Effect of cooling and pre- and postharvest chemical treatments on *Sclerotinia sclerotiorum* in stored carrots (*Daucus carota*)

by

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EFFECT OF COOLING AND PRE- AND POST HARVEST CHEMICAL TREATMENTS ON *Clerotinia sclerotiorum* IN STORED CARROTS (*Daucus carota*)

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A thesis submitted to the Faculty of Graduate Studies of the University of Manitoba in partial fulfillment of the requirements of the degree of

MASTER OF SCIENCE

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ABSTRACT

Boese, Dennis Edward. M.Sc., The University of Manitoba, May, 1990. Effect of cooling and pre- and postharvest chemical treatments on Sclerotinia sclerotiorum of stored carrots (Daucus carota). Major professors: Dr. M.K. Pritchard and Dr. S.R. Rimmer.

The potential of field-applied fungicides and postharvest dips to reduce Sclerotinia sclerotiorum (Lib.) de Bary infection in stored carrots (Daucus carota L.) was investigated. The influence of postharvest cooling on sclerotinia disease development in storage was also examined.

Vinclozolin (3-[3,5-dichlorophenyl]-5-methyl-5-vinyl-1,3-oxazolidine-2,4-dione) applied at 0.84 l ai/ha to the foliage ten days before harvest resulted in almost complete prevention of disease development in storage. Two applications of vinclozolin during the growing season, at a dosage of 0.42 l ai/ha each time, were also effective in reducing sclerotinia rot. Benomyl (methyl 1-[butylcarbamoyl]-2-benzimidazole-carbamate) applied at 1.0 kg ai/ha ten days before harvest also reduced sclerotinia development in storage, but was less effective than vinclozolin.

A benomyl dip before storage reduced S. sclerotiorum development in storage. H_2O and $CaCl_2$ dips were not effective in reducing sclerotinia disease in stored carrots.

Cooling studies demonstrated the importance of rapid cooling after harvest to minimize losses in storage from sclerotinia disease. Carrots cooled to 0-1 °C in 6 hours had the least amount of disease after 15 weeks storage at 5-6 °C, with the amount of disease increasing as the length of the cooling period increased to 72 hours.

Temperature monitoring of three commercial carrot storages revealed that bulk stored carrots required up to 2 months to cool to storage temperatures of 0-1 °C. Warm outside air used for cooling, and employee activity in the storages which required doors to be open both tended to increase the temperatures within the carrot piles. Under these management conditions, losses due to sclerotinia disease could be substantial.

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Chapter I

INTRODUCTION

Carrots (Daucus carota L.) are an important crop in Canada where they are grown commercially in every province. Approximately 125 ha of carrots are grown in Manitoba, and local carrots are usually available from mid-August until the end of February. Manitoba producers also supply markets in several other provinces. The short growing season in Canada necessitates cold storage to extend the supply of high quality carrots.

Sclerotinia rot, caused by Sclerotinia sclerotiorum (Lib.) de Bary, is the major disease of stored carrots in Manitoba. While the disease originates in the field, it is mainly in storage that sclerotinia rot becomes a problem. Carrot losses of up to 40% may occur annually, with the amount of deterioration dependent upon environmental conditions in the field and upon management of the storage.

Correct control of temperature and relative humidity (RH) is essential in keeping carrots disease-free in storage. Storage at temperatures of 0-1 °C and 98- 100% RH is necessary, as S. sclerotiorum can develop rapidly and cause substantial losses at higher temperatures and/or lower levels of humidity.

Passing harvested carrots through a fungicide solution to reduce development of sclerotinia disease in stored carrots is likely not

feasible when large volumes of roots are being handled. Equipment and holding tanks needed to accomplish this are also expensive.

Since infection of the carrot occurs in the field, methods to reduce field infection of carrots by the pathogen would minimize disease development and losses in storage.

The objectives of this study were: (1) to monitor several commercial storages to determine the temperature conditions under which the carrots were being stored, (2) to determine the effect of cooling on the development of sclerotinia disease in carrots placed into long term storage, (3) to compare the effectiveness of field applications of fungicides in controlling sclerotinia rot in carrots placed into cold storage, and (4) to compare the effectiveness of postharvest chemical dips in controlling sclerotinia rot in carrots put into long term storage.

Chapter II

LITERATURE REVIEW

2.1 SCLEROTINIA SCLEROTIORUM

Sclerotinia sclerotiorum, an ascomycetous fungus, is one of the most ubiquitous and economically damaging of all plant pathogens, causing more than sixty diseases in more than 360 species of plants in 64 families (Steadman and Nickerson, 1975; Purdy, 1979). It is found in all major production areas of the world.

In recent years, disease caused by S. sclerotiorum has become more prevalent in North America, including Manitoba. Millions of dollars are lost annually to this pathogen, either from direct reduction in yield, or indirectly from downgrading of quality or the inability to use a crop in a particular rotation because of the prevalence of S. sclerotiorum (Purdy, 1979). Expansion of other crops susceptible to S. sclerotiorum, as well as changes in cultural practices related to carrots, such as increased irrigation and decreased row spacing, have increased losses from this disease (Boland and Hall, 1982, 1986; Grau and Radke, 1984).

Although most of its hosts are herbaceous, S. sclerotiorum attacks some trees and shrubs, as well as field and forage crops, vegetables, fruits, and ornamentals, and a number of weeds (Purdy, 1979).

Most of the literature on Sclerotinia spp. has focused on three species: S. minor, S. sclerotiorum, and S. trifoliorum. S. sclerotiorum, the most common species on the Canadian prairies, is characterized by the production of hyaline, ellipsoidal ascospores which, at maturity, have a cleft aperture. The asci are released from apothecia arising from the sclerotia on stipes. Sclerotia are generally black, and of variable size and shape. The mycelium is cottony and snow-white (Kohn, 1979; Huang, 1982; Mazza, 1989). S. sclerotiorum has no functional conidial state.

S. sclerotiorum is an extremely successful fungus that function either as a soilborne or airborne pathogen (Abawi and Grogan, 1975), depending on the host, the environmental conditions, and the inherent condition of the fungus. Approximately 90% of the lifecycle of Sclerotinia spp. is spent in the soil as resting sclerotia which allow the fungus to survive long periods of adverse conditions (Adams and Ayers, 1979; Willetts and Wong, 1980). Sclerotia can retain their viability in the soil as long as three years (Cook et al., 1975), four or five years (Adams and Ayers, 1979), or five years (McLean, 1958).

2.2 PROCESS OF INFECTION BY S. SCLEROTIORUM

Infection by S. sclerotiorum in many plants, especially beans (Steadman, 1979; Sutton and Deverall, 1983) and canola/rapeseed (Dueck et al., 1983), involves ascopores as the inoculum. However, ascospores do not usually infect susceptible host plants directly, but germinate on dead or senescing plant material to form mycelium (Purdy, 1958; Adams and Ayers, 1976). The organic matter thus serves as a nutrient base for

germination, penetration, and infection (Lumsden, 1979; Purdy, 1979; Boland and Hall, 1987). However, Sedun and Brown (1987) have reported direct infection of healthy host tissue in the absence of nonliving or senescent plant parts. Ascospores of S. sclerotiorum infected fully expanded sunflower leaves without added nutrients, wounds, or senescent tissue. The infection site was at the leaf blade-petiole junction, and occurred in those leaves which had begun to secrete sucrose. Fully expanded leaves were most often infected; green cotyledons were never infected. Sutton and Deverall (1983) found that ascospores of S. sclerotiorum could infect bean and soybean, in the absence of an exogenous nutrient source. Leaf age was important, as young tissues were penetrated by the infection hypha arising from the germinated spore. Infection did not occur in fully expanded leaves. However, direct infection of plants by ascospores is unlikely to be important epidemiologically (Sutton and Deverall, 1983).

2.3 GERMINATION OF S. SCLEROTIORUM

Viable sclerotia of S. sclerotiorum may germinate either carpogenically or myceliogenically (Willetts and Wong, 1980).

2.3.1 Carpogenic germination

Carpogenic germination occurs when sclerotia develop, under appropriate conditions, to produce stipes and apothecia. Ascospores are then formed in the apothecia and released. Cool moist conditions are necessary before sclerotia are capable of carpogenic germination. In the field, lack of consistently adequate soil moisture is the most common factor

limiting germination (Coley-Smith and Cooke, 1971; Abawi and Grogan, 1975, 1979). Continuous moisture near saturation for ten days is required for apothecial formation and development (Abawi and Grogan, 1975, 1979), although it is possible that sclerotia can germinate carpogenically when exposed to a series of short, discontinuous periods of wetting (Boland and Hall, 1987). The microenvironment which occurs within crops after the canopy closes may provide the cooler and wetter conditions and consistently adequate soil moisture required for carpogenic germination and apothecial development (Imolehin et al., 1980; Boland and Hall, 1987).

Abawi and Grogan (1975) found that approximately 48-72 hours of plant surface wetness were required for ascospore infection. Boland and Hall (1987) reported that disease could develop in the field when continuous leaf wetness was extended for more than 39 hours (39-64 hours).

Stipes produced by germinating sclerotia are positively phototropic, and require exposure to light before apothecia will form (McLean, 1958; Coley-Smith and Cooke, 1971; Le Tourneau, 1979). This light requirement ensures that, in nature, apothecia will be formed at or above the soil surface (Coley-Smith and Cooke, 1971). Apothecia of S. sclerotiorum arise primarily from sclerotia at a soil depth of 1-2 cm. Few apothecia are produced from sclerotia buried at 5 cm (Williams and Western, 1965).

2.3.2 Myceliogenic germination

Myceliogenic germination involves the production of mycelium by the conditioned sclerotia, and can occur in either of two ways. Eruptive

germination, as mainly occurs in S. minor, involves the rupturing of the sclerotial rind in the medullary region. Hyphal germination occurs when the individual hyphae emerge through the sclerotial rind without rupturing it (Adams and Tate, 1976; Huang, 1985).

Once sclerotia are conditioned, no exogenous nutrients are needed to initiate germination. However, there are apparently conflicting reports in the literature about the subsequent infection of host plants by sclerotia after germination. Purdy (1958) and Abawi and Grogan (1975) found that myceliogenically germinating sclerotia require a nonliving food base before infection can take place. Zimmer and Hoes (1978) reported that sclerotia must be in close proximity to the host tissue and that a food base was critical to infection by the fungus. In sunflower, root exudates and cortical materials dislodged from roots provided adequate nutrients to bridge the distance between germinating sclerotia and the host tissue (Zimmer and Hoes, 1978). Huang and Dueck (1980), in greenhouse studies, showed that sclerotia of S. sclerotiorum germinated mycelogenically in the soil, and that this mycelium could infect roots and hypocotyls of sunflower seedlings without added nutrients or wounding of the host tissues. Rapeseed, however, was not affected similarly.

2.4 CARROTS

The carrot plant is a biennial of the cool temperate regions, where the root and hypocotyl have evolved as a survival and storage organ (Lewis and Garrod, 1983). Thus the metabolism of the carrot does not stop after harvest, but, although reduced, persists until the physiological

breakdown of the root. Storage deteriorations of commodities are due to microbial attacks, water loss (evaporative loss), and biochemical change (Dennis, 1977; Burton, 1977; Grierson and Wardowski, 1978). Water loss and decay are the main factors leading to reduction in the quality of stored carrots (Phan et al., 1973). Technologies involved in control of storage environments have generally enabled a tight rein to be kept on wilting and dehydration from water loss. Disease is now the major cause of losses for growers with stored carrots.

2.5 SCLEROTINIA DISEASE IN CARROTS

In most seasons S. sclerotiorum infects carrots in the field, but the disease becomes apparent only when in storage (Lewis and Garrod, 1983), although it occasionally manifests itself as a damping-off in the field (Mazza, 1989). In the UK the main inoculum source for sclerotinia disease in carrots is ascospores produced by apothecia in the autumn when conditions are suitable for ascospore production and release, and when senescing carrot leaves can serve as a nutrient base for infection (Geary, 1978). Geary (1978) observed that the leaves most often infected were those leaves just beginning to senesce; newer leaves had low rates of infection, and fully senescent foliage was never found to be infected.

Ascospores released from sclerotia in other fields can travel considerable distances (Abawi and Grogan, 1975). This may be a possible source of infection for carrots in Manitoba.

Infection of carrots is not likely to occur through direct mycelial infection of the roots in the soil by sclerotia in Britain (Geary, 1978) or in Manitoba (Finlayson, 1989). Ascospores produced in autumn are the main source of inoculum in Britain (Geary, 1978). In Manitoba, mycelium from sclerotia which have germinated myceliogenically may infect senescing leaves touching the soil (Finlayson et al., 1989a). Tahvonen (1985), in studies on control of S. sclerotiorum with fungicides, assumed that infection takes place mainly through the tops during harvesting. Infections that develop via senescing leaves can progress down the petioles to enter the carrot root via the crown (Lewis and Garrod, 1983). Crown infections are not easily spotted while the carrots are being piled into storage, because the foliage is trimmed off upon harvesting.

Farm operations which cause mechanical injury to plants increase the chance of infection. Only sclerotia in the top several centimetres of soil are responsible for infection of carrots. Diatchenko (1974) found that when the number of interrow cultivations for weed control was increased from 2 to 4, the amount of losses to S. sclerotiorum increased sharply. Excess cultivation may increase infection in two ways: it may bring more sclerotia close to the surface, and it may increase damage and injury to the carrot plants.

Careless and/or excessive handling of produce, both during harvest and while loading into storage, can break off lateral roots, or bruise, puncture, scratch, or otherwise injure the periderm, facilitating pathogenic entry and infection. Only the most obviously deformed, cracked, or diseased roots, and some debris and clumps of soil are

removed during loading into storage. Additionally. the senescent and broken leaves and the soil debris may act as a source of inoculum if they are carried into storage at harvest time (Geary, 1978).

Sclerotinia rot is a serious postharvest disease in carrots, as it progresses rapidly under optimum environmental conditions, and the pathogen produces extracellular enzymes which macerate the fleshy tissues of the host. Once in storage, S. sclerotiorum can spread between adjacent carrot roots via mycelial growth to infect healthy carrots. Characteristic "nests" or foci of infection may be formed by mycelial growth outward from each original point of infection. Advanced infections can cause severe losses.

Entry and infection by all carrot storage fungi, including S. sclerotiorum, is considerably enhanced by the presence of wounds (Lewis and Garrod, 1983). When first loaded into storage, carrots are highly resistant to infection, but this resistance declines over time (Goodliffe and Heale, 1977, 1978; Heale et al., 1977; Lewis et al., 1981).

Differences in disease resistance have been noted between the various carrot tissues (Davies, 1977; Goodliffe and Heale, 1977; Davies et al., 1981). The periderm is highly resistant to infection, with the pericyclic parenchyma, the underlying phloem parenchyma, and the innermost xylem parenchyma being progressively less resistant to infection. Thus the depth of wounding markedly affects the incidence of infection (Dennis, 1977). Machine harvesting causes both minor surface abrasion of the periderm and deeper wounds of the inner tissues, as well

as complete breaks of the carrot root. The minimal sorting which occurs while the carrots are being loaded into storage is not sufficient to remove all the damaged roots.

2.6 STORAGE OF CARROTS

Long term storage of carrots is necessary in Manitoba to maintain the supply of local carrots as long as possible before imports are brought in to supply the market. Refrigerated storages with forced air ventilation are used to store carrots. The crop is normally stored in bulk in piles up to 4m in depth. Pallet bins are used prior to marketing for some short term storage of carrots.

The two most important factors in keeping carrots disease-free in storage are correct control of temperature and of relative humidity (RH). There is widespread agreement that 0-1 °C represents the optimum keeping temperature for carrots in storage (Lentz, 1966; Phan et al., 1973, Apeland and Hoftun, 1974). Not only do the carrots then have better flavor, color, and texture (Lewis and Garrod, 1983), but the activity of S. sclerotiorum is also lower than at higher temperatures (van den Berg and Lentz, 1968; van den Berg and Yang, 1969).

Van den Berg and Lentz (1966) conducted long term storage tests (up to 9 months) with carrots, at temperatures of 0-1.1 and 2.8-3.9 °C. The lower temperatures resulted in the least disease and the best quality carrots. While the carrots stored at the higher temperatures showed more sprouting and lower table quality, their rate of decay was not increased appreciably. Disease development seemed to be more dependent

on variables such as RH, variety, storage season, and atmospheric composition.

Van den Berg (1981) in tests at 0-1, 2-3, and 3.5-4.5 °C, found that carrot losses from disease were greatest at the highest temperatures, especially at lower RH levels. Other tests showed that the type of decay varied with temperature. Relatively small dry brown lesions prevailed at 0-2 °C, while at 3-8 °C watery soft rot spot caused by Sclerotinia spp. dominated (van den Berg and Lentz, 1966, 1973; van den Berg, 1981).

Carrots can withstand temperatures as low as -1.7 °C (Salunkhe and Desai, 1984) without freezing. Smith (1967) observed blister-like lesions on carrot roots at -0.6 °C, and found more rotting (mainly from Botrytis and Sclerotinia spp.) at 0 °C than at 1.1 °C. The lateral surface of the carrot was affected, not the crown or the tip. Smith (1967) suggested this was a form of low temperature chilling injury, resulting from metabolic changes in root surfaces, which made them more susceptible to infection. Van den Berg and Lentz (1973) stated that if low temperature injury was present, it was small and not consistent throughout. Van den Berg (1981) found that in a few tests decay was slightly less at 2-5 °C than at 0-1 °C. Apeland and Hoftun (1974), in long term storage experiments with carrots, found no clear evidence of low temperature injury, and concluded that 0-1 °C is the optimum storage temperature. Jensen (1971) found that disease caused by Rhizoctonia carotae was enhanced at 1 °C as compared to carrots stored at 5 °C.

Although suggested optimum RH values for carrots in storage vary from 90-95% (Djacenko, 1971) to 98-100% (van den Berg and Lentz, 1978; Raghavan et al., 1980), an RH of close to saturation is generally used.

Djacenko (1971) reported that high humidity and turgid roots favored increased levels of infection by S. sclerotiorum and decreased levels of B. cinerea. Derbyshire (1973) reported that the incidence of S. sclerotiorum and R. carotae was higher under conditions of very high humidity, in contrast to B. cinerea. Jensen (1971) found that high RH favored R. carotae. Van den Berg and Lentz (1966, 1973, 1978) concluded that carrots kept better with less decay at 98-100% RH than when stored at 90-95% RH. These results were unexpected, as it had been believed that high humidities would lead to accelerated decay, either because of effects of the RH level itself, or because of unavoidable condensation of water on the product at low temperatures (van den Berg, 1981). Krahn (1974) supported the view that high humidities do not necessarily lead to an increased incidence of rotting, but linked increased decay with the presence of free water, such as the presence of condensate on the product.

Van den Berg and Lentz (1966, 1974, 1978) reported that surface condensation on carrots did not enhance disease. Tests with carrots, cabbage, and celery showed that condensation did not increase rotting, and in the case of carrots, it even reduced the incidence of disease (van den Berg and Lentz, 1974). Condensation on vegetable surfaces decreased the rate of moisture loss, and in some instances even caused moisture gains. In addition, condensation improved the appearance and color of the product. Although molds grew abundantly over the surfaces

of root vegetables at the high RH levels, they did not cause decay or damage (van den Berg and Lentz, 1978; van den Berg, 1981).

Among the pectolytic enzymes produced by S. sclerotiorum are pectin methylesterase, endopolymethylgalacturonase, endopolygalacturonase, and exopolygalacturonase (Echandi and Walker, 1957; Hancock, 1966; van den Berg and Yang, 1969; Lumsden, 1979). Substantially more pectolytic enzymes were produced on the surfaces of carrots stored at 94-96% RH than those on stored at 98-100% RH (van den Berg and Yang, 1969). The enzymes were produced in significant quantities only when readily metabolizable sources of energy were not available. Low RH levels increased enzyme production, by concentrating nutrients on the surfaces of the carrots to the point where they inhibited growth of S. sclerotiorum and stimulated enzyme production. High RH, on the other hand, reduced disease because of lower pectolytic enzyme production (van den Berg, 1981; van den Berg and Yang, 1969; van den Berg and Lentz, 1978), or because of more rapid deactivation of the enzymes at the higher humidity levels (van den Berg and Lentz, 1978).

2.6.1 Cooling rate

Very few studies have been done on how the rate of cooling affects sclerotinia disease in stored carrots. Rates of cooling vary with the type and size of container, if any, and with the ventilation or circulation of the cooling medium. Rapid removal of field heat is especially important with the leafy vegetables. However, even carrots and other root crops should be cooled the same day that they are harvested (Ryall and Lipton, 1979). In Manitoba, carrot storages are

filled over a period of a number of weeks. The storage doors remain open during the day during continuous filling of the storages, and the doors are shut only when harvesting is completed for the day.

Active cooling of the carrots may be accomplished by use of a refrigeration system using refrigeration coils. In this system the temperature of the evaporating refrigerant in the cooling coil is important in ensuring high relative humidity in the storage air (Watson, 1960). Smaller coils require lower refrigeration temperatures in order to absorb sufficient heat to cool the carrots and keep them at optimum temperature. These lower temperatures cause moisture to condense on the surface of the cooling coils, drying the air being put through the storage. Humidities may also suffer with smaller cooling coils (Watson, 1960).

Another system for cooling carrots is the 'Filacell' system, which employs water as the heat transfer medium to cool the storage air. Cold water is sprayed over the Filacell packing of the refrigeration system (Krahn, 1974). Air is ducted through the Filacell and then back out through the storage facility. As the air passes through the Filacell pads it is cooled and humidified. Condensation of free water on carrots is also avoided. Heat exchange is lessened as the temperature differential lessens, as the temperature of the cooling carrots approaches the temperature of the water used in the Filacell system. However, antifreeze solutions such as glycol can be added to obtain lower Filacell temperatures down to -1.6 °C (Krahn, 1974), just above the freezing point of carrots.

Cold outside air may be ducted directly into the storages to cool the carrots. However, this air has a low relative humidity, and care must be taken to ensure the RH is kept in the saturated range with supplemental misting. According to Krahn (1974), under typical Canadian prairie conditions, outside atmospheres may have humidities as low as 10%.

2.7 CONTROL OF SCLEROTINIA DISEASE IN CARROTS

2.7.1 Cultural practices

The main ways in which cultural practices aid in controlling S. sclerotiorum are either by preventing inoculum from coming in contact with the host crop, or, if inoculum is present, to make conditions for infection and disease progression less likely.

Crop rotation is recommended for control of S. sclerotiorum in carrots (Diatchenko, 1974), snap beans (Natti, 1971), and soybeans (Grau and Radke, 1984), among others. Effective crop rotation will reduce infection to levels sufficiently low to allow carrots to be planted without major losses in disease once they are in storage. According to Crete (1980) a three-year rotation of carrots with nonsusceptible crops such as beets, spinach, corn, or cereals will reduce disease levels. However, others have found that a three-year rotation did not reduce sclerotial populations significantly (Schwartz and Steadman, 1978; Williams and Stelfox, 1980).

Plant architectural traits can influence disease incidence and severity. Germination and infection by S. sclerotiorum are favored by

high RH and moisture levels which are prevalent under dense crop canopies (Rainbow, 1970; Schwartz et al., 1987). Potato plants with a dense, spreading growth habit had more S. sclerotiorum lesions than plants of a more open and upright growth habit (Partyka and Mai, 1962). Susceptible cultivars of lettuce had senescent lower leaves which were in contact with the soil, thus creating both a cooler, wetter microclimate around the base of the plant, and a nutrient source for infection by S. minor. (Hawthorne, 1974). Schwartz and Steadman (1978) observed that apothecial growth of S. sclerotiorum was less in bean cultivars with open and upright growth habits than in those with dense or viny growth. Open and upright habits allow drying of plant and soil surfaces, and allow air circulation and light penetration (Schwartz et al., 1987).

Manipulation of plant populations and plant spacing can similarly affect the microclimate. Use of lower plant populations or greater spacing among plants allows a greater time before the crop canopy is filled. Development of sclerotinia disease has been reported after closure of the crop canopy in white beans (Boland and Hall, 1987), tomatoes (Rainbow, 1970), and soybeans (Grau and Radke, 1984).

Irrigation allows wetting of plant and soil surfaces, and its use has been reported to enhance severity of sclerotinia disease (Schwartz and Steadman, 1978; Grau and Radke, 1984; Boland and Hall, 1987). Irrigation in southern Alberta allowed sclerotinia white mold to become a problem in beans, even in years when there was a severe drought in the area (Huang et al., 1988).

2.7.2 Disease resistance

The development of genetic resistance to S. sclerotiorum in cultivated carrots has not been a goal of carrot breeding programs (Lewis and Garrod, 1983). Lauritzen (1932) compared 14 carrot cultivars for susceptibility to infection by S. sclerotiorum. Although differences between cultivars were noted, repeatable results could not be obtained, and thus Lauritzen concluded that these differences in susceptibility were of no practical importance. Finlayson et al. (1989b) grew five commercial carrot cultivars under field conditions and tested them for differences in levels of sclerotinia disease after long term storage. Significant differences in susceptibility to S. sclerotiorum were found at harvest, and after 3 months in storage at 5-6 °C. Conductivity assays were also done to determine electrolyte losses from carrot root discs infected by S. sclerotiorum (Finlayson et al., 1989b). Significant differences were shown by the carrot cultivars in response to infection, indicating variations in the integrity and permeability of cell membranes when carrot roots were exposed to S. sclerotiorum.

2.7.3 Preharvest treatment of S. sclerotiorum

Soil biocides, soil solarization, and other soil treatments have been used to reduce inoculum of S. sclerotiorum in the soil (Partyka and Mai, 1962; Steadman, 1979; Ben-Yephet, 1988).

Foliar protectants can be used against S. sclerotiorum in many crops, and have provided various degrees of control (Natti, 1971). Benomyl is one of the fungicides most commonly used for reduction of infection by S. sclerotiorum. It is a systemic fungicide which, when applied to

foliage and soil, is active against sclerotia, stipes, apothecia, ascospores, and mycelium of S. sclerotiorum (Hawthorne and Jarvis, 1973; Tu, 1983; Yarden et al., 1986). More recently, fungicides such as vinclozolin have become available which are active specifically against sclerotium-producing fungi such as S. sclerotiorum (Dueck et al., 1983).

Natti (1971) demonstrated maximum control of white mold in bean blossoms when benomyl was applied 3-7 days before full bloom (prebloom), and at full bloom. This period is the time when buds and blossoms are at an active stage of development. Benomyl acts not only via surface protective action, but is also systemically taken up by bean foliage and translocated into developing buds and blossoms (Natti, 1971). Fungicidal effectiveness was retained even after the blossoms senesced and dropped. Timing of fungicidal application is also critical in tomatoes, as benomyl applied after the crop canopy closed failed to penetrate the foliage (Rainbow, 1970). This closed canopy also provided a more humid environment for development of S. sclerotiorum.

Yarden et al. (1986) found that a combination of benomyl and thiram applied to the soil gave enhanced control of S. sclerotiorum, and allowed a lower dosage of benomyl to be used. As well, benomyl degradation in the soil was suppressed by the thiram.

Benomyl, chlorothalonil, and iprodione are currently recommended in Manitoba for control of S. sclerotiorum in canola and field beans (Anonymous, 1988).

A single application of one of several fungicides to rapeseed in the early bloom stage effectively controlled sclerotinia stem rot (Dueck et

al., 1983). Benomyl applied at dosages of 0.5 kg ai/ha or more, vinclozolin at 0.75 kg ai/ha or less, or benomyl at 0.25 kg ai/ha in combination with vinclozolin controlled disease consistently and satisfactorily. Control was less consistent with iprodione. Application of fungicide at the early bloom stage precedes initiation of infections, and occurs before the crop canopy becomes so dense that fungicide spray penetration is prevented (Dueck et al., 1983).

Sclerotinia and botrytis disease were substantially reduced in stored carrots by spraying the tops with one of several fungicides the day before harvest (Tahvonen, 1985). Chemical residues in the carrots were thus low, because the short time between treatment with fungicides and the harvesting of carrots did not permit the movement of the fungicides into the roots (Tahvonen, 1985). The percentage of roots infected by S. sclerotiorum was reduced to 2.9% from 43.8% in 1978/79, and to 0% from 11.9% in 1981/82 when thiophanate methyl was used the day before harvesting. Benomyl was used to control B. cinerea, reducing disease levels to 2.4% from 6.5% in 1980/81, and to 12.8% from 30.7% in 1981/82. Vinclozolin reduced B. cinerea in carrots to 2.9% from 19.7 % in 1980/81, and to 17.8% from 30.0% in 1981/82 (Tahvonen, 1985).

2.7.4 Postharvest treatment of S. sclerotiorum

A number of fungicides, used as dips at harvest before crops are stored, have been found to be effective in reducing decay in storage. A benomyl dip of 0.25-0.5 g/l gave almost complete control of disease after storage for 5 months (Derbyshire and Crisp, 1978). Lockhart and Delbridge (1974) obtained enhanced control of storage rots (mainly

caused by Botrytis cinerea and Rhizoctonia spp.) by postharvest treatment of washed carrots with benomyl or thiabendazole. Geeson et al. (1988) found that benomyl or iprodione dips of 0.5 g ai/l significantly reduced decays caused by B. cinerea and S. sclerotiorum, but increased disease caused by Rhizoctonia carotae.

Opinion is divided as to the merits of washing carrots before storage. Derbyshire (1973) found that a water dip could improve the storage potential of carrots compared to unwashed carrots. This improvement was evident only in hand lifted roots; in machine harvested carrots washing had a deleterious effect. Carrots washed and graded prior to cold storage were significantly less decayed than those stored directly from the field (Lockhart and Delbridge, 1972). However, washing of carrots may increase decay because of buildup of infective propagules in wash water, and their subsequent spread into hydrated or watersoaked lenticels and wounds (Eckert, 1977; Eckert and Ogawa, 1983).

In Manitoba, time constraints at harvest also come into play. The carrot harvest in Manitoba is a busy time, and labor is at a premium for the many tasks that have to be done then. Dipping of carrots involves extra time and labor. For this reason carrots in Manitoba are not washed, but are put into bulk storage unwashed and undipped. As well, while benomyl is used commercially in eastern Canada, treatment of unwashed carrots with the fungicide in Manitoba may result in reduced control compared to treated washed carrots (Anonymous, 1989). Vinclozolin is not registered for use on carrots in Manitoba.

2.7.5 Role of calcium in control of sclerotinia disease

Calcium is an essential constituent of the cell, and contributes to the structural integrity of plant tissues as part of the pectin complexes in the middle lamella of the cell wall. Decreased cell wall rigidity and altered membrane permeability have been noted as a result of calcium deficiencies in plants (Poovaiah, 1986). A deficiency in calcium is known to increase susceptibility to microbial attacks in several plants (Deverall and Wood, 1961; Skou, 1971), and high calcium content in the host has often been associated with disease resistance (Bateman and Lumsden, 1965; Bateman and Millar, 1966).

Endopolygalacturonase and pectin methylesterase are the primary pectic enzymes produced by S. sclerotiorum. Endopolygalacturonase does not attack calcium pectate except in the presence of a chelator of Ca^{++}, and the addition of Ca^{++} to a mixture of enzymes and plant tissue inhibits or retards tissue maceration due to the increased formation of calcium pectate (Bateman and Lumsden, 1965; Bateman and Millar, 1966).

S. sclerotiorum produces oxalic acid, which aids pathogenesis by enhancing the macerating ability of the fungus (Tu, 1984). Oxalic acid acts synergistically with polygalacturonase activity by lowering pH closer to optimum for the enzyme, and by rendering cell walls more susceptible to hydrolysis by polygalacturonase (Maxwell and Lumsden, 1970). Calcium may, when present in excess, prevent the oxalic acid from removing the calcium from the pectic substances in the middle lamella (Skou, 1971).

Calcium has been used successfully for a number of years for the commercial storage of apples. Conway and Sams (1987), in testing the effects of $CaCl_2$, $MgCl_2$, and $SrCl_2$ on apples stored for 5 months, found that calcium was the best cation for reducing decay, maintaining fruit firmness, and suppressing ethylene production.

Skou (1971), using 1.5-2.0 g $CaCl_2 \cdot 2H_2O$/l, tested the effect of calcium dips on irradiated and non-irradiated carrots. Calcium gave protection against disease (caused by S. sclerotiorum and B. cinerea) by reducing the effects of oxalic acid and pectic enzymes which degrade substances of the middle lamella. $CaCl_2$ worked equally well on non-irradiated and irradiated carrots, and gave protection which increased with the number and severity of wounds (or impairment of the cork/periderm layer) (Skou, 1971).

Chapter III

METHODS AND MATERIALS

This thesis project consisted of four parts: (1) temperature monitoring of three commercial carrot storages to investigate the temperature conditions carrots undergo in storage; (2) determination of the effect of cooling on infection by Sclerotinia sclerotiorum of stored carrots; (3) comparison of the effectiveness of field treatments to prevent or delay infection of carrots by Sclerotinia sclerotiorum; (4) comparison of the effectiveness of postharvest prestorage dipping treatments to prevent or delay infection of stored carrots by Sclerotinia sclerotiorum.

3.1 SOURCES OF FUNGAL MATERIAL

The fungal material (SS13) used in the 1985 experiments was isolated by Menzies (1983) from a diseased chicory plant found in a commercial carrot storage at Jamor Farms, Portage la Prairie, Manitoba. Isolate SS13 was grown on potato dextrose agar (PDA) prior to use in the 1985 experiments. A mycelial plug from this culture was transferred to a healthy carrot root to confirm the infectivity of isolate SS13 on carrots. In 1986, S. sclerotiorum which had been taken from an infected carrot from the 1985 experiments was used.

3.2 TEMPERATURE MONITORING OF CARROT STORAGES

The temperature conditions of carrots undergoing cooling and storage in three commercial storages were investigated. Two carrot storages were monitored at Jamor Farms, Portage la Prairie, MB. One consists of a wooden storage building cooled by a Filacell refrigeration unit and/or a refrigeration coil. The other is a steel building cooled by a refrigeration coil or by outside air once air temperature decreases in the fall. A third carrot storage, located at Connery's Riverdale Farms, Portage la Prairie, was also monitored. This storage utilizes a refrigeration coil for cooling.

For recording of carrot temperatures in storage, 24-gauge Type T (iron-constantin) thermocouple wire with ripcord coating (Thermo-Electric, Brampton, Ontario) was used. Thermocouples were made, and the probe end of the wire was coated with silicon seal caulking (Canadian General Electric, Toronto, ON) for protection from moisture to prevent false temperature readings.

Probe wires were connected to temperature recorders for the temperature data received, and were calibrated to 0 °C in an ice-water bath. At Jamor Farms a Honeywell Electronik 16 multipoint chart recorder (Honeywell, Inc., Philadelphia, PA) was used in the 1985 season, while in 1986 a CR5 digital recorder (Campbell Scientific, Logan, UT) was used. Temperatures were recorded at Connery's Riverdale Farms both years with a Campbell Scientific CR5 digital recorder.

The thermocouple wires were placed among the carrots as the crop was being piled into storage. Wires were put into the storage throughout

the depth of the pile in order to monitor temperatures and cooling throughout. Carrot piles were generally about 4 m high. Probes were placed into the pile at the surface (4 m above the bottom) and at heights of 3 m, 2 m, and 1 m from the bottom. Only one location was measured for each depth. Temperature probes were placed in the ducts in all three storages to measure the air temperature prior to its entry into the carrot pile. Temperatures were monitored until the wires were exposed by carrots being removed from storage. Relative humidity levels in the three storages were measured several times during the growing season with a sling psychrometer.

3.3 COOLING EXPERIMENTS

The effect of cooling on incidence of infection by S. sclerotiorum in stored carrots was investigated in these experiments.

Preparation of the field for seeding included application of nitrogen (N) and phosphorus (P) at a dosage of 36 kg/ha each, applied the previous fall. Postemergent linuron (0.55 kg ai/ha) was applied for weed control, and carbaryl (0.95 l ai/ha) and fenvalerate (18.6 g ai/ha) for the control of leafhoppers. The plots were irrigated when required.

3.3.1 Cooling experiment 1985

Carrots (cv. 'Paramount') were planted May 17 in 4-row beds with a row spacing of 40 cm. Carrots were seeded with a cone and belt seeder at 87 seeds/m. Beginning September 10 the carrots were harvested by hand using a spading fork. Soil was cleaned off the roots, and the carrots

were topped and taken back to the University of Manitoba. Cleaning of the carrots was done to the approximate degree which they would receive if machine harvested.

In any commercial carrot operation there is some minor sorting and grading of the carrots as they go into storage. This was also done with the carrots used in the cooling experiments. Carrots with defects, such as any signs of disease, most broken carrots, and most undersize and oversize carrots were not included.

The carrots were packed into 11 kg (25 lb.) bags, and into each of the bags were placed two discs of sclerotinia-infected carrot root slices. One carrot disc was placed 1/4-1/3 of the way from the bottom of the bag, while the other was placed 1/4-1/3 of the way from the top of the bag. Each bag was punched with forty 0.5 cm holes for ventilation.

The carrot discs (2.0 mm thickness) were prepared by cutting them to 1.0 cm diameter with a cork borer, and then sterilizing in a bleach/sterile distilled water solution (1:4 v/v). The discs were placed on petri plates of PDA cultured with S. sclerotiorum, where the slices were overrun and colonized by the fungus.

The bags of carrots were subjected to four different cooling rates by lowering the temperature to 0-1 °C over a period of 6, 24, 48, or 72 hours. The carrots were cooled in a mobile Vegetable Rapid-Chilling Unit. This unit can rapidly chill vegetables to any desired temperature. The temperature was adjusted downward in a stepwise fashion to obtain the desired rate of cooling. Six stepwise increments

were used for each cooling time. Thus the 6-hour treatment received hourly temperature adjustments, while for the 72-hour cooling time the temperature was adjusted every 12 hours. Temperatures achieved by the Vegetable Rapid-Chilling Unit were checked prior to the experiment to ensure accuracy of cooling. The carrots were stored at 5-6 °C for more rapid disease development.

Five replicates were used for each cooling rate, and each replicate consisted of two 11 kg bags of carrots. The two bags from each replicate were then placed into storage for two different lengths of time, to serve as subtreatments testing the the effect of the different lengths of cooling over time. However, because of the advancement of sclerotinia rot in the bags of carrots from both storage periods, all carrots were removed from storage after 15 weeks. The percentage by weight of healthy and diseased carrots was then determined, and statistical analysis, using Duncan's Multiple Range Test, was performed.

3.3.2 Cooling experiment 1986

In 1986 the carrots (cvs. 'Paramount' and 'Six Pak II') were machine harvested from a commercial field at Jamor Farms. The same degree of sorting and culling was applied to the carrots as in 1985, and they were then packed into 11 kg bags. However, the inoculum used in 1986 was discs of sclerotinia-infected PDA (1.0 cm diameter), rather than the sclerotinia-infected carrot discs used the previous year, to preclude the possibility that other organisms might also infect the bags of carrots.

Other experimental procedures were the same as in 1985, with the same cooling rates. However, only one 11 kg bag of carrots was used for each replicate, as only one storage period was used. After 14.5 weeks the carrots were removed from storage and the percentage of healthy and diseased carrots, by weight, was recorded. Statistical analysis of results was then performed.

3.4 FIELD APPLICATION OF FUNGICIDES FOR CONTROL OF S. SCLEROTIORUM

This study assessed the effectiveness of fungicides in controlling sclerotinia disease development in stored carrots, through the application of chemicals to the growing crop. Vinclozolin was used in 1985, while benomyl was also tested in 1986.

3.4.1 Field experiment 1985

Carrots (cv. 'Paramount') were planted 17 May at Jamor Farms. The experiment was designed as a Random Complete Block Design (RCBD) consisting of three treatments replicated four times. Each plot consisted of one four-row bed 6 m long. Four-row beds, instead of the commercial 3-row beds, were used to give a greater plant density and plant canopy to enhance disease development. The rows within the beds were spaced 40 cm apart. Seeding was done with a belt and cone seeder at 87 seeds/m. On June 21, 25 g of sclerotia were dug into the soil to a maximum depth of 1-2 cm between the center two rows of the 4-row plot. The outer two rows of each 4-row plot served as guard rows. The sclerotia needed for this experiment were produced from isolate SS13 by culturing the organism on petri plates of PDA, until the fast growing

mycelium reached the edge. The PDA was then cut into cubes and placed in foil-lined cake pans of autoclaved, pureed baked beans (Heinz Beans with Pork in Tomato Sauce, Heinz Ltd., Leamington, ON) for 10-14 days at room temperature, to allow formation of sclerotia. The sclerotia were conditioned in plastic bags of well-moistened, sterilized sand for 6-8 weeks before use.

The three treatments consisted of a control which was not inoculated with sclerotia nor treated with fungicide, a treatment inoculated with sclerotia, and an inoculated treatment sprayed with vinclozolin (at a total dosage of 0.84 l ai/ha) for disease control. This vinclozolin treatment was applied twice during the growing season, at a dosage of 0.42 l ai/ha each time, once on June 26, while the crop canopy was still open, and once on July 24, when the crop canopy was closed. Vinclozolin was sprayed with a CO_2 backpack sprayer (35 psi) with a water volume of 1 l vinclozolin/140 l H_2O.

At the end of the growing season the carrots were manually harvested, beginning October 15. After topping, the carrots were cleaned to the approximate degree they would be cleaned if machine harvested. The carrots from each plot were put into two 11 kg bags punched with forty 0.5 cm holes for ventilation, and were stored at 5-6 °C. After storage for 11.5 or 17 weeks, the bags were assessed for infection by S. sclerotiorum. One bag from each plot was assessed at each sampling, and statistical analysis, using Duncan's Multiple Range Test, was performed.

3.4.2 Field experiment 1986

Carrots were planted May 23 at Jamor Farms in 4-row beds 6 m in length. The rows were spaced at 40 cm. The experiment was designed as a RCBD, consisting of five treatments replicated four times. Treatments consisted of two different chemicals, benomyl and vinclozolin, with vinclozolin being applied at 0.84 l ai/ha on September 3, and at 0.42 or 0.84 l ai/ha on September 30. Benomyl was applied at 1.0 kg ai/ha on September 30 in a volume of 1 kg benomyl/1000 l H_2O. An unsprayed control was also included. The time of fungicide application was changed from earlier in the season in 1985 (June 26 and July 24) to September 3 and 30 in accordance with the results of Tahvonen (1985), who obtained good disease control of S. sclerotiorum and B. cinerea in stored carrots with applications of benomyl, thiophanate methyl, or vinclozolin the day before harvest.

All treatments, including the control, were inoculated in 1986. Although experiments in 1985 had shown no significant differences in disease development between the uninoculated and inoculated treatments, all plots were inoculated to ensure infection of the carrots. Sclerotinia-infected millet seed was used as the inoculum in 1986. S. sclerotiorum, obtained from infected carrots of the previous year's experiment, was cultured on petri plates of PDA for 3-4 days at room temperature, until the agar was covered by mycelium. During the same period, the millet seed was soaked overnight, drained, and autoclaved (121 °C, 102 kPa) in 1.14 L jars for two 20-minute periods. The mycelium-covered agar was then cut into cubes. One-half of each petri dish was mixed into each jar of autoclaved millet. After 6-7 days of

incubation at room temperature the inoculum was ready for use. 250 ml of inoculum was sprinkled between the inner two rows of each plot on August 19.

The carrots were harvested manually, beginning October 10. After topping and hand cleaning, two 11 kg ventilated plastic bags of carrots were prepared from each plot, and were put into storage at 5-6 °C for 13 or 18 weeks. One bag was assessed for infection at each sampling, after which statistical analysis was performed.

3.5 POSTHARVEST CONTROL OF S. SCLEROTIORUM

This experiment examined the incidence of sclerotinia disease in carrots treated with a postharvest dip prior to storage.

3.5.1 Postharvest disease control experiment 1985

A bulk sample of carrots was manually dug, topped, and cleaned as described previously. The treatments used consisted of an undipped control and dips of water, $CaCl_2$ (2.0 g/l), and benomyl (0.5 g ai/l). Treatments were replicated four times. The purpose of the H_2O dip was twofold: firstly, to see if water itself was an effective control treatment, and secondly, the water dip served as a check against the effects of the water component of the chemical treatments.

Each dipping treatment lasted for 5 minutes in 100 l of water or fungicide solution, after which the carrots were removed and packed into two 11 kg bags per replicate. Fresh solution was prepared for each treatment.

Two discs (1.0 cm diameter, 2.0 mm thickness) of sclerotinia-infected carrots were put into each bag as a source of disease. The samples were then placed into storage at 5-6 °C for 11 or 16 weeks. Assessment of the percentage of healthy and diseased carrots, by weight, was then completed, and statistical analysis was performed.

3.5.2 Postharvest disease control experiment 1986

In the 1986 storage season, machine harvested carrots (cvs. 'Paramount' and 'Six Pak II') from Jamor Farms were used. Four treatments were applied in 1986: an undipped treatment, a water dip, and benomyl dips of 0.25 and 0.5 g ai/l. Treatments were replicated four times. One 11 kg bag of carrots was prepared from each plot. Other experimental procedures were the same as the previous year. Carrots were stored for 14.5 weeks and assessed for disease, after which statistical analysis was performed.

Chapter IV

RESULTS AND DISCUSSION

4.1 STORAGE TEMPERATURE MONITORING STUDY

Data from the monitoring of the carrot storages showed that all three storages required an extended period to cool the crop to a desirable temperature (Appendices A-C). Carrot temperatures in both the 1985 and the 1986 storage seasons remained above the desired range of 0-1 °C for up to two months, depending on factors such as the length of harvest, the weather encountered during harvesting, and the refrigeration system used. In both storage seasons Connery's carrots cooled faster than either of the storages at Jamor Farms, probably due in part to differences in their refrigeration systems. Additionally, Jamor Farms also had trouble in the fall of 1985 with the efficiency of their refrigeration system. However, in many other ways the three storages were similar, and the data in Figs. 1-4, which cover temperatures monitored in the steel shed at Jamor Farms, essentially can be applied to any of the three storages.

Initial carrot temperatures were approximately 6-7 °C (Figure 1), and decreased to 3-6 °C over the period indicated. Filling into storage was occurring at this time, and as a consequence the temperatures were quite variable, with high peaks in the daytime. The variable temperatures resulted from the addition of uncooled carrots possessing a higher heat

Figure 1. Temperature monitoring of a carrot storage, steel shed, Jamor Farms, Portage la Prairie, MB, 19-29 September 1985

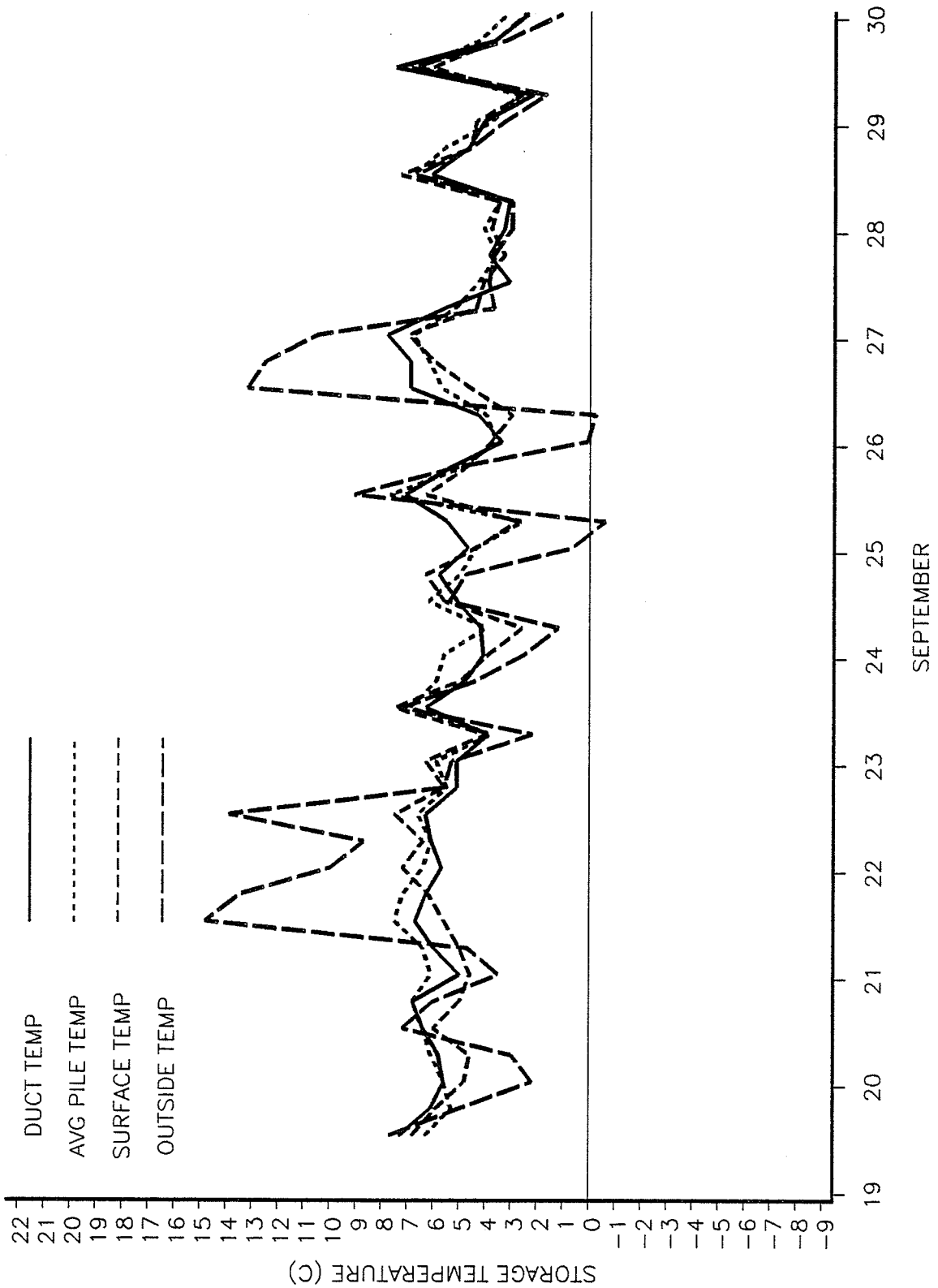
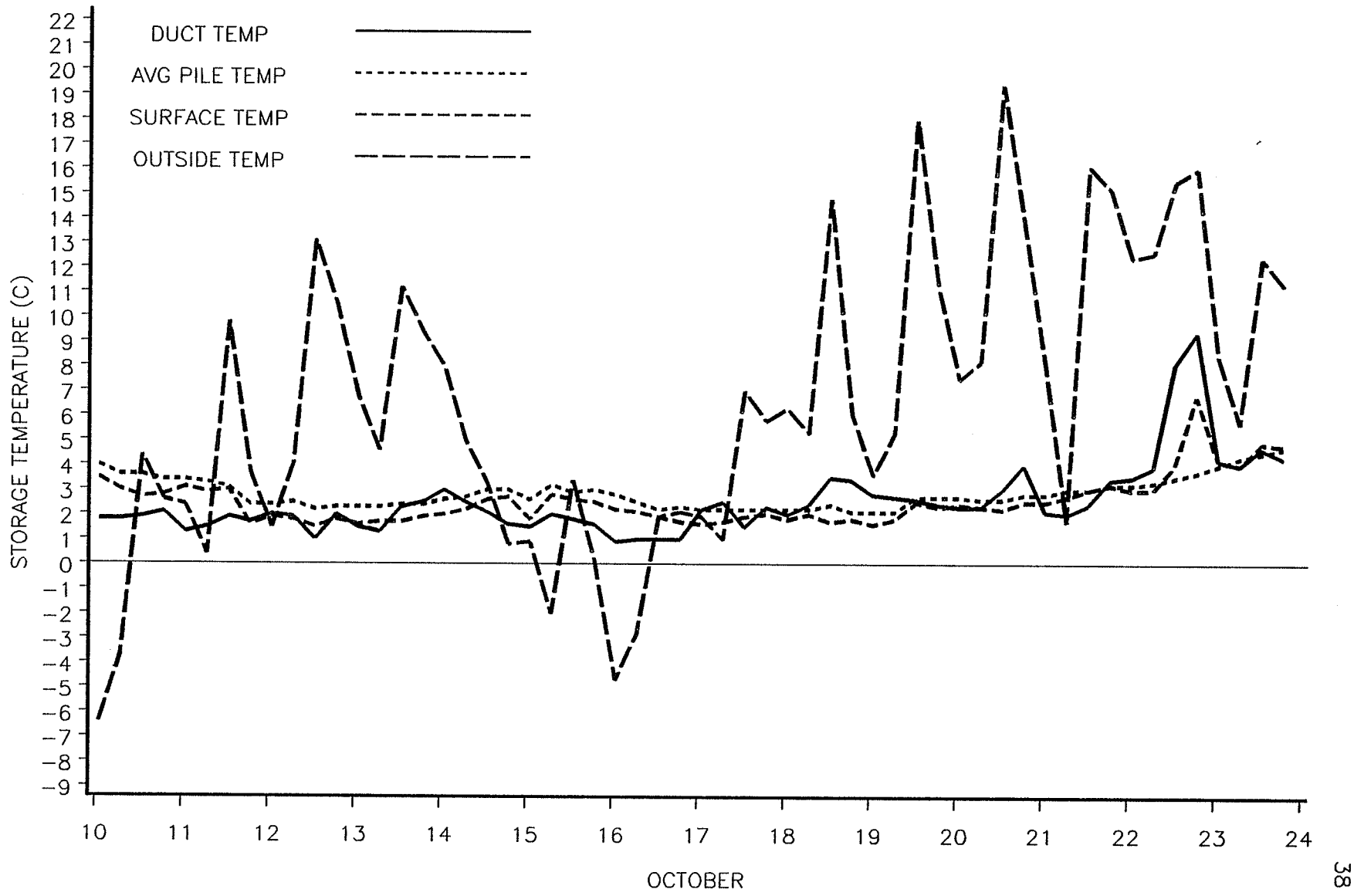

DUCT TEMP
AVG PILE TEMP
SURFACE TEMP
OUTSIDE TEMP
STORAGE TEMPERATURE (C)
22
21
20
19
18
17
16
15
14
13
12
11
10
9
8
7
6
5
4
3
2
1
0
−1
−2
−3
−4
−5
−6
−7
−8
−9
19
20
21
22
23
24
25
26
27
28
29
30
SEPTEMBER

Figure 2. Temperature monitoring of a carrot storage, steel shed, Jamor Farms, Portage la Prairie, MB, 10-23 October 1985


DUCT TEMP
AVG PILE TEMP
SURFACE TEMP
OUTSIDE TEMP
STORAGE TEMPERATURE (C)
22
21
20
19
18
17
16
15
14
13
12
11
10
9
8
7
6
5
4
3
2
1
0
−1
−2
−3
−4
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−6
−7
−8
−9
10
11
12
13
14
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19
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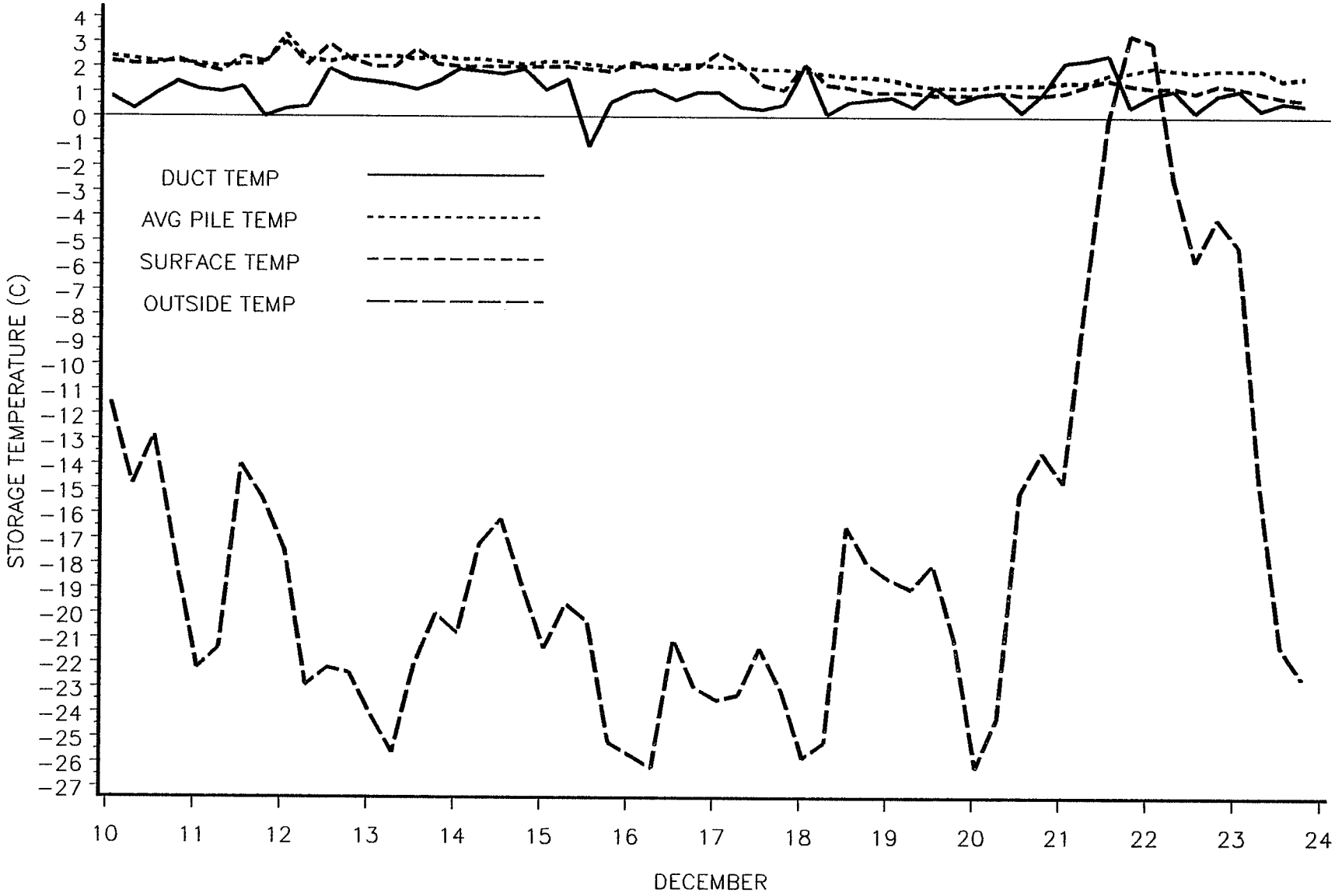
21
22
23
24
OCTOBER

Figure 3. Temperature monitoring of a carrot storage, steel shed, Jamor Farms, Portage la Prairie, MB, 10-23 December 1985


DUCT TEMP
AVG PILE TEMP
SURFACE TEMP
OUTSIDE TEMP
STORAGE TEMPERATURE (C)
4
3
2
1
0
−1
−2
−3
−4
−5
−6
−7
−8
−9
−10
−11
−12
−13
−14
−15
−16
−17
−18
−19
−20
−21
−22
−23
−24
−25
−26
−27
10
11
12
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16
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18
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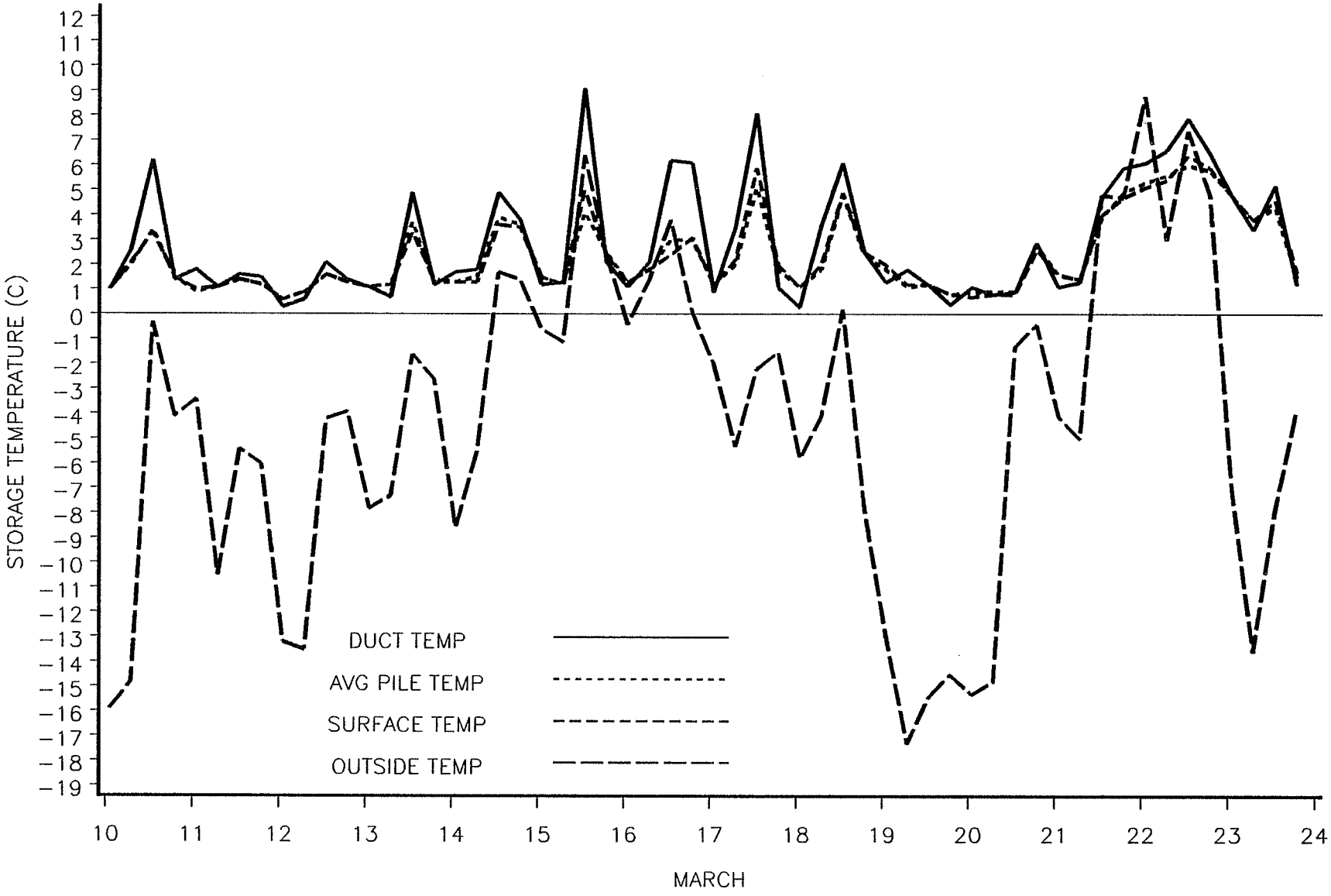
22
23
24
DECEMBER

Figure 4. Temperature monitoring of a carrot storage, steel shed, Jamor Farms, Portage la Prairie, MB, 10-23 March 1986


STORAGE TEMPERATURE (C)
12
11
10
9
8
7
6
5
4
3
2
1
0
−1
−2
−3
−4
−5
−6
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−17
−18
−19
DUCT TEMP
AVG PILE TEMP
SURFACE TEMP
OUTSIDE TEMP
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MARCH

load, as well as entry of warm air into storage through open doors. Further, the refrigeration was only used at night when filling was completed for the day.

October 1985 data for the steel shed at Jamor Farms (Figure 2) showed that average pile temperatures were 2.5-4 °C, instead of 0-1 °C (van den Berg and Lentz, 1973), which is recommended for the storage of carrots. Although the temperature of the carrots was still decreasing slowly in the first half of Figure 2, the temperature was high because the storage was still being filled. However, the daily swings in temperature shown in Figure 1 did not occur in Figure 2. In the second half of Figure 2 it can be seen that warm temperatures, combined with filling of the steel storage, caused the temperatures of the carrots to rise to about 4 °C.

Data two months later, from 10-23 December 1985 (Figure 3), indicated that outside temperatures were very low (generally below -13 °C). Average pile temperatures had decreased to 1.5-2 °C. Low outside temperatures in November and December (Appendix A), were probably responsible for reducing the average pile temperature from the 2.5-4 °C in October 1985, to 1.5-2 °C in December 1985. However, outside temperatures rose above freezing, and were higher than the pile temperatures, on 22 December. Subsequently the average carrot temperatures also rose, but did not immediately subside after outside temperatures dropped again.

Once outside temperatures were sufficiently low in winter, cold outside air was used in the steel shed. The data in Figure 3 reflect

temperatures recorded with outside air used as the cooling source. Use of outside air obviated need for use of the refrigeration coil in the steel shed, as cold air was ducted directly into the air plenums from outside. Connery's carrot storage and the wooden carrot storage at Jamor Farms did not use cold outside air for cooling.

The data in Figure 4 show storage temperatures in March 1986, at the end of the storage season. It can also be seen that outside temperatures were much more variable than in December. Duct temperatures, surface temperatures, and the average pile temperatures all showed dramatic daily swings in temperature. These changes reflect a combination of the effects of working in the steel shed with the doors open, in order to remove carrots for marketing, as well as the effects of outside temperatures above freezing for part of the period.

Monitoring of RH with a sling psychrometer in the three carrot storages, including the steel storage when outside air cooled the carrots, showed levels of 98-99%. The use of cold outside air can offer potential problems, because it has a low humidity. Under typical Canadian prairie conditions, outside atmospheres may have humidities as low as 10% (Krahn, 1974). Moisture can be added to the incoming air, but the humidifiers and the refigeration system have to be working well to ensure that the RH does not drop. Low RH will lead to dehydration and/or disease problems. Jamor Farms used misters in this storage to inject moisture into the airstream of the plenum, to ensure that RH levels did not drop. Misting was also done in the wooden storage and at Connery's Riverdale Farms, even though they did not utilize outside air. In addition, vegetables such as carrots, which possess a high

coefficient of transpiration, come to a rapid equilibrium of humidity in the pile at a point which approaches saturation (Lentz and van den Berg, 1973).

The temperature monitoring carried out in the three storages in 1985 and 1986 confirmed the importance of good storage management by growers in order to minimize sclerotinia disease in storage. In Manitoba, carrot storages are filled over a period of a number of weeks. Bulk storage is used for the carrots, with piles up to 4m in depth. The doors of the storage facility remain open all day during filling. Connery's were more often able to close their shed doors between loads of carrots than were Jamor Farms. It was rare for the doors to be closed until the end of the day at Jamor Farms. Having the storage doors open all day without being able to cool the carrots presented two problems for the growers. Firstly, carrot temperatures remained high, because outside heat and the field heat of the crop could only be removed at night for a few hours. Secondly, at the higher temperatures, the carrots had a higher heat of respiration, further adding to the heat load.

Field heat is the last of the heat loads to be removed from the crop. All other sources of heat must first be removed before the temperature of the carrots will be reduced. If the total heat load is very great (or the refrigeration system is not cooling efficiently), growers will find their carrots take longer to cool. This can be seen in Figure 2. When outside air temperatures were high from 18-24 October, the temperatures monitored in the storage also increased. During this period the average pile temperature increased 2.5 C°. Similarly,

temperatures in December 1985 (Figure 3) can be seen to decrease over a number of days until the outside temperature rose above 0 °C. Pile temperatures then rose slightly too, as the heat load, from the carrots and from outside temperatures, was too great for the refrigeration system to keep up.

Temperatures were reduced in storage when the outside temperature dropped, or when carrot harvesting was halted for several days for some reason. With the shed doors closed, the infiltration heat load was reduced, and more of the refrigeration capacity was available to reduce the temperature of the carrots and their heat of respiration. However, in spite of these drops, carrot temperatures remained high for nearly two months, even at Connery's Riverdale Farms, which was the fastest cooling storage. According to Ryall and Lipton (1979), carrots and other root crops should be cooled to storage temperatures the same day they are harvested. Figure 3 shows that average pile temperatures were still 1.5-2 °C three months after loading into storage began. The carrots had cooled to 1 °C by mid-November, two months after filling began, but by mid-December (Figure 3), temperatures were up to 1.5-2 °C. Pile temperatures cooled to below 1 °C by the end of December, however.

The air in the ducts was frequently warmer than the pile temperatures, thus increasing the temperature of the carrots. Neither Jamor Farms nor Connery's Riverdale Farms monitored their storage temperatures. This would be helpful, as it would allow a continuous check of conditions obtaining in the pile. The use of computer technology by the growers would allow them to switch their cooling source from outside air to a refrigeration coil or a Filacell

refrigeration system, or to have an alarm sound if duct temperatures rise above pile temperatures.

Development of sclerotinia became apparent at Jamor Farms within 4 weeks of filling the storage. The surfaces of the two storage piles were monitored, and showed that most infections began on the crown end or on the fine "tail" at the end of the tap root. Few infections started from wounds. As early as 4 weeks in storage, a mycelial weft began to spread from the infected carrots which acted as the foci of infection onto adjacent carrots.

Examination of the carrots being loaded into all three storages showed that only the occasional carrot was visibly infected with sclerotinia rot in the field; these were usually carrots which were visibly cracked or deformed in some manner prior to harvesting. S. sclerotiorum was isolated from sterilised slices of freshly harvested carrots to confirm the presence of the pathogen.

The effect of free water on decay in carrots was also noted. In agreement with van den Berg and Lentz (1966, 1974, 1978) and contrary to Krahn (1974), condensation did not lead to increased levels of sclerotinia disease among the carrots. Even where condensation from refrigeration equipment dripped onto the carrot pile, decay was not increased.

Visual observations of the carrots were not carried out at Connery's Riverdale Farms after filling of the storage, as the structure of the facility did not allow for access to the top of the carrot pile.

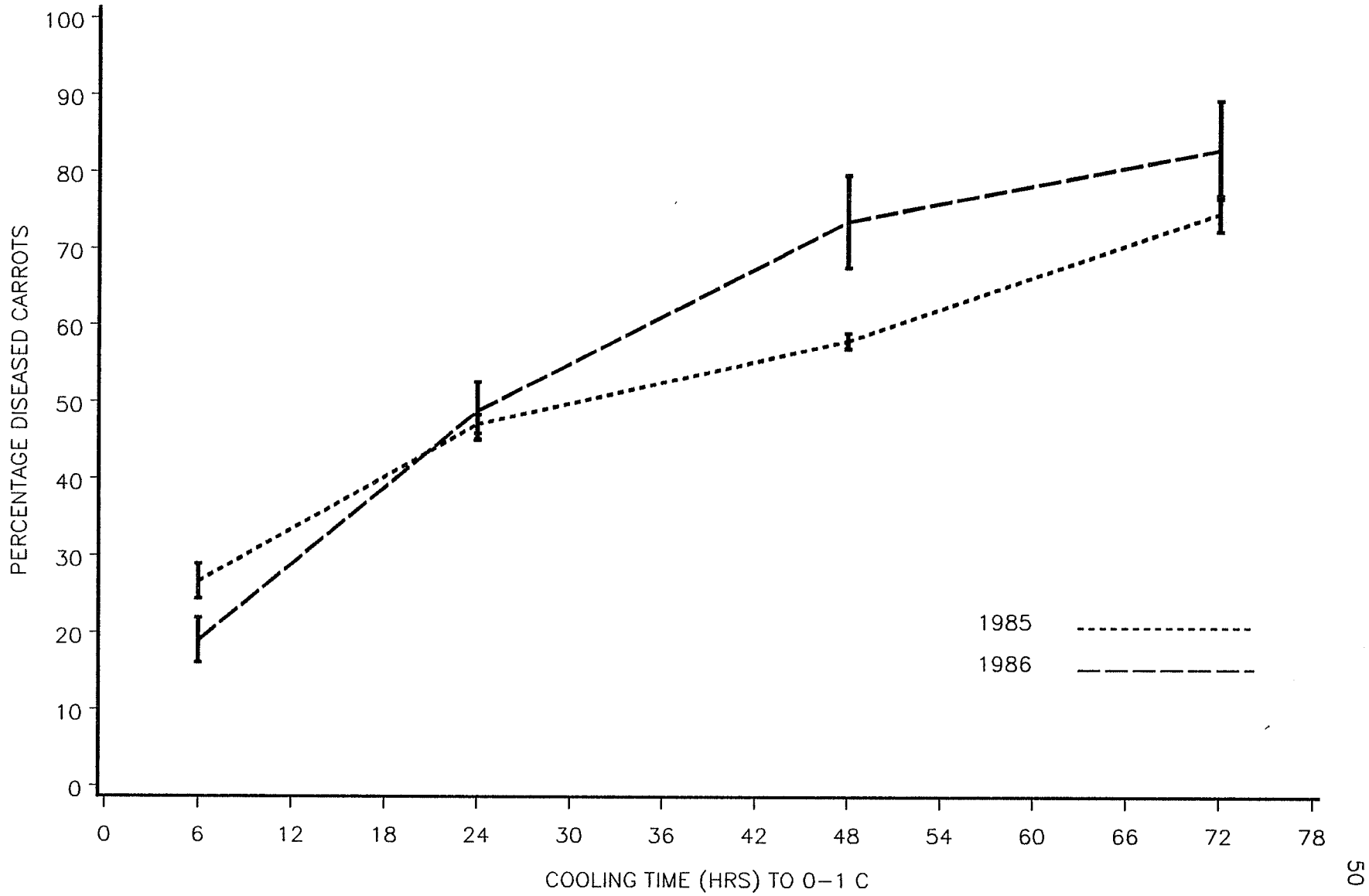
4.2 COOLING STUDY

In 1985 the carrots cooled in the shortest time of 6 hours had the least losses from sclerotinia disease after 15 weeks storage, and disease development increased as the length of the cooling period increased up to 72 hours (Figure 5). Differences were highly significant between each of the 4 cooling times. Two storage periods were intended to be used for the carrots in 1985. However, because of the advancement of sclerotinia rot in the carrots, all carrots were removed from storage after 15 weeks.

In 1986 only one storage period was used for each of the 4 cooling times. Once again the 6 hr treatment had the least amount of losses from infection by S. sclerotiorum after 14.5 weeks storage. Differences were highly significant between the 6, 24, and 48 hr cooling treatments, while the carrots cooled in 48 or 72 hrs were not different significantly from each other.

Only healthy carrots with no signs of infection were used in 1985 and 1986, although the carrots were not physically graded out to supermarket quality. In all other ways the carrots handled in the study were intended to be similar to those handled commercially. The occasional carrot with a small wound similar to those which would escape culling out during piling into a commercial storage at harvest time was also included. Conditions differed considerably from those of commercial storages, since the carrots were stored in plastic bags, not in bulk. The carrots were also packed into the bags, thus being in closer contact with each other than the random and looser piling prevailing within the storages. This would allow rapid spread of infection outward from the initial point or points of infection in the bags.

Figure 5. Sclerotinia disease in carrots cooled to 0-1 °C at various rates and stored at 5-6 °C for 15 weeks (1985) or 14.5 weeks (1986)


PERCENTAGE DISEASED CARROTS
100
90
80
70
60
50
40
30
20
10
0
0
6
12
18
24
30
36
42
48
54
60
66
72
78
COOLING TIME (HRS) TO 0–1 C
1985
1986

Visual observations of the carrots in 1985 and 1986 showed that disease development first occurred at either the crown end or the tip end of the carrot. Since only carrots with no visible signs of infection were included in the experiment, it is likely that infection at the crown end resulted from entrance of mycelium into senescing or damaged leaf tissue, and from there it moved down the petiole into the susceptible xylem parenchyma cells of the crown (Lewis and Garrod, 1983). This infection was consistent with the process noted by Geary (1978) and Finlayson et al. (1989a). Mycelial infection at the tip end occurred most often when the tiny "tail" present at the tip of the tap root was damaged or broken off. Infection by the fungus via entrance at sites of wounding along the length of the root was not a major cause of disease, as the wounded carrots were not preferentially infected by the fungus. In some cases primary sites of infection could not be established.

This study demonstrated that temperature and cooling have an important effect in minimizing infection by S. sclerotiorum in stored carrots. Results are consistent with those of several others. Finlayson et al. (1989b) correlated the electrolyte loss in carrot discs with the aggressiveness of S. sclerotiorum at different temperatures. Using temperatures of 2, 4, 6, 8, 10, 15, and 20 °C, they found that much more electrolyte loss (relative to the control), and therefore more tissue damage due to S. sclerotiorum, occurred at the higher temperatures. Electrolyte loss in the carrot discs rapidly decreased from 20°C down to 6 °C. From 6 °C down to 2 °C there was no significant decrease in electrolyte loss in the carrot discs. Hence 6 °C would seem

to be a significant threshold below which growers should strive to keep the temperature of their carrots.

The cooling studies of 1985 and 1986 demonstrate that temperature plays an important role in keeping stored carrots free of infection by sclerotinia disease,and together with the temperature monitoring study emphasize the necessity of rapid cooling of carrots. At harvest the doors to the carrot storages in Manitoba remain open all day while carrots are loaded in. Cooling cannot be accomplished while this is done. It is optimistic to expect a carrot storage with many tonnes of carrots to be able to cool carrots to 0-1 °C within six hours. However, as Figure 5 demonstrates, there is a near linear effect between the cooling undergone by the stored carrots and the amount of losses from sclerotinia disease. Reducing the temperature of the carrots in commercial storage as quickly as possible will reduce the chance of losses due to infection by S. sclerotiorum.

4.3 COMPARISON OF FIELD TREATMENTS FOR CONTROL OF S. SCLEROTIORUM

Fungicides were applied to carrots in the field to assess their effectiveness in reducing the incidence of sclerotinia rot in storage.

Vinclozolin was sprayed twice during the growing season. The first spray earlier in the season (26 June), while the canopy was still open, was intended to prevent an early infection of the carrot which would localize itself in the crown. The second spray was applied on 24 July after the canopy had closed. By this time the leaves were beginning to touch each other, and some older leaves were beginning to senesce and touch the ground, allowing mycelial infection from the ground.

In 1985 vinclozolin reduced significantly the amount of disease that developed in the stored carrots which had been inoculated with sclerotia in the field, compared to inoculated carrots not treated with the fungicide (Table 1). Only 1% by weight of the roots of plants treated with fungicide showed evidence of sclerotinia disease after 11 weeks of storage, compared to 44% and 51% for the untreated inoculated and the uninoculated treatments respectively. After 17 weeks 3% of the vinclozolin-treated carrots were diseased, versus 48% and 57% for the untreated inoculated and the uninoculated carrots, respectively.

Two storage periods were used in order to test the effectiveness of the treatments over time. There was a similar trend for both storage periods, with the vinclozolin treatment giving markedly better control of S. sclerotiorum. Although differences were not significant, there was a slight increase in disease development for the longer storage period of 17 weeks. More disease development with a longer storage period is to be expected. However, most of losses from sclerotinia disease occurred during the first 11.5 weeks, with a smaller increase in disease occurring in the next 5.5 weeks. This is probably a result of sclerotinia disease development during the initial period when carrots were cooling to storage temperatures of 5-6°C. Lewis and Garrod (1983) report that the resistance of carrot roots to infection is very high, with the periderm and pericycle being particularly impenetrable. Thus it is also possible that the initial large increase in disease in the first 11.5 weeks, followed by a smaller in the next 5.5 weeks, is due to preferential spread of the fungus within the carrots it had already infected, rather than surmounting periderm barriers to infect other

Table 1. Effect of field-applied vinclozolin fungicide on sclerotinia rot in carrots stored at 5-6 °C for 11.5 or 17 weeks in 1985

Treatment	Diseased carrots (% by wt)[1]	
	11.5 Weeks	17 Weeks
Uninoculated	51 a[2]	57 a
Inoculated[3]	44 a	48 a
Inoculated + Vinclozolin[4]	1 b	3 b

[1] Values are means of 4 observations per treatment.

[2] Means within columns followed by same letter are not different significantly using Duncan's Multiple Range Test.

[3] 25 g of sclerotia were applied per plot as inoculum.

[4] Vinclozolin applied on 26 June and 24 July at a dosage of 0.42 l ai/ha each time.

carrots. However, it is more likely that the initial slow cooling, rather than barriers to infection, accounts for the differences in disease development between the two storage periods.

The uninoculated treatment was intended to act as a control to ascertain what levels of inoculum were naturally prevalent in the field. There was no significant difference in disease development in the stored carrots between this treatment and the inoculated treatment after both the 11.5 and the 17 week storage periods. Environmental conditions may not have been conducive to good germination and growth of the sclerotia in the inoculated treatments. 1985 was a wet year, and it is possible that the sclerotia rotted. Upon examination of the plots during the growing season, no sclerotia were found where they had been placed in the rows, and no apothecia were ever observed.

Since differences in the amount of disease development in storage between the vinclozolin treatment and the treatments which did not receive fungicide were so dramatic, it was decided to expand the study in 1986, using two different times and rates of application of vinclozolin, and adding a treatment of benomyl. No uninoculated treatments were used in 1986. The time of fungicide application was changed from June/July in the 1985 experiments to shortly before harvest in 1986 for several reasons. First was that there were no spray recommendations for use on carrots in Manitoba, since the chemical is not registered here. Secondly, Tahvonen (1985) had obtained good control of disease by spraying the tops of carrots the day before harvest, using benomyl, thiophanate methyl, or vinclozolin, and it was decided to move the time of application closer to the date of harvest for this reason. Only one spray application was made per treatment.

The early application of vinclozolin on 3 Sept was for the purpose of controlling infection developing from senescing leaves during the season. The late application on 30 Sept shortly before harvest probably reduced inoculum levels, but it did not prevent the earlier occurring infections via the crown. The 1986 experiments showed that the carrots receiving the higher dosage of vinclozolin (0.84 l ai/ha) on 30 Sept, had the least losses from sclerotinia disease in storage (Table 2). The high level of vinclozolin applied early (3 Sept), and the benomyl (1.0 kg ai/ha) sprayed on 30 Sept, provided less control of S. sclerotiorum, while the low dosage of vinclozolin (0.42 l ai/ha) applied on 30 Sept was the least effective treatment. The control, which received no application of fungicides, had significantly fewer healthy carrots after both 13 and 18 weeks of storage. These trends were similar for all treatments for both storage periods, although higher disease losses were found in the 18 week storage period.

Timing of spraying is apparently not a critical factor in control of sclerotinia disease in stored carrots. Good results were obtained by spraying earlier in the season, as well as shortly before harvest. Timing of spray applications is known to be much more critical in several other crops, as the epidemiology of the disease dictates timing of fungicide application. Natti (1971) demonstrated maximum control of white mold (S. sclerotiorum) in bean blossoms when benomyl was applied 3-7 days before full bloom (prebloom) and at full bloom. This period is the time when buds and blossoms are at an active stage of development. Timing is also critical in tomatoes, as benomyl applied after the crop canopy closed failed to penetrate (Rainbow, 1970). Additionally, this

Table 2. Effect of field-applied vinclozolin or benomyl fungicide on sclerotinia rot in carrots stored at 5-6 °C for 13 or 18 weeks in 1986

Treatment[1]	Diseased carrots (% by wt)[2]	
	13 Weeks	18 Weeks
Control	42 a[3]	54 a
Benomyl (1.0 kg ai/ha) - 30 Sept	14 bc	26 b
Vinclozolin (0.84 l ai/ha) - 3 Sept	11 c	16 c
- 30 Sept	2 d	3 d
Vinclozolin (0.42 l ai/ha) - 30 Sept	19 b	30 b

[1] All plots inoculated with 250 ml of sclerotinia-infected millet inoculum.

[2] Values are means of 4 observations per treatment.

[3] Means within columns followed by same letter are not different significantly using Duncan's Multiple Range Test.

closed canopy also gave a more humid environment for development of S. sclerotiorum. Tahvonen (1985) reduced sclerotinia and botrytis disease in stored carrots by spraying the tops with benomyl, thiophanate methyl, or vinclozolin on the day before harvest. Fungicide residues were low in the carrot roots, because the short time between spraying and harvesting did not allow movement of the fungicides into the roots.

Since the application of fungicides in the field is an effective method of controlling sclerotinia disease in storage, it would appear that good coverage of the carrot plant is essential. Further studies could determine whether best coverage is obtained by coverage of the crown region where S. sclerotiorum is established at harvest, or by targeting the senescing leaves which can become infected when touching the ground. While timing of fungicide applications does not appear to be critical, further studies could clarify when during the growing season spraying would be of most benefit. Further investigations into field control of infection of stored carrots by S. sclerotiorum should also examine the number of sprays, the volume of water used, and nozzle arrangements needed for best control of the fungus to determine what protection measures are of maximum efficacy. Until vinclozolin is registered for use on carrots in Manitoba it is probably not very useful to continue investigations of control of S. sclerotiorum using this fungicide.

4.4 POSTHARVEST TREATMENTS FOR CONTROL OF S. SCLEROTIORUM

Results of the postharvest dipping study in 1985 showed that after storage for 11 weeks there were no differences between any of the four

treatments in the amount of sclerotinia development, although the water dipped and benomyl treatments had a lower percentage of diseased roots (Table 3). A great deal of variability was present in both the undipped control and the $CaCl_2$ treatments after 11 weeks of storage in 1985. Hence, even though disease levels were quite different between the H_2O and benomyl treatments versus the control and $CaCl_2$ treatments (Table 3), the amount of variability between replicates negated significant differences for the treatments after 11 weeks of storage. After 16 weeks of storage the control and the $CaCl_2$ treatments had significantly more disease development than the benomyl and the water dip treatments. The control and the $CaCl_2$ treatments were not significantly different from each other, however, nor were the benomyl and the water dip.

Sclerotia formed on some of the rotted carrots in all treatments. The sclerotia which formed in the bags treated with calcium chloride showed an abnormal morphology, being smaller in size than the sclerotia in the other treatments, only 1.5-2 mm in diameter. However, when the abnormal sclerotia were germinated on plates of PDA and allowed to form new sclerotia, these were normal in size.

Calcium is an important constituent of the cell and its proper functioning. Calcium chloride was chosen as a treatment to ascertain if it would aid in the maintenance of the integrity of the carrot periderm during long term storage, and under attack by S. sclerotiorum. It has been used in treating apples in the commercial apple industry to promote periderm integrity and increase storage ability, and was included in the 1985 experiments on carrots for this reason.

Table 3. Effect of prestorage dips on sclerotinia disease in carrots stored at 5-6 °C for 11 or 16 weeks in 1985

Treatment	Diseased carrots (% by wt)	
	11 Weeks	16 Weeks
Undipped Control	23[1] a[2]	35 a
H_2O-Dipped	5 a	17 b
$CaCl_2$ (2.0 g/l)	22 a	36 a
Benomyl (0.5 g ai/l)	5 a	6 b

[1] Values are means of four observations per treatment.

[2] Means within columns followed by same letter are not different significantly using Duncan's Multiple Range Test.

The failure of $CaCl_2$ to reduce sclerotinia development in stored carrots is contrary to the work of Skou (1971), who obtained good results with a treatment of 1.5-2.0 g $CaCl_2 \cdot 2H_2O$/l on both irradiated and nonirradiated carrots. He found that the benefits of the calcium dips increased as the amount and severity of wounds increased. Calcium reduced the effect of oxalic acid and pectic enzymes, both of which are produced by S. sclerotiorum. It had been expected that dipping carrots in a calcium chloride solution would show a similar protective effect, but this was not the case. It also might have been expected that if the $CaCl_2$ failed to work, the water component of the $CaCl_2$ solution might have functioned similarly to the water dip. However, the amount of disease present in the $CaCl_2$-dipped carrots after storage for 11 or 16 weeks, was instead similar to those shown by the undipped carrots, where there was no effect of water (Table 3).

In 1986 no calcium chloride dip was used, and another benomyl dip, at half the previous dosage, was added. After 14.5 weeks of storage, the high level of benomyl (0.5 g ai/l) was the only treatment to reduce significantly the development of sclerotinia disease (Table 4). The two levels of benomyl did not result in significantly different disease development. In addition, the control, the H_2O-dipped, and the low benomyl treatments were not significantly different from each other.

It is obvious from this study over two years that benomyl postharvest dips can protect stored carrots from infection by S. sclerotiorum. Even though variability in replicates in the carrots stored for 11 weeks in 1985 negated any significant differences, benomyl applied at 0.5 g ai/l was different significantly after 16 weeks in 1985, and was also

Table 4. Effect of prestorage dips on sclerotinia disease in carrots stored at 5-6 °C for 14.5 weeks in 1986 for 14.5 weeks in 1986

Treatment	Diseased carrots (% by weight)
Undipped Control	26[1] a[2]
H_2O-Dipped	23 a
Benomyl -(0.25 g ai/l)	21 ab
-(0.5 g ai/l)	17 b

[1] Values are means of four observations per treatment.

[2] Means within columns followed by same letter are not significantly different at the .05 level (Duncan's Multiple Range Test).

different in 1986. Part of the benefit of dipping, whether in water or in fungicide solution, may be from the effect of cold water in aiding removal of field heat from the carrots.

The water dip was not effective in reducing the amount of sclerotinia rot in stored carrots. In 1985, disease development in the H_2O-dipped treatments was not significantly different from the control after 11 weeks, although there were differences after 16 weeks storage. The H_2O-dipped treatment was not significantly different from the control in 1986. However, depending on the economics of the additional cost of using benomyl as a dip for carrots versus the additional protection given, growers may decide that dipping their carrots in water alone to aid in precooling the roots before storage provides enough added protection. With any dipping or washing of carrots it is imperative that the water or solution used not get too dirty (Eckert and Ogawa, 1983). Infective propagules may build up and subsequently enter via wounds and susceptible tissues.

A factor which may account for the resistance of growers to use dipping as means of protecting their carrots from sclerotinia disease is that harvest time is an extremely busy time for them. Labor is at a premium, and the labor used for dipping would be taken away from other tasks which need to be done before the end of harvest and winter. Growers have so far been unwilling to switch priorities in their allocation of labor. However, if so, it is possible that they could harvest fewer carrots to obtain the same amount of carrots free of disease at the end of the storage period.

Chapter V

GENERAL DISCUSSION

The results and observations of this study verify that losses from infection of stored carrots by Sclerotinia sclerotiorum are related to grower management, both in the field and in storage.

The cooling studies firmly demonstrated that rapid cooling from ambient field temperatures to storage temperatures of 0-1 °C is of paramount importance. The 6 hour cooling time provided the lowest disease levels from infection by S. sclerotiorum, while the increasingly longer cooling times gave increasingly higher levels of sclerotinia disease. Finlayson et al. (1989a) showed that electrolyte loss in carrot discs was a simple and accurate measurement of infection by S. sclerotiorum, and that the growth of the organism increased sharply above 6 °C. This temperature would appear to be a threshold below which growers should strive to cool their carrots as quickly as possible after harvest, with temperatures of 0-1 °C optimum for storage.

Temperature monitoring of the three carrot storages showed that the carrot piles were not reaching optimum temperatures for several weeks. While it is obvious that carrot storages containing many tonnes of carrots will not cool to desirable temperatures in 6 hours, or even several days, the data from the cooling rate study and the temperature monitoring study together emphasize the importance of cooling carrots in storage as quickly as possible. Carrot storages contain not only

carrots that are partially cooled, but also freshly harvested roots at a higher temperature that have a higher heat of respiration. Temperature monitoring does not reflect this unless monitoring probes are continuously maintained in the newly loaded portions of the carrot pile. Growers in Manitoba do not monitor their carrot piles and storages at all, but it would be to their benefit to do so.

Air flow in carrot storages should be the minimum that will allow control of temperature and humidity. However, increased air velocities during the initial cooling period would aid in removing heat. Closing the storage doors whenever possible is another measure which will aid in lowering storage temperatures by minimizing heat infiltration.

Observations of carrots in both the cooling rate study and the temperature monitoring study indicated that infection of the carrot roots began either in the crown region of the carrot or, to a slightly lesser extent, in the tiny "tail" at the end of the taproot upon damage to the root. Damage to the crop is almost impossible to avoid in the mechanical harvesting of carrots. Tucker (1974) demonstrated the effect of mechanical harvesting on the storage performance of carrots and showed that the prestorage condition of the periderm is an important factor in maintaining the quality of carrots in storage. Finlayson et al. (1989b) have shown that mycelium of S. sclerotiorum infects carrots in the field during the growing season via senescing leaves and petioles touching the ground, whence it travels down the petiole to localize in the crown by the end of the growing season. However, the spread of sclerotinia rot occurs almost exclusively in storage. Observations during the temperature monitoring study showed that mycelium appeared

within 4 weeks of loading into storage, spreading outward from the foci of infection as the length of the storage season advanceed. The crucial factor triggering the progress of the organism from a dormant parasite to a destructive pathogen, may well be stimuli caused by the damage to the carrot crown provided by the harvesting machine as it tops the carrots. Additional stimuli for development of active infection may also be provided by the change in environment, from growth of the carrot in the ground to the abrupt cessation of growth with the shift to a storage environment.

However, if good management practices and fast cooling rates do not suffice to keep levels of S. sclerotiorum infection low in storage, growers may want to utilize preharvest or postharvest treatments of the carrots to control S. sclerotiorum. Application of benomyl and vinclozolin provided good control of the disease in storage. However, the use of vinclozolin is not a registered treatment for carrots in Manitoba. Further investigations into the use of field applications for the control of S. sclerotiorum, using benomyl, could include such factors as timing and number of sprays, volume of water used, and the spray nozzle arrangement needed for best control of sclerotinia disease.

The use of postharvest fungicide dips did not appear to be as successful as field application of fungicides, as it provided less control of S. sclerotiorum in the stored carrots. In addition, the acceptance of dipping will depend upon the willingness of growers to allocate time and labor to this task during the busy harvest season.

BIBLIOGRAPHY

Abawi, G.S., and R.G. Grogan. 1975. Source of primary inoculum and effects of temperature and moisture on infection of beans by Whetzelinia sclerotiorum. Phytopathology 65:300-309.

Abawi, G.S., and R.G. Grogan. 1979. Epidemiology of diseases caused by Sclerotinia species. Phytopathology 69:899-904.

Adams, P.B., and W.A. Ayers. 1979. Ecology of Sclerotinia species. Phytopathology 69:896-899.

Adams, P.B., and C.J. Tate. 1976. Mycelial germination of sclerotia of Sclerotinia sclerotiorum on soil. Plant Dis. Rep 60:515-518.

Anonymous. 1988. Field Crop Production Guide for Manitoba 1988-90. Manitoba Agriculture. 102 pp.

Anonymous. 1989. Vegetable Crop Production Guide 1989-90. Manitoba Agriculture. Agdex 250-32.

Apeland, J., and H. Hoftun. 1974. Effects of temperature-regimes on carrots during storage. Acta Hort. 38:291-308.

Bateman, D.F., and R.D. Lumsden. 1965. Relation of calcium content and nature of the pectic substances in bean hypocotyls of different ages to susceptibility to an isolate of Rhizoctonia solani. Phytopathology 55:734-738.

Bateman, D.F., and R.L. Millar. 1966. Pectic enzymes in tissue degradation. Annu. Rev. Phytopath. 4:119-146.

Ben-Yephet, Y. 1988. Control of sclerotia and apothecia of Sclerotinia sclerotiorum by metham-sodium, methyl bromide and soil solarization. Plant Pathol. 37:25-27.

Boland, G.J., and R. Hall. 1982. White mold of soybeans. Can. J. Plant Pathol. 4:304.

Boland, G.J., and R. Hall. 1986. Growthroom evaluation of soybean cultivars for resistance to Sclerotinia sclerotiorum. Can. J. Plant Sci. 66:559-564.

Boland, G.J., and R. Hall. 1987. Epidemiology of white mold of soybean in Ontario. Can J. Plant Pathol. 9:218-224.

Burton, W.G. 1977. Storage requirements as determined by potential deterioration. Ann. Appl. Biol 85:157-158.

Coley-Smith, J.R., and R.C. Cooke. 1971. Survival and germination of fungal sclerotia. Annu. Rev. Phytopathol. 9:65-92.

Conway, W.S., and C.E. Sams. 1987. The effects of postharvest infiltration of calcium, magnesium, or strontium on decay, firmness, respiration, and ethylene production in apples. J. Amer. Soc. Hort. Sci. 112:300-307.

Cook, G.E., J.R. Steadman, and M.G. Boosalis. 1975. Survival of Whetzelinia sclerotiorum and initial infection of dry edible beans in Western Nebraska. Phytopathology 65:250-255.

Crete, R. 1980. Diseases of Carrots in Canada. Agriculture Canada. Pub. 1615.

Davies, W.P. 1977. Infection of carrot roots in cool storage by Centrospora acerina. Ann. Appl. Biol. 85:163-164.

Davies, W.P., B.G. Lewis, and J.R. Day. 1981. Observations on infection of stored carrot roots by Mycocentrospora acerina. Trans. Br. Mycol. Soc. 77:139-151.

Dennis, C. 1977. Susceptibility of stored crops to microbial infection. Ann. Appl. Biol. 85:430-432.

Derbyshire, D.M. 1973. Post harvest deterioration of vegetables. Chemistry and Industry (17 Nov):1052-1054.

Derbyshire, D.M., and A.F. Crisp. 1978. Studies on treatments to prolong the storage life of carrots. Experimental Hort. 30:23-28.

Deverall, B.J., and R.K.S. Wood. 1961. Infection of bean plants (Vicia fabae L.) with Botrytis cinerea and B. fabae. Ann. Appl. Biol. 49:461-472.

Diatchenko, V.S. 1974. Methods of controlling carrot and onion diseases under storage conditions. Acta Hort. 38:397-410.

Djacenko, V.S. 1971. Some biological peculiarities and storage methods of carrots. Acta Hort. 20:80-91.

Dueck, J., R.A.A. Morrall, and D.L. McKenzie. 1983. Control of Sclerotinia sclerotiorum in rapeseed with fungicides. Can. J. Plant Pathol. 5:289-293.

Echandi, E., and J.C. Walker. 1957. Pectolytic enzymes produced by Sclerotinia sclerotiorum. Phytopathology 47:303-306.

Eckert, J.W. 1977. Control of postharvest diseases. In Antifungal Compounds. ed. M.D. Siegel and H.D. Sisler. New York: Marcel Dekker. pp. 269-352.

Eckert, J.W., and J.M. Ogawa. 1983. The chemical control of postharvest diseases: deciduous fruits, berries, vegetables and root/tuber crops. Ann. Rev. Phytopathol. 26:433-469.

Finlayson, J.E., M.K. Pritchard, and S.R. Rimmer. 1989a. Electrolyte leakage and storage decay of five carrot cultivars in response to infection by Sclerotinia sclerotiorum. Can. J. Plant Pathol. 11:313-316.

Finlayson, J.E., S.R. Rimmer, and M.K. Pritchard. 1989b. Infection of carrots by Sclerotinia sclerotiorum. Can. J. Plant Pathol. 11:242-246.

Geary, J. 1978. Host:parasite interactions between the cultivated carrot (Daucus carota L.) and Sclerotinia sclerotiorum ((Lib.) de Bary). Ph.D. Thesis. University of East Anglia. U.K. 220 pp.

Geeson, J.D., K.M. Browne and H.P. Everson. 1988. Storage diseases of carrots in East Anglia 1978-82, and the effects of some pre- and post-harvest factors. Ann. Appl. Biol. 112:503-514.

Goodliffe, J.P., and J.B. Heale. 1977. Factors affecting the resistance of cold-stored carrots to Botrytis cinerea. Ann. Appl. Biol. 87:17-28.

Goodliffe, J.P., and J.B. Heale. 1978. The role of 6-methoxy mellein in the resistance and susceptibility of carrot root tissue to the cold-storage pathogen Botrytis cinerea. Physiol. Plant Pathol. 12:27-43.

Grau, C.R., and V.L. Radke. 1984. Effects of cultivars and cultural practices on sclerotinia stem rot of soybean. Plant Dis. 68:56-58.

Grierson, W., and W.F. Wardowski. 1978. Relative humidity effects on the postharvest life of fruits and vegetables. HortScience 13:570-574.

Hancock, J. 1966. Degradation of pectic substances associated with pathogenesis by Sclerotinia sclerotiorum in sunflower and tomato stems. Phytopathology 56:975-979.

Hawthorne, B.T. 1974. Sclerotinia minor on lettuce: effect of plant growth on susceptibility to infection. N.Z. J. Agric. Res. 17:387-392.

Hawthorne, B.T., and W.R. Jarvis. 1973. Differential activity of fungicides on various stages in the life cycle of Sclerotinia spp. N.Z. J. Agric. Res. 16:551-557.

Heale, J.B., V. Harding, K. Dodd and P.B. Gahan. 1977. Botrytis infection of carrot in relation to the length of the cold storage period. Ann Appl. Biol. 85:453-457.

Huang, H.C. 1982. Morphologically abnormal sclerotia of Sclerotinia sclerotiorum. Can. J. Microbiol. 28:87-91.

Huang, H.C. 1985. Factors affecting myceliogenic germination of Sclerotinia sclerotiorum. Phytopathology 75:433-437.

Huang, H.C., and J. Dueck. 1980. Wilt of sunflower from infection by mycelial-germinating sclerotia of Sclerotinia sclerotiorum. Can. J. Plant Pathol. 2:47-52.

Huang, H.C., M.J. Kokko and L.M. Phillippe. 1988. White mold of dry bean (Phaseolus vulgaris L.) in southern Alberta, 1983-87. Can. Plant Dis. Surv. 68(1):11-13.

Imolehin, E.D., R.G. Grogan and J.M. Duniway. 1980. Effect of temperature and moisture tension on growth, sclerotial production, germination, and infection by Sclerotinia minor. Phytopathology 70:1153-1157.

Jensen, A. 1971. Storage diseases of carrots, especially Rhizoctonia crater rot. Acta Hort. 20:125-129.

Kohn, L.M. 1979. Delimitation of the economically important plant pathogenic Sclerotinia species. Phytopathology 69:881-886.

Krahn, R. 1974. Long term storage of perishable vegetables. Acta Hort. 38:443-450.

Lauritzen, J.I. 1932. Development of certain storage and transit diseases of carrots. J. Agr. Res. 44:861-912.

Lentz, C.P. 1966. Rates of moisture loss of carrots under refrigerated storage conditions. Food Technol. 20(4):201-204.

Lentz, C.P., and L. van den Berg. 1973. Factors affecting temperature, relative humidity and moisture loss in fresh fruit... and vegetable storage. ASHRAE Journal 15:55-60.

Lentz, C.P., L. van den Berg, and R.S. McCullough. 1971. Study of factors affecting temperature, relative humidity, and moisture loss in fresh fruit and vegetable storages. Can. Inst. Food Technol. J. 4(4):146-153.

Le Tourneau, D. 1979. Morphology, cytology, and physiology of Sclerotinia species in culture. Phytopathology 69:887-890.

Lewis, B.G., and B. Garrod. 1983. Carrots. In Post-Harvest Pathology of Fruits and Vegetables. ed. C. Dennis. Toronto: Academic Press. pp. 103-124.

Lewis, B.G., W.P. Davies and B. Garrod. 1981. Wound-healing in carrot roots in relation to infection by Mycocentrospora acerina. Ann. Appl. Biol. 99:35-42.

Lockhart, C.L., and R.W. Delbridge. 1972. Control of storage diseases of carrots by washing, grading, and post-harvest fungicide treatments. Can. Plant Dis. Surv. 52:140-142.

Lockhart, C.L., and R.W. Delbridge. 1974. Control of storage diseases of carrots with postharvest fungicide treatments. Can. Plant Dis. Surv. 54(2):52-54.

Lumsden, R.D. 1979. Histology and physiology of pathogenesis in plant diseases caused by Sclerotinia species. Phytopathology 69:890-896.

Maxwell, D.P., and R.D. Lumsden. 1970. Oxalic acid production by Sclerotinia sclerotiorum in infected bean and in culture. Phytopathology 60:1395-1398.

Mazza, G. 1989. Carrots. In Quality and Preservation of Vegetables. ed. N.A.M. Eskin. Boca Raton, FL: CRC Press. pp. 75-119.

McLean, D.M. 1958. Role of dead flower parts in infection of certain crucifers by Sclerotinia sclerotiorum (Lib.) D. By. Plant. Dis. Rep. 42:663-666.

Menzies, J.G. 1983. Variability and pathogenicity of Sclerotinia sclerotiorum and the effects of host seedling exudates on sclerotial germination. M.Sc. Thesis. University of Manitoba, Winnipeg, Manitoba. 127 pp.

Natti, J.J. 1971. Epidemiology and control of bean white mould. Phytopathology 61:669-674.

Partyka, R.E., and W.F. Mai. 1962. Effects of environment and some chemicals on Sclerotinia sclerotiorum in laboratory and potato field. Phytopathology 52:766-770.

Phan, C.T., H. Hsu, and S.K. Sarkar. 1973. Physical and chemical changes occurring in the carrot root during storage. Can. J. Plant Sci. 53:635-641.

Poovaiah, B.W. 1986. Role of calcium in prolonging storage life of fruits and vegetables. Food Technol. (May 1986):86-89.

Purdy, L.H. 1958. Some factors affecting penetration and infection by Sclerotinia sclerotiorum. Phytopathology 48:605-609.

Purdy, L.H. 1979. Sclerotinia sclerotiorum: history, diseases and symptomatology, host range, geographic distribution, and impact. Phytopathology 69:875-880.

Raghavan, G.S.V., R. Bovell, and M. Chayet. 1980. Storability of fresh carrots in a simulated jacketed storage. Trans. ASAE 23:1521-1524.

Rainbow, A.F. 1970. Sclerotinia disease of dwarf tomatoes. N.Z. J. Agric. 121(6):58-62.

Ryall, A.L., and W.J. Lipton. 1979. Handling, transportation, and storage of fruits and vegetables. Vol. 1. Vegetables and melons. Westport, CT: AVI Pub. Co. 588 pp.

Salunkhe, D.K., and B.B. Desai. 1984. Postharvest Biotechnology of Vegetables. Boca Raton, FL: CRC Press. 313 pp.

Schwartz, H.F., and J.R. Steadman. 1978. Factors affecting sclerotium production, and apothecium production by, Sclerotinia sclerotiorum. Phytopathology 68:383-388.

Schwartz, H.F., D.H. Casciano, J.A. Asenga, and D.R. Wood. 1987. Field measurement of white mold effects upon dry beans with genetic resistance or upright plant architecture. Crop Sci. 27:699-702.

Sedun, F.S., and J.F. Brown. 1987. Infection of sunflower leaves by ascospores of Sclerotinia sclerotiorum. Ann. Appl. Biol. 110:275-285.

Skou, J.P. 1971. Effects of calcium on storage rot in irradiated and non-irradiated carrots. Acta Hort. 20:115-124.

Smith, W.H. 1967. Low temperature and the storage of carrots. J. Food. Technol. 2:89-94.

Steadman, J.R. 1979. Control of plant diseases caused by Sclerotinia species. Phytopathology 69:904-907.

Steadman, J.R., and K.W. Nickerson. 1975. Differential inhibition of sclerotial germination in Whetzelinia sclerotiorum. Mycopathologia 57:165-170.

Tahvonen, R. 1985. The prevention of Botrytis cinerea and Sclerotinia sclerotiorum on carrots during storage by spraying the tops with fungicide before harvesting. Ann. Agric. Fenn. 24:89-95.

Tu, J.C. 1983. Efficacy of iprodione against alternaria black pod and white mold of white beans. Can. J. Plant Pathol. 5:133-135.

Tu, J.C. 1984. Tolerance of white bean (Phaseolus vulgaris) to white mold (Sclerotinia sclerotiorum) associated with tolerance to oxalic acid. Physiol. Plant Pathol. 26:111-117.

Tucker, W.G. 1974. The effect of mechanical harvesting on carrot quality and storage perfromance. Acta Hort. 38:359-372.

Van den Berg, L. 1978. Water vapor pressure. In Postharvest physiology of vegetables. New York: Marcel Dekker, Inc. pp. 203-236.

Van den Berg, L. 1981. The role of humidity, temperature, and atmospheric composition in maintaining vegetable quality during storage. In:Quality of selected fruits and vegetables of North America. ed. R. Teranishi and H. Barrera-Benitez. Washington, DC: American Chemical Society. pp. 95-107.

Van den Berg, L., and C.P. Lentz. 1966. Effect of temperature, relative humidity, and atmospheric composition on changes in quality of carrots during storage. Food. Technol. 20(7):104-107.

Van den Berg, L., and C.P. Lentz. 1968. Effect of relative humidity and temperature on survival and growth of Botrytis cinerea and Sclerotinia sclerotiorum. Can. J. Bot. 46:1477-1481.

Van den Berg, L., and C.P. Lentz. 1973. High humidity storage of carrots, parsnips, rutabagas, and cabbage. J. Amer. Soc. Hort. Sci. 98:129-132.

Van den Berg, L., and C.P. Lentz. 1974. High humidity storage of some vegetables. Can. Inst. Food Sci. Technol. 7:260-262.

Van den Berg, L., and C.P. Lentz. 1978. High humidity storage of vegetables and fruits. HortScience 13:565-569.

Van den Berg, L., and S.M. Yang. 1969. Effect of relative humidity on production of extracellular pectolytic enzymes by Botrytis cinerea and Sclerotinia sclerotiorum. Can. J. Bot. 47:1007-1010.

Watson, E.L. 1960. Prompt handling plus proper cooling for better quality. Can. Food. Ind. 31(6):33-35.

Willetts, H.J., and J.A.-L. Wong. 1980. The biology of Sclerotinia sclerotiorum, S. trifoliorum, and S. minor with emphasis on specific nomenclature. Bot. Rev. 46:101-165.

Williams, G.H., and J.H. Western. 1965. The biology of Sclerotinia trifoliorum Erikss. and other species of sclerotium-forming fungi. I. Apthecium formation from sclerotia. Ann. Appl. Biol. 56:253-260.

Williams, J.R., and D. Stelfox. 1980. Influence of farming practices in Alberta on germination and apothecium production of sclerotia of Sclerotinia sclerotiorum. Can. J. Plant Pathol. 2:169-172.

Yarden, O., Y. Ben-Yephet, J. Katan, and N. Aharonson. 1986. Fungicidal control of Sclerotinia sclerotiorum in soil with a combination of benomyl and thiram. Plant Dis. 70:738-742.

Zimmer, D.E., and J.A. Hoes. 1978. Diseases. In Sunflower Science and Technology. ed. J.F. Carter. Madison, WI: American Society of Agronomy.

Appendix A

Appendix A. Temperature monitoring of a carrot storage, 1985 average daily temperatures (°C)[1], steel shed, Jamor Farms, Portage la Prairie, MB.

Date	Air Plenum	Carrot Pile	Pile Surface	Outside Air
Sept 19	6.7	5.8	6.4	7.5
20	6.1	6.2	5.1	4.6
21	6.0	6.8	5.4	9.1
22	5.8	6.2	5.7	9.6
23	5.0	5.6	5.7	4.8
24	4.8	5.1	4.6	3.5
25	5.7	4.9	4.6	3.6
26	5.4	4.9	4.2	4.4
27	5.1	5.0	4.7	5.7
28	4.3	5.0	4.8	4.4
29	4.4	4.4	4.2	4.0
30	4.2	4.7	4.2	3.5
Oct 1	3.9	4.8	4.0	4.4
2	4.4	4.3	4.0	5.9
3	4.1	5.0	4.3	8.6
4	4.7	4.8	3.9	6.1
5	4.4	4.3	4.2	5.3
6	3.9	3.8	3.5	4.2
7	3.0	3.4	3.9	2.1
8	2.8	2.7	3.2	-1.4
9	2.3	3.3	3.5	-3.9
10	1.9	3.7	3.0	-0.8
11	1.6	3.1	2.7	4.1
12	1.7	2.4	1.8	7.3
13	1.9	2.4	1.7	8.0
14	2.3	2.8	2.4	4.5
15	1.7	2.8	2.4	0.6
16	1.0	2.4	2.0	-0.9
17	2.2	2.2	1.8	3.9
18	2.8	2.2	1.8	8.1
19	2.6	2.4	2.1	9.6
20	2.9	2.7	2.4	12.2
21	2.5	3.0	2.9	10.3
22	6.2	3.4	4.2	11.4
23	4.4	4.4	4.5	9.5
24	3.4	4.5	4.3	8.1
25	3.4	4.1	3.8	7.3
26	3.5	4.1	3.8	7.3
27	3.0	4.3	4.0	3.2
28	3.1	3.9	3.7	7.9

Date		Air Plenum	Carrot Pile	Pile Surface	Outside Air
Oct	29	3.1	3.6	3.2	4.6
	30	2.4	3.6	3.2	5.7
	31	2.1	3.5	3.1	5.8
Nov	1	2.8	3.1	2.7	3.5
	2	1.6	3.1	2.8	1.8
	3	1.4	2.9	2.4	2.4
	4	1.8	2.5	2.0	3.2
	5	1.5	2.4	1.9	5.0
	6	0.7	2.1	1.5	-1.4
	7	0.2	1.5	1.0	-4.7
	8	0.6	1.2	0.6	-8.4
	9	0.8	1.2	0.6	-9.5
	10	0.6	1.3	0.9	-12.7
	11	1.0	1.4	1.1	-10.4
	12	1.4	1.2	1.3	-8.8
	13	0.7	1.9	1.6	-11.1
	14	0.6	1.3	1.7	-7.1
	15	0.6	1.3	1.5	-3.7
	16	0.7	1.2	1.4	-1.9
	17	0.3	1.0	1.4	-7.1
	18	0.3	0.9	1.2	-10.4
	19	0.2	0.7	1.1	-10.3
	20	0.4	0.8	1.1	-8.8
	21	0.5	0.8	1.1	-16.0
	22	1.0	1.0	1.4	-13.1
	23	0.5	0.9	1.5	-20.3
	24	0.3	1.0	1.4	-23.1
	25	0.2	0.9	1.2	-22.9
	26	0.4	0.8	1.1	-19.0
	27	-0.6	0.5	1.3	-26.2
	28	0.9	1.0	1.4	-22.7
	29	0.3	0.8	1.6	-28.9
	30	0.5	0.9	1.7	-28.7
Dec	1	1.8	1.3	1.8	-26.2
	2	2.1	1.8	2.0	-22.4
	3	1.7	1.9	2.4	-22.3
	4	2.6	2.3	2.6	-13.2
	5	2.0	2.4	2.6	-19.0
	6	1.9	2.5	2.7	-10.7
	7	1.9	2.5	2.3	-11.0
	8	1.7	2.4	2.2	-8.7
	9	1.8	2.3	2.2	-6.4
	10	0.9	2.3	2.2	-14.2
	11	0.9	2.1	2.1	-13.3
	12	1.0	2.3	2.3	-21.1
	13	1.3	2.4	2.2	-23.0
	14	1.8	2.2	2.0	-18.3
	15	0.5	2.1	1.9	-21.6
	16	1.0	2.1	2.0	-24.0
	17	0.6	1.9	1.8	-22.8
	18	0.9	1.7	1.7	-21.4
	19	0.8	1.3	1.0	-19.2

Date		Air Plenum	Carrot Pile	Pile Surface	Outside Air
Dec	20	0.8	1.3	0.9	-19.8
	21	1.9	1.6	1.3	-4.7
	22	0.8	1.9	1.2	-2.4
	23	0.6	1.7	0.9	-16.0
	24	0.6	1.4	0.6	-23.3
	25	0.5	1.4	0.7	-19.4
	26	0.9	1.4	0.9	-9.5
	27	0.1	1.2	0.4	-18.1
	28	0.6	1.3	0.4	-10.8
	29	0.5	1.3	0.5	-12.8
	30	0.5	1.5	0.7	-9.4
	31	1.0	1.5	0.9	-18.3

[1] Average daily temperatures are average of 1 a.m., 7 a.m., 1 p.m., and 7 p.m. readings.

Appendix B

Appendix B. Temperature monitoring of a carrot storage, 1985 average daily temperatures (°C)[1], wooden shed, Jamor Farms, Portage la Prairie, MB.

Date		Air Plenum	Carrot Pile	Pile Surface	Outside Air
Oct	6	3.5	6.3	6.7	4.2
	7	2.9	5.8	6.0	2.1
	8	2.4	5.0	4.8	-1.4
	9	3.2	4.8	3.1	-3.9
	10	2.0	4.2	1.4	-0.8
	11	2.8	2.5	2.3	4.1
	12	1.1	4.1	2.2	7.3
	13	0.9	3.0	2.0	8.0
	14	0.8	2.5	1.8	4.5
	15	1.2	2.8	2.2	0.6
	16	0.8	2.8	1.7	-0.9
	17	0.9	2.8	2.1	3.9
	18	1.4	4.3	2.6	8.1
	19	1.8	3.7	3.0	9.6
	20	2.4	4.9	4.1	12.2
	21	2.8	4.9	3.8	10.3
	22	3.3	4.6	4.3	11.4
	23	2.9	4.4	4.0	9.5
	24	2.3	3.8	2.4	8.1
	25	2.8	3.9	3.6	7.3
	26	3.2	3.9	3.5	7.3
	27	2.4	3.4	3.6	3.2
	28	2.6	3.8	3.7	7.9
	29	2.4	3.6	4.1	4.6
	30	1.2	2.9	1.8	5.7
	31	2.9	4.0	3.3	5.8
Nov	1	1.9	3.8	2.2	3.5
	2	1.0	3.5	1.3	1.8
	3	1.8	3.7	2.8	2.4
	4	0.9	3.2	1.7	3.2
	5	1.9	3.5	2.5	5.0
	6	2.3	3.7	2.5	-1.4
	7	2.5	3.8	3.0	-4.7
	8	0.7	3.0	1.3	-8.4
	9	1.3	3.2	2.2	-9.5
	10	1.2	2.8	1.7	-12.7
	11	1.4	1.9	2.5	-10.4
	12	1.1	1.6	2.6	-8.8
	13	1.4	1.8	2.4	-11.1
	14	1.3	2.9	1.6	-7.1

Date	Air Plenum	Carrot Pile	Pile Surface	Outside Air
Nov 15	1.5	2.6	2.2	-3.7
16	0.8	2.1	1.0	-1.9
17	1.2	2.3	1.6	-7.1
18	1.3	2.4	1.3	-10.4
19	1.3	2.5	2.2	-10.3
20	1.5	2.5	2.6	-9.9
21	1.3	2.3	1.9	-16.0
22	1.7	2.2	1.7	-13.1
23	1.1	2.3	1.5	-20.3
24	1.2	2.0	1.9	-23.1
25	1.1	1.8	1.5	-22.9
26	0.9	1.7	1.1	-19.0
27	1.6	1.9	2.1	-26.2
28	1.4	1.8	1.5	-22.7
29	0.9	1.6	1.4	-28.9
30	1.1	1.4	1.6	-28.7
Dec 1	1.0	1.4	1.3	-26.2
2	1.6	1.4	1.8	-22.4
3	1.2	1.5	1.3	-22.3
4	1.2	1.4	1.3	-13.2
5	1.3	1.3	1.2	-19.0
6	1.1	1.5	1.5	-10.7
7	1.3	1.4	1.3	-11.0
8	1.3	1.2	1.4	-8.7
9	1.3	1.2	1.2	-6.4
10	1.4	1.2	1.2	-14.2
11	1.7	1.4	1.5	-13.3
12	1.0	1.2	1.4	-21.1
13	0.7	1.5	0.9	-23.0
14	1.1	1.4	1.1	-18.3
15	1.4	1.2	1.2	-21.6
16	1.0	1.1	1.0	-24.0
17	1.1	1.1	1.1	-22.8
18	0.8	1.1	0.9	-21.4
19	1.6	1.2	1.3	-19.2
20	1.5	1.0	1.4	-19.8
21	1.4	0.9	1.3	-4.7
22	1.4	1.0	1.3	-2.4
23	1.4	0.9	1.2	-16.0
24	1.0	1.0	1.2	-23.3
25	0.6	0.9	0.8	-19.4
26	0.5	0.7	0.5	-9.5
27	0.6	0.3	0.6	-18.1
28	1.0	0.4	0.8	-10.8
29	0.8	0.4	0.8	-12.8
30	0.6	0.6	0.9	-9.4
31	0.9	0.8	1.0	-18.3

[1] Average daily temperatures are average of 1 a.m., 7 a.m., 1 p.m., and 7 p.m. readings.

Appendix C

Appendix C. Temperature monitoring of a carrot storage, 1985 average daily temperatures (°C)[1], Connery's Riverdale Farms, Portage la Prairie, MB.

Date		Air Plenum	Carrot Pile	Pile Surface	Outside Air
Sept	23	6.5	4.6	5.8	4.8
	24	5.5	3.9	4.1	3.5
	25	3.9	2.8	3.9	3.6
	26	4.2	3.2	4.3	4.4
	27	3.6	3.3	4.1	5.7
	28	3.8	2.1	3.6	4.4
	29	2.9	2.0	2.0	4.0
	30	3.5	2.4	3.1	3.5
Oct	1	4.0	2.9	4.0	4.4
	2	3.7	2.4	3.3	5.9
	3	3.6	2.2	2.4	8.6
	4	4.6	2.6	3.2	6.1
	5	3.2	2.7	3.1	5.3
	6	2.4	1.9	1.6	4.2
	7	2.0	1.6	1.3	2.1
	8	2.0	1.4	1.7	-1.4
	9	1.5	1.1	1.3	-3.9
	10	2.0	1.5	1.6	-0.8
	11	2.7	2.1	2.4	4.1
	12	2.4	2.0	1.7	7.3
	13	2.6	2.0	2.1	8.0
	14	2.4	1.9	2.0	4.5
	15	2.3	1.8	2.0	0.6
	16	2.5	1.7	1.7	-0.9
	17	2.5	1.8	1.8	3.9
	18	2.3	1.7	1.5	8.1
	19	2.3	1.7	1.9	9.6
	20	3.0	2.0	2.5	12.2
	21	2.5	2.0	1.9	10.3
	22	2.4	2.2	2.1	11.4
	23	2.6	2.2	2.0	9.5
	24	2.6	2.0	1.8	8.1
	25	2.3	1.9	1.6	7.3
	26	2.2	1.8	1.5	7.3
	27	2.2	1.6	1.3	3.2
	28	1.9	1.5	1.3	7.9
	29	1.7	1.3	1.2	4.6
	30	1.7	1.3	1.1	5.7
	31	1.7	1.5	1.3	5.8

Date		Air Plenum	Carrot Pile	Pile Surface	Outside Air
Nov	1	2.0	1.7	1.2	3.5
	2	1.8	1.5	1.1	1.8
	3	2.0	1.6	1.2	2.4
	4	2.1	1.7	1.2	3.2
	5	2.1	1.6	1.2	5.0
	6	2.0	1.6	1.3	-1.4
	7	2.0	1.6	1.1	-4.7
	8	2.0	1.6	1.1	-8.4
	9	1.8	1.4	1.4	-9.5
	10	1.6	1.3	1.1	-12.7
	11	1.4	1.3	0.8	-10.4
	12	1.6	1.2	1.4	-8.8
	13	1.8	1.4	1.4	-11.1
	14	1.5	1.4	1.5	-7.1
	15	1.6	1.2	0.8	-3.7
	16	1.5	1.9	0.7	-1.9
	17	1.1	0.9	0.8	-7.1
	18	1.1	0.8	0.7	-10.4
	19	1.0	0.8	0.9	-10.3
	20	1.1	0.8	0.6	-9.9
	21	1.1	0.8	0.6	-16.0
	22	1.1	0.9	0.6	-13.1
	23	1.1	0.9	0.5	-20.3
	24	1.1	0.9	0.6	-23.1
	25	1.0	0.7	0.3	-22.9
	26	1.0	0.7	0.2	-19.0
	27	1.0	0.7	0.3	-26.2
	28	0.9	0.7	0.4	-22.7
	29	1.1	0.9	0.5	-28.9
	30	1.1	0.8	0.6	-28.7
Dec	1	0.8	0.6	0.3	-26.2
	2	0.7	0.5	0.3	-22.4
	3	1.0	0.6	0.3	-22.3
	4	1.1	0.8	0.7	-13.2
	5	1.1	0.7	0.3	-19.0
	6	1.2	0.7	0.4	-10.7
	7	1.3	1.0	0.6	-11.0
	8	1.1	0.8	0.4	-8.7
	9	0.9	0.6	0.3	-6.4
	10	0.9	0.4	0.4	-14.2
	11	0.9	0.6	0.3	-13.3
	12	0.9	0.6	0.3	-21.1
	13	0.9	0.5	0.4	-23.0
	14	0.9	0.6	0.3	-18.3
	15	0.7	0.4	0.1	-21.6
	16	1.0	0.5	0.4	-24.0
	17	0.9	0.5	0.2	-22.8
	18	1.0	0.5	0.3	-21.4
	19	1.2	0.7	0.4	-19.2

Date		Air Plenum	Carrot Pile	Pile Surface	Outside Air
Dec	20	1.0	0.6	0.4	-19.8
	21	1.1	0.7	0.4	-4.7
	22	0.9	0.6	0.2	-2.4
	23	0.8	0.5	0.1	-16.0

[1] Average daily temperatures are average of 1 a.m., 7 a.m., 1 p.m., and 7 p.m. readings.