but not in other turfgrasses.	Unless resistant P. pratensis cultivars are in use it may be necessary to use mercurous + mercuric chlorides to control severe outbreaks. Two applications of fungicide at least are needed, the last one as late as possible before the development of a permanent snow cover. Effective materials are: mercurous + mercuric chlorides, quintozene, chloroneb, oxycarboxin + thiram + carboxin, phenyl mercuric acetate, triadimefon fenarimol. Borax is also effective in combination with other materials, but its use is risky because of possible phytoxicity.

[1] For synonyms see main text.

[2] Some of the fungicides listed may not be registered for control of these diseases in Canada. Mercurial fungicides are not allowed in National Parks or in British Columbia. Other materials may be effective, but those listed are fungicides of which the author has experience.

[3] Thiobendazole may favour development of mycelium of T. canadensis on treated turf.

Table 9 Snow molds of turfgrasses - Identification - Summary

Disease	Pathogen	Diagnosis
Fusarium or microdochium patch and pink snow mold	Anamorph - Microdochium nivale (Fries) Samuels & Hallett synonym - Fusarium nivale (Fries) Sorauer	Fusarium patch symptoms appear in cool autumn or spring weather particularly, as patches 2.5 to 5 cm across of water-soaked, yellow, orange-brown or brown grass which may coalesce. In pink snow mold, after snow melt, patch centres bleach and have an orange-brown or brown margin with white mycelium. A pink colour may develop on the infected leaves. To identify the fungus incubate disease plugs or isolates in full light or nuv at 14 to 17 C and examine for spores produced in salmon-pink sporodochia. Grow on PDA or PSA medium. Spores from turfgrasses are typically 0- 1-septate and heelless or nearly so. Spores from cereals are similar in morphology, but multiseptate.
Sclerotinia snow mold	Myriosclerotinia borealis (Bub. & Vleug.) Kohn synonym - Sclerotinia borealis Bub. & Vleug.	After snow melt in spring, patches of grass with water-soaked leaves and sparse grey mycelium appear. These are up to about 15 cm across. Patches may be coalescent. Infected leaves bleach almost white, wrinkle, and may turn thread-like, darkening with saprophytic fungi. Sclerotia first cream- to putty-coloured or even faintly pink, globular, elongate or flake-like and arched with plant vascular remains attached are found in sheaths, crowns and leaf axils, on or within leaves. They vary in size according to host, but are commonly up to 7-8 x 3-4 mm. When mature they turn black and wrinkle when dry and readily detach from the host. Infected plants are usually dead.

Table 9 Snow molds of turfgrasses - Identification - Summary (cont.)

Disease	Pathogen	Diagnosis
Grey snow mold	Typhula incarnata Lasch. ex Fr.	At or after snow melt, or sometimes after a cold, wet period in winter with little or no snow, discrete patches may be only 2-5 cm across, but may increase under a snow cover up to 0.5 m. At snow melt sparse to dense white to greyish-white mycelium may mat together the patches. Globular to flattened spherical, faintly pink sclerotia, up to 5 mm in diam. are in or on infected tissues of leaves, and plant bases. Sclerotia darken from orange-brown to pinkish-orange to reddish-brown or dark brown and wrinkle on drying and may be firmly attached. Sporophores to about 20 mm in height with pale pink or white stipes and pink to rose-coloured clubs may develop in moist autumn weather from sclerotia on previously attacked turf. Cortical cells of sclerotia are lobate, interlocking like pieces of jigsaw puzzle. In culture young sclerotia are white, then pink, but may turn chestnut brown and become irregularly-shaped and are formed in rings without much aerial mycelium. When fully swollen, they are resilient and gelatinous.
Speckled snow mold	Typhula ishikariensis Imai vars.	Dark-coloured sclerotia give patches of diseases a speckled appearance although field symptoms of T. ishikariensis vars. are similar to those caused by T. incarnata. Sclerotia of T. ishikariensis are never pink or red, but dark amber to dark chestnut when fresh and dark brown to almost black when dry. They are not gelatinous. Sporophores have a greyish-white clavula shading into smoky-brown stipe bases.

Table 9 Snow molds of turfgrasses - Identification - Summary (cont.).

Disease	Pathogen	Diagnosis
	var. ishikariensis Årsvoll & Smith	Sclerotia are usually firmly attached to plant tissue, 0.3-2 mm diam. and sporophores are 4-20 mm tall. Little aerial mycelium is produced on potato-malt extract (BASM) agar and sclerotia are formed in concentric rings in petri dish cultures. Sclerotial rinds often rough, but rarely ridged or wrinkled. Rind cells rarely digitate and spherical cells with thickened walls present in some isolates. Cell outlines smoother than in var. idahoensis.
	var. idahoensis Årsvoll & Smith	Sclerotia not usually firmly attached to plant, 0.5-2 mm diam. and sporophores 3-14 mm tall. Usually there is little aerial mycelium on BASM agar and sclerotia are formed either in concentric rings or in a central pile in petri dish cultures. Sclerotial rinds often wrinkled, ridged, or with splits in the surface. Rinds cells less regular in outline than var. ishikariensis, sometimes with digitate and lobate cells and with disjunctions and ridges.
	var. canadensis Smith & Årsvoll	Sclerotia superficial, easily detached, suspended in mycelium between leaves or in abundant greyish wefts covering leaves, 0.2-1.6 mm in diam. Sporophores 1-11 mm tall. In culture on BASM at 6 C aerial mycelium is abundant with sclerotia suspended in it rather than in concentric rings on agar. Sclerotial rind sometimes rough, but without ridges and with superficial mycelium attached. Rind cells resemble those of var. ishikariensis rather than idahoensis, but less rounded than the formed and not as lobate as the latter.

Table 9 Snow molds of turfgrasses - Identification - Summary (cont.)

Disease	Pathogen	Diagnosis
Cottony snow mold (LTB snow mold)	Coprinus psychromorbidus Redhead & Traquair Nonsclerotial low-temperature tolerant basidiomycete	LTB phase: After snow melt in spring patches 15 cm or more emerge showing white, abundant to sparse mycelium, particularly at patch margins. Patches often do not coalesce and show green ribbons of undamaged grass between them, but symptoms vary on Poa pratensis, P. annua and Agrostis spp. The latter two species are usually killed and P. pratensis very slow to recover. There are NO sclerotia and NO spores, but the mycelium which is fluffy in culture on BASM agar has abundant clamp connections at 6 C.
Cottony snow mold (SLTB snow mold)	Coprinus psychromorbidus Sclerotial low-temperature tolerant basidiomycete	SLTB phase: Disease patches are usually smaller than those of the LTB and the aerial mycelium less abundant, cobwebby stretching from leaf to leaf. Hyphae are c. 1 um diam. with abundant clamp connections. They aggregate to form sclerotia on grass leaves and on the underside of leaves and twigs lying on the turf. Sclerotia are at first grey, turning grey-brown and finally charcoal-black, often irregularly shaped 0.25-1.5 mm, but usually more than 1 mm in any dimension, sometimes in the form of flakes. The sclerotial rind is made up of several layers of rounded, pigmented cells. When fully swollen in water the sclerotia exude milky contents when pierced with a needle. The fungus and the LTB are conspecific with C. psychromorbidus, but the SLTB is less pathogenic than the LTB and is also psychrophilic; optimum temperature c. 15 C.

Table 9 Snow molds of turfgrasses - Identification - Summary (cont.)

Disease	Pathogen	Diagnosis
	Coprinus psychromorbidus	Fruiting stage: Mesophilic, occurs on oversummering plants of cereals, forage legumes, grasses as a saprophyte. May also fruit on wooden pegs or canes in greenhouse plant containers used for infested sod. The caps are typically of Coprinus type, delicate, 7-12 mm wide conical to flat when mature with a narrow central boss. The stalk is 40-70 mm long and 2-3 um wide with a slightly swollen base. The cap surface is felted with recurved, orange-yellow to yellow-brown scales.
Brown root rot	Phoma sclerotiodes (Preuss) ex Sacc.	Grass plants show a basal brown rot at snow melt with sclerotia-like protopycnidia up to c. 0.8 mm in diam. on lower crowns and roots down to c. 10 cm deep in soil. They are usually solitary on roots, greenish-black, globose to conoid or slightly elongate on grass roots, dished on the abaxial side with a short attachment process and without locules at this stage. The protpycnidial wall is thin at this stage. Mature pycnidia are hemispherical, solitary, or confluent in culture with thickened wall and several locules which lead to beaked ostioles up to 200 um long. Spores are discharged through the ostioles as a cream or yellow cirrus. Conidia are hyaline, elliptical, biguttulate, 4-8 x 2-3.5 um.
Frost scorch or "string-of-pearls"	Sclerotium rhizoides Auersw.	Plants stunted with wilted, curled or rolled leaves, bleaching white, tips tending to become tendril-like, bases remaining green. Sclerotia formed in bead-like rows, superficially on withered leaves, oval, spherical or oblong, 1-5 mm diam., white or grey darkening to almost black and rough. No spores.

Table 3 Snow molds of turfgrasses - Identification - Summary (cont.)

Disease	Pathogen	Diagnosis
	Nectria tuberculariformis (Rehm ex Sacc.) Winter Anamorph - Acremonium boreale Smith & Davidson	Sclerotia-like stromata of the anamorph scattered on stems, leaves, bark and overwintered seeds c. 0.1-2.0 x 0.1-1.0 mm, lenticulate, globose or spindle-shaped, white at first, becoming bright orange, then light brown and hard. Sub-epidermal at first, then erumpent. Conidia develop on stromatic surface in cream, pink, orange, or orange-red masses, heteropolar, straight, polysymmetric, oblong to elliptic, sometimes waisted, c. 5.0-6.3 x 1.5-2.0 um. Perithecial ascomata in caespitose groups, with bases immersed in the stroma, white to orange, globose, non-papillate, 170-260 um. Asci unitunicate, cylindrical to narrowly clavate, c. 49-69 x 7-9 um, 6- 8- spored, uniseriate to biseriate. Ascospores elliptic to elliptic-fusiform, hyaline, equally 2-celled, not constricted, c. 9-12 x 2.5-3.2 um.

m Table 10 Temperature ranges (°Celsius) for snow molds in artificial culture[1]

Fungus	Minimum	Optimum	Maximum	Authority
Microdochium nivale (Monographella nivalis)[2]	0	22	30	Dahl, 1934
	0	20	32	Wollenweber & Reinking, 1935
	0 to 1	20 to 21	32.5	Bennett, 1933
	Survives at -20			
	-5	22	30	Ekstrand, 1955
	0.5	18 to 22		Smith, 1953
	-6	21	28	Årsvoll, 1975
Typhula incarnata	0	9 to 12	18	Remsberg, 1940
	-5	10 to 15	22 to 23	Ekstrand, 1955
	-5	8 to 15	22 to 23	Tasugi, 1935
	0	10	25	Remsberg & Hungerford, 1933
	0	8	25 to 30	Volk, 1937
		10		Yang, 1945
	-6	9 to 12	21	Årsvoll, 1975
	-6	12 to 15	21	Årsvoll & Smith, 1978
	-7	7 to 15		Tomiyama, 1955
			20	Dejardin & Ward, 1971
T. ishikariensis var. ishikariensis = T. borealis?	-6	9 to 12	20	Årsvoll & Smith, 1978
	-5	10	21	Ekstrand, 1955
T. ishikariensis var. idahoensis	-6	9	18	Årsvoll, 1975
	-6	3 to 15	18 to 20	Årsvoll & Smith, 1978
T. ishikariensis var. canadensis = T. hyperborea?	-6	6 to 9	21	Årsvoll & Smith, 1978
	-5	5 to 10	15 to 20	Ekstrand, 1955
Myriosclerotinia borealis	-7	7 to 15		Tomiyama, 1955
	-5	5 to 10	15 to 20	Ekstrand, 1955
	-5	0 to 5	15 to 20	Jamalainen, 1974
	-6	3 to 6	18	Årsvoll, 1975
Coprinus psychromorbidus LTB phase	-4	15	26	Broadfoot & Cormack, 1941
	-2	15	25 to 30	Smith, unpublished

Table 10 Temperature ranges (°Celsius) for snow molds in artificial culture[1] (cont.)

Fungus	Minimum	Optimum	Maximum	Authority
Coprinus psychromorbidus				
SLTB phase	-3	10 to 15 (some 0 to 5)	20 to 25	Smith, unpublished Traquair & Smith, 1982
From fruiting body	-3	c 25	c 30	Smith, unpublished
Nectria tuberculariformis	-6	9 to 15	25	Smith & Davidson, 1979
(*Acremonium boreale*)	-6	9 to 18		Samuels et al. (1984)
Sclerotium rhizoides		16		Stout, 1911
Phoma sclerotiodes	00	15 to 17	27	Sanford, 1933
	-7	15		Smith, unpublished

[1]From Smith (1969 and 1980, revised)

[2]Although *Monographella nivalis* is the teleomorph of *Microdochium nivale* there is some doubt whether the fungus from turf grasses is the same as that from winter cereals (Smith, 1983).