20

Fungi

LISE KORSTEN and FRITZ C. WEHNER

University of Pretoria, Pretoria, South Africa

I. INTRODUCTION

Traditionally fungi were accepted as a single kingdom along with bacteria (Monera), plants (Plantae), animals (Animalia), and protists (Protista) according to the five-kingdom scheme of Whittaker (1969). However, with advances in ultrastructural, biochemical, and particularly molecular biology, treatment of fungi as one of the five kingdoms of life has become increasingly untenable. The organisms studied by mycologists are now established as polyphyletic and are referred to in 11 phyla and one form-phylum in three kingdoms of Eukaryota—namely Protozoa, Chromista, and Fungi. Members of the Protozoa are predominantly unicellular, plasmodial, or colonial phagotrophic, wall-less in the trophic state, with tubular mitochondrial cristae and nontubular flagellar mastigonemes. The Chromista are unicellular, filamentous or colonial primarily phototrophic organisms, often with cellulosic cell walls and with tubular mitochondrial cristae and flagellar mastigonemes. True fungi feed by absorption, contain chitin and β-glucans in their cell walls, have mitochondria with flattened cristae and are mostly non-flagellate; if flagellate, flagellar mastigonemes are absent (Hawksworth et al., 1995).

Of the more than 70,000 fungal species that have been described, only a relatively small number can be regarded as primary postharvest pathogens of vegetables. Most of these belong to the Ascomycota or Deuteromycota, with a few species in the Basidiomycota, Zygomycota (all true fungi) or Oomycota (kingdom Chromista). These phyla are characterized by the following:

Ascomycota: Meiospores (ascospores) produced endogenously in asci, which are either naked or contained within an ascoma.

Basidiomycota: Meiospores (basidiospores) produced exogenously on basidia of various shapes.

Deuteromycota: Sexual reproduction absent or unknown. Mitospores (conidia) absent or formed on conidiophores existing singly, clustered in specialized structures such as sporodochia or synnemata, or produced in a conidioma. Represent the anamorphs (asexual stages) of many ascomycetes and basidiomycetes.

Zygomycota: Meiospores (zygospores) produced by gametangiomy. Mitospores (sporangiospores) produced in sporangia.

Oomycota: Meiospores (oospores) produced by gametangy. Motile mitospores (zoospores) biflagellate, at least one of the flagella having mastigonemes (Agrios, 1997; Alexopoulos et al., 1996; Hawksworth et al., 1995).

From the above it appears there are no primary postharvest pathogens in the Protozoa and Chytridiomycota, the latter being the only phylum of Fungi having a flagellate phase. Two pathogens that could represent these taxa are *Spongospora subterranea* f. sp. *subterranea*, the cause of powdery scab of potato, and *Synchytrium endobioticum*, causal agent of wart disease of potato, respectively. Both these organisms infect tubers before harvesting and are known to occasionally increase during storage (Hooker, 1983).

One or more fungi can attack vegetables before, during, or after harvesting. Although limited in number, the spectrum of fungal species that can affect postharvest quality of vegetables is diverse (Table 1). Most of the pathogens are specific to a particular vegetable or to a few vegetable species, whereas others have a more extensive host range, e.g., *Botrytis cinerea*, *Fusarium oxysporum*, *Lasiodiplodia theobromae*, *Macrophomina phaseolina*, *Pythium ultimum*, *Rhizopus stolonifer*, *Sclerotinia sclerotiorum*, and *Sclerotium rolfsii*. Similarly, certain species are restricted to an infection of vegetables after harvest, whereas many others can also infect growing crops. *B. cinerea*, for instance, causes blossom blights, damping-off, stem cankers or rots, leaf spots, and tuber, corm, bulb and root rots in addition to the storage rots listed in Table 1 (Agrios, 1997). Sometimes several species of a genus can attack a single crop, e.g. Fusarium dry rot of potato caused mainly by *Fusarium solani*, but frequently also by *F. avenaceum*, *F. equiseti*, *F. oxysporum*, *F. sambucinum*, *F. sporotrichioides*, and various other *Fusarium* spp. (common names for plant diseases, APSnet, 1996; Snowdon, 1992). Also, infection by one pathogen can predispose hosts to infection by another pathogen causing a different disease—e.g. powdery scab of potato caused by *S. subterranea* f. sp. *subterranea* can promote infection of potato tubers by the late blight fungus *Phytophthora infestans* and other organisms (Snowdon, 1992).

II. SURVIVAL AND DISSEMINATION OF INOCULUM

Since the edible component of most vegetable crops is produced in soil (bulbs, roots and tubers) or in close proximity to it (flower, leaf, stem and fruit vegetables), soil constitutes an important source of initial inoculum. The majority of postharvest pathogens are therefore adapted to survival in soil—e.g., sclerotia of *Sclerotinia* and *Sclerotium* spp., microsclerotia of *Polyscytalum pustulans*, chlamydospores of *Fusarium* spp., phaeodictyospores of *Alternaria solani*, thick-walled oospores of *Phytophthora* and *Pythium* spp., zygospores of Mucorales, cystosori of *S. subterranea* f.sp. *subterranea*, etc. (Domsch et al., 1980; Harris et al., 1997; Hooker, 1983). Other sources of survival include trellis stakes (*Phoma* spp.), storage crates (*L. theobromae*, *Rhizoctonia carotae*, *Rhizopus* spp.), crop residues (*Alternaria alternata*), weeds (*Colletotrichum capsici*, *Phomopsis phaseoli*), propagating

material (*Helminthosporium solani*, *Phytophthora capsici*), and even footwear (*Phoma lingam*) (Besri, 1983, Clarke and Moyer, 1988; Jensen, 1971; Snowdon, 1992).

Dissemination of fungal spores essentially depends on their nature—e.g., motile spores are mostly dispersed by free water, dry nonmotile spores by air currents, and wet nonmotile spores by rain or irrigation splashing. However, the various types of spores can also be dispersed by other mechanisms. Contaminated tools and equipment are effective means of transmitting pathogens—e.g., *P. lingam* and *Phytophthora porrii* (Geeson, 1976; Snowdon, 1992). Insects carry many pathogens; for example, *Macrophomina phaseolina* is carried by tuber moth (Paharia, 1960), *Rhizopus oryzae* and *R. stolonifer* by fruit flies (Butler and Bracker, 1963; Jones et al., 1991), *L. theobromae* by cockroaches and sweet potato weevils (Clarke and Moyer, 1988), and *Geotrichum candidum* by flies and fruit flies (Butler, 1961). Resistant structures produced by some fungi can survive passage through the alimentary canal of farm animals and are so disseminated to other areas, e.g., *S. subterranea* f. sp. *subterranea* (Morse, 1914) and certain *Pythium* spp., according to Dowson (1931) (Snowdon, 1992).

III. FUNGAL ATTACK MECHANISMS

The evolution of fungal plant pathogens toward a high degree of specialization for individual plant species is reflected in the different ways that plants are attacked (Jackson and Taylor, 1996). Certain pathogens are unable to penetrate intact plant surfaces and, consequently, are opportunistic parasites that infect only through wounds or natural openings (lenticels, stomata, buds, growth cracks, etc.). Alternatively, opportunists infect plants weakened by physical or physiological stresses such as chilling, frost damage, sunscald, senescence, etc. Certain pathogens rely on an initial recognition of a living host to induce the production of infection structures that allow penetration and colonization of the host. Important mechanisms involved in direct or wound penetration involve the transition from saprophytism to parasitism and the degree of virulence once pathogenicity has been established (see Chap. 25).

Certain, postharvest vegetable pathogens penetrate intact surfaces of the host directly, whereas others infect through natural openings or wounds. Infections leading to postharvest decay can be separated into three categories, viz. incipient, latent (or quiescent), and wound infections.

A. Incipient Infections

The concept of incipience refers to an initial or early infection stage beginning to develop. This may occur at any time before or after harvest (Bruton, 1994). Incipient infections often pass unnoticed through the culling process during harvesting, packaging, or retail display. They may remain active and cause decay, although further development can in most instances be arrested by postharvest fungicide applications or refrigeration. An active decay sometimes appears during transport and storage, which can lead to "nests" of decaying produce. The storage environment as well as the duration of storage frequently determine the development of such nests. Nests are much more likely to develop as the vegetable nears the end of its postharvest life.

B. Latent or Quiescent Infections

Quiescent infections result when the pathogen's development becomes arrested. Quiescence can be initiated at virtually any stage of infection, penetration to colonization

Table 1 Fungal Pathogens of Vegetables That Cause Postharvest Losses

Pathogen	Disease	Crop[a]	Primary mode of infection
Acrothecium carotae Arsvoll	*Acrothecium* rot	Car	?
Alternaria spp.	*Alternaria* storage rot	Swe	Wound
Alternaria alternata (Fr.:Fr.) Keissler	*Alternaria* rot (Cau), black pod (Bea, Cucs), black shoulder (Tom), tuber rot (Pot)	Bea, Cau, Cucs, Pot, Tom	Preharvest, incipient
Alternaria brassicae (Berk.) Sacc.	*Alternaria* rot	Bras, Sws, Tur	?
Alternaria brassicicola (Schwein.) Wiltshire	*Alternaria* rot	Cab, Cau	?
Alternaria dauci (Kühn) Groves and Skolko	Black rot	Car, Par	Incipient, wound
Alternaria porri (Ellis) Cif.	Purple blotch	Gar, Oni	Preharvest, wound
Alternaria radicina Meier, Drechsler and Eddy	Black rot	Car, Cel, Par	Incipient, wound
Alternaria raphani Groves and Skolko	*Alternaria* rot	Bro, Cab	?
Alternaria solani Sorauer	*Alternaria* rot/tuber rot	Pot	Wound
Ascochyta boltshauseri Sacc.	Speckle disease	Bea	Preharvest
Ascochyta fabae Speg.	*Ascochyta* pod spot	Bea	Incipient
Ascochyta pisi Lib.	*Ascochyta* pod spot	Pea	Incipient
Aspergillus alliaceus Thom and Church	Yellow mold rot	Oni	?
Aspergillus flavus Link:Fr.	*Aspergillus* rot	Cas, Pep, Tom	Wound
Aspergillus niger v. Tieghem	*Aspergillus* black mold rot (Car), *Aspergillus* rot, black mold rot (Oni)	Car, Cas, Oni, Pep, Tom, Yam	Wound
Botrytis aclada Fresen.	Neck rot	Oni	?
Botrytis byssoidea J.C Walker	Neck rot	Oni	?
Botrytis cinerea Pers.:Fr.	Brown strain (Oni), gray mold, gray mold rot, storage rot (Bee)	Aub, Bee, Bras, Car, Cel, Cru, Cucs, End, Leg, Let, Oni, Pep, Pars, Pot, Rhu, Sol, Swe, Yam	Incipient, latent, wound
Botrytis fabae Sardina	Chocolate spot, gray mold rot	Legs	Incipient

Botrytis porri Buchw.	*Botrytis* rot, gray mold rot	Gar, Lee	Wound
Botrytis squamosa J.C. Walker	Neck rot	Gar, Oni	Incipient
Ceratocystis fimbriata Ellis and Halst.	Black rot	Swe	Incipient, wound
Choanephora cucurbitarum (Berk and Ravenel) Thaxt.	*Choanephora* fruit rot, *Choanephora* rot	Bee, Cau, Cucs	Wound
Cladosporium cladosporioides (Fresen.) de Fries	*Cladosporium* rot	Aub	Wound
Cladosporium cucumerinum Ellis and Arth.	*Cladosporium* rot, gummosis	Cucs	Wound
Cladosporium fulvum Cooke	*Cladosporium* rot	Tom	
Cladosporium herbarum (Pers.) Link	*Cladosporium* rot	Pep, Tom	Wound
Cladosporium oxysporum Berk. and Curt.	*Cladosporium* rot	Tom	Wound
Colletotrichum erumpens Sacc.	Anthracnose	Rhu	Latent
Colletotrichum capsici (Sydow) Butler and Bisby	Anthracnose	Pep	Latent
Colletotrichum coccodes (Wallr.) S.J. Hughes	Anthracnose	Sol	Latent
Colletotrichum dematium (Pers.:Fr.) Grove	Anthracnose	Bee, Pep, Spi, Tom	Latent
Colletotrichum gloeosporioides (Penz.) (Penz. and Sacc. in Penz.)	Anthracnose	Pep, Tom	Latent
Colletotrichum lindemuthianum (Sacc. and Magnus) Lams.-Scrib.	Anthracnose	Bea, Cow	Latent, wound
Colletotrichum orbiculare (Berk and Mont.) Arx	Anthracnose	Cucs	Latent
Colletotrichum truncatum (Schw.) Andris and Moore	Anthracnose	Bea	Latent
Cylindrocladium clavatum Hodges and May	*Cylindrocladium* rot	Pot	Wound
Epicoccum nigrum Link	Storage rot	Swe	Wound
Fusarium acuminatum Ellis. and Everh.	*Fusarium* rot	Cucs	Wound
Fusarium avenaceum (Corda ex. Fr.) Sacc.	*Fusarium* dry rot, *Fusarium* rot	Asp, Aub, Car, Cucs, Pot, Tom	Latent, wound

Table 1 Continued

Pathogen	Disease	Crop[a]	Primary mode of infection
Fusarium culmorum (W.G. Smith) Sacc.	*Fusarium* rot	Asp, Gar	Incipient
Fusarium equiseti (Corda) Sacc.	*Fusarium* dry rot, *Fusarium* fruit rot	Aub, Cucs, Pot, Tom	Latent, wound
Fusarium graminearum Schwabe	*Fusarium* rot	Cucs	Wound
Fusarium moniliforme Sheldon	*Fusarium* rot	Asp, Aub, Coc, Pep, Yam	Wound
Fusarium oxysporum Schlecht. emend. Snyd. and Hans.	Basal rot (Buls), *Fusarium* crown rot, *Fusarium* dry rot, *Fusarium* rot (Yam), surface rot (Swe)	Art, Asp, Bulb, Cab, Cocs, Cucs, Pep, Pot, Sol, Swe, Yam	Incipient, wound
Fusarium sambucinum Fuckel	*Fusarium* dry rot	Pot	Latent, wound
Fusarium scirpi Lambotte and Fautr.	*Fusarium* rot	Cucs	Wound
Fusarium semitectum Berk. and Ravenel	*Fusarium* fruit rot	Cucs	?
Fusarium solani (Mart.) Appel and Wollenw. emend. Snyd. & Hans.	*Fusarium* dry rot (Pot), *Fusarium* fruit rot, *Fusarium* rot	Cas, Cocs, Cucs, Pot, Squ, Swe, Yam	Wound
Fusarium sporotrichioides Sherb.	*Fusarium* dry rot	Pot	Latent, wound
Geotrichum candidum Link ex Leman	Rubbery rot (Pot), sour rot	Car, Oni, Pep, Pot, Tom	Wound
Helminthosporium solani Durieu and Mont.	Silver scurf	Pot	?
Lasiodiplodia theobromae (Pat.) Griffon and Maubl.	*Botryodiplodia* rot, Java black rot (Swe), *Lasiodiplodia* fruit rot (Cucs)	Cas, Coc, Cucs, Oni, Swe, Tom, Yam	Incipient, wound
Macrophomina phaseolina (Tassi) Goidanich	Charcoal rot, *Macrophomina* rot	Bee, Car, Cucs, Gar, Oni, Pep, Pot, Swe, Tom	Preharvest, wound
Microdochium panattonianum (Berl.) Sutton	Anthracnose	Bras, Leaf	Incipient, latent
Monilochaetes infuscans Halst. ex Harter	Scurf	Swe	Preharvest
Mucor mucedo Mich. ex St.-Am.	*Mucor* rot	Tom	?
Mucor piriformis Fischer	*Mucor* rot	Tom	?
Mucor racemosus Fresen.	Storage rot	Swe	?
Mycocentrospora acerina (Hartig) Deighton	Licorice rot	Car, Cel, Par	Incipient, wound

Mycosphaerella brassicicola (Duby) Lindau in Engl. and Prantl	Ring spot	Bras	Preharvest, incipient
Myrothecium roridum Tode:Fr.	Crater rot	Cucs, Sols	?
Penicillium spp.	Blue mold rot, storage rot (Bee)	Bee, Swe	Wound
Penicillium aurantiogriseum Dierckx	Blue/green mold rot	Gar, Oni, Tom, Yam	Wound
Penicillium citrinum Thom	Blue mold	Gar, Oni	Wound
Penicillium corymbiferum Westling	Blue mold	Gar, Oni	Wound
Penicillium crustosum Thom	Blue/green mold rot	Swe, Yam	Wound
Penicillium digitatum (Pers.:Fr) Sacc	Blue mold rot	Cucs, Gar, Oni	Wound
Penicillium expansum Link	Blue mold rot	Car, Gar, Oni, Tom	Wound
Penicillium funiculosum Thom	Blue mold	Gar, Oni	Wound
Penicillium gladioli McCulloch and Thom	Blue/green mold rot	Cucs, Yam	Wound
Penicillium hirsutum Dierkx	Blue mold	Gar, Oni	Wound
Penicillium italicum Wehmer	Blue mold rot	Cucs, Tom	Wound
Penicillium oxalicum Curie and Thom	Blue/green mold rot	Asp, Gar, Oni, Yam	Wound
Penicillium sclerotigenum Yaman	Blue/green mold rot	Yam	Wound
Peronospora parasitica (Pers.:Fr.) Fr.	Downy mildew	Bras	Incipient
Phoma apiicola Kleb.	*Phoma* crown rot	Cel	?
Phoma betae A.B. Frank	*Phoma* rot	Bee	Incipient, wound
Phoma complanata (Tode ex. Fr.) Desm.	*Phoma* rot	Car	?
Phoma cucurbitacearum (Fr.:Fr.) Sacc	*Didymella* black rot	Cucs	Wound
Phoma destructiva Plowr.	*Phoma* rot	Sols	Incipient, wound
Phoma exigua Desmaz.	*Phoma* rot	Aub, Pot, Tom	?
Phoma exigua Desmaz. var. *exigua*	Gangrene, *Phoma* rot	Bee	Incipient, latent, wound
Phoma exigua Desmaz. var. *foveata* (Foister) Boerema	Gangrene	Pot	Incipient, latent, wound
Phoma lingam (Tode: Fr.) Desmaz.	*Phoma* rot	Bras, Sws, Tur	?
Phoma lycopersici Cooke	*Phoma* rot	Aub, Tom	Preharvest, incipient
Phomopsis capsici Punith	*Phomopsis* rot	Pep	Incipient
Phomopsis cucurbitae McKeen	*Phomopsis* black rot	Cucs	Preharvest

Table 1 Continued

Pathogen	Disease	Crop[a]	Primary mode of infection
Phomopsis phaseoli (Desm.) Grove	Dry rot (Swe), *Phomopsis* pod blight (Bea), *Phomopsis* rot	Bea, Pep, Swe, Tom	Incipient
Phomopsis vescans (Sacc. and Sydow) Harter	*Phomopsis* rot	Aub	Incipient
Phytophthora cactorum (Lebert and Cohn) Schröter	*Phytophthora* rot, Rubbery brown rot (Car)	Asp, Car, Rhu	?
Phytophthora capsici Leonian	Buckeye fruit rot (Sols), *Phytophthora* rot	Cucs, Sols	Preharvest
Phytophthora cryptogea Pethybr. and Lafferty	*Phytophthora* rot (Asp) Pink rot (Pot)	Asp, Cas, Pot	Incipient
Phytophthora drechsleri Tucker	Buckeye fruit rot (Tom), *Phytophthora* wet rot (Bee), pink rot (Pot)	Bee, Cas, Pot, Tom	Incipient
Phytophthora erythroseptica Pethybr.	Pink rot	Cas, Pot	Incipient
Phytophthora infestans (Mont.) de Bary	Late blight	Pot, Tom	Incipient, wound
Phytophthora megasperma Drechsler	*Phytophthora* rot (Asp), rubbery brown rot	Asp, Car, Pot	?
Phytophthora nicotianae Breda de Haan	Buckeye fruit rot, *Phytophthora* rot	Bea, Rhu, Sols	?
Phytophthora palmivora (E. Butler) E. Butler	*Phytophthora* rot	Tom	?
Phytophthora porri Foister	Bulb rot (Buls), rubbery brown rot	Bras, Buls, Cab, Car	?
Phytophthora richardiae Buisman	*Phytophthora* rot	Asp	?
Polyscytalum pustulans (N.M. Owens and Wakef.) M.B. Ellis	Skin spot	Pot	Wound
Pseudocercosporella capsellae (Ellis and Everh.) Deighton	*Cercosporella* spot	Bras	?
Pythium aphanidermatum (Edson) Fitzp.	*Pythium* fruit rot (Aub, Tom), *Pythium* root rot (Bee), *Pythium* rot	Aub, Bea, Bee, Cucs, Swe, Tom	Wound

Pythium arrhenomanes Drechsler	*Pythium* fruit rot (Pot, Tom), watery wound rot (Bea, Cucs)	Bea, Cucs, Pot, Tom	Wound
Pythium debaryanum Auct. non R. Hesse	Leak (Pot), *Pythium* blight (Bea), *Pythium* fruit rot, Watery wound rot (Sols)	Bea, Car, Pot, Sols	Wound
Pythium myriotylum Drechsler	*Pythium* cottony leak, *Pythium* fruit rot	Aub, Cocs, Cucs, Tom	?
Pythium scleroteichum Drechsler	*Pythium* rot	Swe	Preharvest
Pythium spinosum Sawada	*Pythium* rot	Yam	Wound
Pythium splendens Braun	*Pythium* rot	Coc, Rhu, Swe	Preharvest
Pythium sylvaticum Campbell and Hendrix	*Pythium* rot	Yam	Wound
Pythium ultimum Trow	Beetlet rot, Leak (Pot), Pod rot (Bea), *Pythium* rot, *Pythium* fruit rot (Tom), rootlet rot (Swe), rubbery slate rot (Car)	Bee, Car, Cocs, Pot, Pum, Swe, Tom, Yam	Preharvest, wound
Rhizoctonia carotae Rador	Crater rot	Car	Incipient
Rhizoctonia crocorum DC. ex Fr.	Violet root rot	Car, Par	Preharvest
Rhizoctonia solani Kühn	Belly rot (Cucs), black scurf (Pot), bottom rot (Let), brown crown rot (Car), *Rhizoctonia* fruit rot (Tom), *Rhizoctonia* rot (Bee), sprout rot (Swe), Web blight (Bea)	Bea, Bee, Car, Cucs, Let, Pot, Swe, Tom	Preharvest incipient
Rhizopus oryzae Went and Prinsen Geerligs	*Rhizopus* soft rot	Bee, Bras, Car, Cas, Legs, Par, Pot, Swe, Tom, Yam	Wound
Rhizopus stolonifer (Ehrenb.:Fr.) Vuill.	Mushy rot (Oni), *Rhizopus* rot, *Rhizopus* soft rot	Bee, Bras, Car, Cucs, Legs, Oni, Par, Pot, Sols, Swe, Yam	Wound
Rosellinia sp.	*Rosellinia* black rot	Pot	Preharvest
Sclerotinia minor Jagger	*Sclerotinia* rot (Pot), watery soft rot, White mold (Legs, Pot, Sols)	Asp, Bras, Car, Cel, Leaf, Legs, Let, Sols, Par, Pot	Incipient
Sclerotinia sclerotiorum (Lib.) de Bary	*Sclerotinia* crown rot (Bee), *Sclerotinia* pink rot (Cel), *Sclerotinia* rot, storage rot (Swe), watery soft rot, white mold (Bea, Pot, Tom)	Bea, Bee, Bras, Car, Cel, Gar, Legs, Let, Oni, Pot, Sols, Tom, Swe, Sws, Tur	Preharvest, incipient

Table 1 Continued

Pathogen	Disease	Crop[a]	Primary mode of infection
Sclerotium cepivorum Berk.	White rot	Buls, Car, Rhu	Preharvest
Sclerotium rolfsii Sacc.	*Sclerotium* rot (Bee, Cocs, Swe), southern blight (Pot, Sols), white rot	Bee, Buls, Car, Cau, Cucs, Legs, Let, Par, Pot, Rhu, Sols, Swe, Yam	Preharvest, incipient
Spongospora subterranea (Wallr.) Lagerh. f.sp. *subterranea* Tomlinson	Powdery scab	Pot	Preharvest
Stemphylium botryosum Wallr.	Black mold rot	Let, Tom	Incipient
Stemphylium herbarum E. Simmons	Black mold rot	Let, Tom	Incipient
Synchytrium endobioticum (Schilberszky) Percival	Wart	Pot	Preharvest
Trichoderma harzianum Rifai	Trichoderma rot	Cas	Wound
Trichoderma koningii Oudem.	Punky rot	Swe	Wound
Trichothecium roseum (Pers.:Fr) Link	Pink mold rot	Cucs, Pot, Tom	Wound
Ulocladium consortiale (Thüm.) E. Simmons	Black mold rot	Tom	Incipient

[a] Composites:

Bras: Brassicas: Broccoli (*Brassica oleracea* L. var. *italica* Plenck); Brussel sprouts (*Brassica oleracea* L. var. gemmifera DC.); Cabbage (*Brassica oleracea* L. var. *capitata* Alef.); Chinese cabbage (*Brassica campestris* var. *pekinensis* (Laur.) Olsso); Cauliflower (*Brassica oleracea* L. var. *botrytis* Alef.)

Buls: Bulbs: Chives (*Allium schaenoprasum*), Garlic (Allium sativum L.); Leek (*Allium ampeloprasum* L. var. porrum (L.) Gay); Onion (*Allium cepa* L.); Shallot (*Allium cepa* L. var. *aggregatum*)

Cocs: Cocoyams: Taro (*Colocasia esculenta* L.); Tannia (*Xanthosoma sagittifolium* L.)

Cucs: Cucurbits: Cucumber (*Cucumis sativus* L.); Marrow, Pumpkin, Squash (*Cucurbita* spp.); Melon (*Cucumis melo* L.); Watermelon (*Citrullus lonatus* Thunb.)

Leaf: Leafy vegetables: Chicory (*Cichorium intybus* L.); Endive (*Cichorium endivia* L.); Lettuce (*Lactuca sativa* L.)

Legs: Legumes:Bean (*Phaseolus vulgaris* L.); Pea (*Pisum sativum* L.)
Sols: Solanaceous fruit vegetables: Aubergine (*Solanum melongena* L.); Peppers (*Capsicum annuum* L.); Tomato (*Lycopersicon esculentum* Mill.)
Specific crops:
Art: Artichoke (*Cynara scolymus* L.)
Asp: Asparagus (*Asparagus officinalis* L.)
Aub: Aubergine
Bea: Bean
Bee: Beet (*Beta vulgaris* L.)
Cab: Cabbage
Car: Carrot (*Daucus carota* L.)
Cas: Cassava (*Manihot esculenta* Crantz)
Cau: Cauliflower
Cel: Celery (*Apium graveslens* L.)
End: Endive
Gar: Garlic
Lee: Leek
Let: Lettuce
Oni: Onion
Par: Parsnip (*Pastinaca sativa* L.)
Pea: Pea
Pep: Peppers
Pot: Potato (*Solanum tuberosum* L.)
Pum: Pumpkin
Rhu: Rhubarb (*Rheum rhabarbarum* L.)
Spi: Spinach (*Spinacia oleracea* L.)
Squ: Squash
Swe: Sweetpotato (*Ipomoea batatas* (L.) Lam.)
Sws Swede (*Brassica napus* L. var. *napobrassica* . . .)
Tom: Tomato
Tur: Turnip (*Brassica rapa* L.)
Yam: Yam (Dioscorea spp.)
Sources: Compiled from APS common names of plant diseases (APSnet 1996), APS Compendium series (Clark and Moyer, 1988; Hall, 1991; Jones et al., 1991; Schwartz and Mohan, 1995; Zitter et al., 1996), Ellis and Ellis (1985); Snowden (1992).

(Prusky, 1996). After arrival in infection courts, pathogen structures may remain dormant for varying periods of time; however, these periods are regarded as part of a latent or quiescent infection only if a parasitic relationship has developed (Verhoeff, 1974; Swinburne, 1983). Dormant pathogens usually become active when the vegetable is ripening (Verhoeff, 1974). The mechanisms that trigger the resumption in pathogen development are unknown (Bruton, 1994). Simmonds (1963) presented four possible explanations, which were redefined by Verhoeff (1974), Swinburne (1993) and Prusky (1996), and are elaborated on in Chapter 25.

C. Wound and Injury Infections

Many pathogens associated with quiescent infections penetrate host surfaces through wounds or natural infections, whereas others are restricted to wounds for gaining entry. Some of the most devastating postharvest diseases of vegetables originate from mechanical or physiological damage to the surface of the vegetable (Derbyshire, 1973; Friedman, 1960). Random mechanical injuries inevitably occur during harvesting and handling of vegetables, even when these operations are carried out carefully. Indeed, severing the vegetable from the plant can create an infection court for wound pathogens (Day et al., 1972; Geeson, 1976; Semb, 1971). Much more injury is inflicted by mechanical than by hand harvesting (Derbyshire, 1973; Tucker, 1974), whereas excessive pressure created during storage of some crops (e.g., potatoes) damages the lenticels, thereby predisposing them to infection (Dennis, 1987). In addition, the harvest, transport, grading and packing of certain produce can produce physical damage in the form of cuts, abrasions and bruising, which often aggravates disease (Cappellini et al., 1984). *R. stolonifer*, in particular, frequently occurs in solanaceous vegetables damaged by rough handling during and after harvesting (Snowdon, 1992). Rodents, and insects such as cockroaches, flies and weevils can also create wounds on vegetables, thereby providing points of entry for fungi (Clark and Moyer, 1988; Snowdon, 1992).

Physiological injuries induced by environmental stress can predispose vegetables to attack by fungi. Preharvest cold damage or postharvest chilling injury, for instance, can change membrane permeability and the activity of membrane-bound enzymes, leading to an accumulation of toxic intermediates that can damage or kill plant cells (Lyons, 1973). This disruption of the cellular function influences susceptibility to fungal invasion. For example, in unchilled tomatoes, infection by *B. cinerea* usually occurs through the calyx, whereas after chilling, the entire fruit is susceptible (Dennis, 1983; Tomkins, 1963). Infection by *B. cinerea* is also enhanced by chilling injury of peppers, eggplant, and cucumber (Dennis, 1983). Chilling injury has also been associated with infection of tomatoes and peppers by *Alternaria* spp. and cucumbers by *Phoma cucurbitacearum* (Dennis, 1983; Van Steekelenburg, 1982). Tomatoes may be infected by *Phoma lycopersici*, *P. destructiva*, and *P. exigua* through natural cracks at the stem end or through mechanical injuries (Jones et al., 1991; Snowdon, 1992), by *Phomopsis phaseoli* at weak points such as the blossom-end, sunscald lesions or wounds (Luttrell, 1947), and by *Cladosporium herbarum* and *C. oxysporum* through skin weakened by sun scald or chilling injury (Snowdon, 1992).

D. Infection Process

The initial stage of a pathogen's attack is often the adherence of a fungal spore to the plants surface. Adhesion has been related to intermolecular forces developing between

the surfaces of plant and pathogen, although certain spores contain adhesive substances that facilitate attachment (Brown and Howard, 1994). Germ tubes can either form a superficial mycelium preceding penetration or penetrate directly (Verhoeff, 1974). Fungi that penetrate directly produce fine hyphae or a penetration peg arising from an appressorium. Appressoria, which are clearly defined structures formed at the tip of a germ tube, attach to plant surfaces to facilitate penetration and subsequent infection. In certain fungi, penetration occurs only if melanin pigments accumulate in the appressorial wall. Melanin confers rigidity to the appressorium, whereas an accumulation of solutes by this structure leads to water absorption and increased turgor pressure. As this pressure accumulates at the base of the penetration peg, the tip of that peg physically breeches the host surface (Agrios, 1997).

Surface waxes on vegetables may provide signals for certain pathogens to form appressoria and infection pegs. Little is known about the surface waxes of vegetables. However spores of *Colletotrichum gloeosporioides* landing on very hydrophobic surfaces, such as fruit wax produce appressoria (Podilia et al., 1993; Prusky and Plumbley, 1992). Kolattukudy et al. (1995) argued that this type of signaling between plant and pathogen was specific, since other plant waxes would not stimulate production of appressoria by *C. gloeosporioides*. The fatty alcohol fraction, which is 5% of the whole wax, was the most active inducer, with C_{30} and C_{32} as the major components. In tests with authentic compounds, C_{24} and longer chain alcohols were most effective. By contrast, appressoria were not induced by the very-long-chain alcohols found in many plant waxes probably because some of these compounds may inhibit the process (see Chap. 25).

Macerating enzymes, secreted by the pathogen, almost invariably assist penetration of plant barriers by fungi. Many fungi have been shown to produce cutinases, which degrade cutin, the main component of the cuticle. Cutinases are esterases that can be induced in fungi by hydroxylated fatty acid monomers of cutin (Kolattukudy, 1985; Trail and Koller, 1990). Several observations support the involvement of cutinases in the penetration of the host cuticle. For example, enzymatic activity is highest at the point of penetration. The application of cutinase inhibitors to plant surfaces prevents infection. Cutinase-deficient mutants exhibiting reduced virulence became fully virulent when cutinase was applied to the plant surface. Fungi that infect only through wounds and do not produce cutinase can penetrate directly if transformed with a cutinase gene from another fungus (Brown and Howard, 1994; Kolattukudy, 1985; Schäfer, 1994; Trail and Koller, 1990). There are, however, exceptions to the rule. Stahl and Schäfer (1992), for instance, found that cutinase is not required for fungal pathogenicity on pea.

For most fungal pathogens of harvested vegetables, tissue invasion and lesion formation primarily depend on the pathogen's ability to produce pectolytic enzymes that degrade the middle lamellae of plant tissue. The consistency of fresh fruit and vegetables largely depends on pectic substances in the middle lamellae of cell walls that bind the cells together. Pectolysis leads to tissue maceration—i.e., loss of coherence and separation of individual cells. The membranes of separated cells increase in permeability, which leads to cell death and release of host metabolites. The metabolites may be used as a substrate for further development of the pathogen (Dennis, 1987). Pectin-degrading enzymes are involved in many plant diseases, particularly those characterized by macerated tissues. Three fundamentally different types of pectinases exist—namely, pectin methyl esterases that release methyl groups from carboxyls, polygalacturonases that hydrolyze α-1,4-galacturonosyl bonds, and transeliminases or lyases that split the α-1,4-galacturonosyl bonds by rearrangement of hydrogens. Various postharvest fungal pathogens produce both

endo- and exohydrolases, polygalacturonases, as well as lyases (Griffin, 1994). Although no known fungus produces each type of pectinase, several produce more than one kind (Chesson, 1980; Hancock et al., 1964; Lumsden, 1979; Sherwood, 1966; Verhoeff and Warren, 1972). Most of the pectinases are regulated by catabolic repression, although instances of constitutive enzyme formation and induction by galacturonate have been reported. Lyase formation is favored by alkaline pH (Bateman and Basham, 1976). Cell wall–degrading enzymes can be inactivated in host tissue by compounds such as polyphenols, tannins and proteins (see Chap. 25).

Other enzymes involved in plant tissue degradation—e.g., cellulases, hemicellulases, proteases, amylases, phospholipases, etc.—are not discussed here, as they mostly play a secondary (albeit sometimes important) role in postharvest pathology.

IV. EFFECT OF ENVIRONMENTAL FACTORS ON DISEASE DEVELOPMENT

Environmental factors that promote infection of a postharvest pathogen in the field contribute to the severity of decay during storage. For instance, abnormally cool weather during the growing season of tomatoes can result in chilling injury providing a weakened surface that can easily be penetrated by opportunistic parasites such as *Cladosporium* spp., which are incapable of infecting sound fruit (Snowdon, 1992). If prevailing conditions are overcast, cool and damp, tomato plants do not thrive and fruit can more readily be infected by *Trichothecium roseum*, the cause of pink mold rot in storage (Welch et al., 1975). Cool, wet conditions in the field are also conductive to infection of tomato fruit by *Stemphylium herbarum* (Butler, 1959).

During storage, the three cardinal factors leading to disease development are temperature, humidity, and the gaseous environment. Of these, temperature probably has the most profound effect because it affects evaporation and the physiology of the host, pathogen and epiphytic microbes. For many vegetables, storage at low temperature is the most effective and practical way of delaying the development of fungal decay. This is due to retardation of the maturation process, thereby prolonging the disease resistance associated with immaturity, and/or by inhibiting pathogen growth or development (Dennis, 1987). However, sound tomatoes can become susceptible to infection if stored at low temperatures, particularly if the fruit is contaminated with *Fusarium* spp. (Snowdon, 1992). Postharvest chilling injury can also result in the surface of vegetables becoming more susceptible to direct invasion by various pathogens—e.g., *B. cinerea* on peppers, *Pythium* spp. on yam, and *Cladosporium* spp. on tomato (Raj et al., 1986; Ramsey and Heiberg, 1952; Snowdon, 1992).

With various postharvest fungal pathogens, storage at 0°C will only delay the onset of decay as the organisms are capable of growth, albeit slowly, at this temperature (Dennis and Cohen, 1976). Watery soft rot of carrot, celery and brassicas caused by *Sclerotinia sclerotiorum* and gray mold rot of carrot caused by *B. cinerea* are typical examples of diseases that can develop at 0°C (Lowings, 1955; Snowdon, 1992), whereas *Pseudocercosporella capsella* continues to develop on brassicas at temperatures below 4°C (Crossan, 1954). Fungi such as *R. stolonifer*, on the other hand, are incapable of growth at temperatures below 4.5 to 5°C (Dennis and Cohen, 1976; Pierson, 1966). With tuberous or root crops such as potato, sweet potato, onion, carrot and yam, it often is advantageous to store the commodity at temperatures above the ideal storage temperatures for a short period after harvest (Dennis, 1987). During this "curing" period, wounds heal and the periderm

toughens. If done immediately after harvesting, this curing process can prevent or reduce wound infections during subsequent storage.

Relative humidity in the storage environment affects the activity of fungi on the surface of vegetables, but the effect depends on temperature. For vegetables that can be stored at 0°C, particularly those prone to evaporative loss, storage at 98% to 100% relative humidity is recommended (Dennis, 1987). Water loss from carrots, by as little as 8%, substantially increased their susceptibility to infection by *B. cinerea* and *R. stolonifer* (Goodliffe and Heale, 1977; Heale et al., 1977; Thorne, 1972). With cabbage, on the other hand, some dehydration of the outer wrapper leaves reduces fungal attack (Geeson and Browne, 1979). Where vegetables have to be stored at higher temperatures, as to prevent chilling injury, lower relative humidity in storage helps to avoid excessive microbial growth as well as condensation due to temperature fluctuations. Fortunately, quite a number of vegetables requiring such storage—e.g., tomato, peppers, aubergine and cucumber—have a waxy cuticle allowing them to retain moisture more effectively than leafy tissues (Robinson et al., 1975).

Controlled atmosphere (CA) storage employing low oxygen (<5%) or increased carbon dioxide levels (5% to 20%) have been used to slow down respiration of both the host and the pathogen, thereby suppressing development of postharvest rots. The efficacy of CA can further be improved by the inclusion of carbon monoxide, Fungi like *Rhizoctonia solani* and *Sclerotium rolfsii* are known to be intolerant of low oxygen and/or high carbon dioxide levels (Durbin, 1955; Mitchell and Mitchell, 1973), but conflicting results have been reported for other species, particularly *B. cinerea*. *B. cinerea* can grow, albeit at a reduced rate, at oxygen levels as low as 1% to 1.4% (Adair, 1971; Follstad, 1966). A raised carbon dioxide concentration of 8% stimulated growth if 2% oxygen was supplied and inhibition occurred only at carbon dioxide concentrations higher than 20% (Jarvis, 1977). Despite this apparent tolerance to a CA environment, storage in a CA containing 5% to 6% carbon dioxide and 3% oxygen consistently reduced spoilage by *B. cinerea* (Bohling and Hansen, 1977; Geeson and Browne, 1980; Henze, 1977). The controlling effect of CA therefore seems to be a consequence of delayed plant senescence, which restricts infection by the fungus (Yoder and Whalen, 1975). The effect of carbon monoxide on fungi associated with postharvest decay of vegetables has not been investigated extensively, but as most fungi are obligate aerobes it is obvious that the blockage of electron transport by carbon monoxide would impede their metabolic activity, albeit apparently to varying degrees (El-Goorani and Sommer, 1979).

V. HEALTH RISK OF FUNGAL VEGETABLE DISEASES TO HUMANS AND ANIMALS

Fungi, particularly those producing dry spores, such as *Alternaria*, *Aspergillus*, *Cladosporium* and *Penicillium*, are common allergens (Lacey, 1991). Although species of these genera frequently cause disease in vegetables, cases of allergic alveolitis, rhinitis, sinusitis, or asthma (in humans) attributable to these fungi in a postharvest vegetable environment are rare. Similarly, species in the genera *Alternaria*, *Aspergillus*, *Cladosporium*, *Fusarium*, *Geotrichum*, *Mucor*, *Mycocentrospora*, *Penicillium*, *Phoma*, *Rhizopus*, *Trichoderma*, and *Ulocladium* have been reported to cause opportunistic mycoses in humans (Kwon-Chung and Bennett, 1992; Larone, 1995). However, such opportunistic infections mostly occur only in humans compromised by malnutrition, alcoholism, cancer, diabetes, leukemia, infectious disease, trauma from surgery or injury, altered microbiota from prolonged use

of antibiotics, or immunosuppression by drugs, hormones, genetic deficiencies, etc. (Prescott et al., 1999). Nevertheless, as much as 90% of the mortality of AIDS is attributed to infection by organisms not normally pathogenic to healthy individuals (Mills and Masur, 1990), and such infections could therefore become more prevalent as the HIV epidemic spreads.

Mycotoxins are secondary metabolites produced by fungi that are harmful to humans and animals. Approximately 300 mycotoxins have been described and various outbreaks of mycotoxicoses have been documented (Cole and Cox, 1981; Marasas and Van Rensburg, 1979; Scudamore, 1998; Wyllie and Morehouse, 1977/78). Most of these cases have been ascribed to the consumption of contaminated grains or legumes. Several fungi infecting fruit and vegetables are also capable of producing mycotoxins. Manufacturers of juices, purees, baby foods, etc. use bulk quantities of fresh produce, rendering quality control of individual fruit or vegetables impractical and, hence, mycotoxin contamination of the processed product is more likely. Also, fungi that colonize the diseased or dead tissues associated with postharvest decays could be major sources of mycotoxins. By contrast, produce infected by nontoxigenic primary invaders (Sarantinos et al., 1996) or colonized by saprobic fungi (Ashworth et al., 1965), sometimes is protected from invasion by toxigenic fungi. Extensively decayed produce often is fed to farm animals. Mycotoxins in such feed could eventually end up in products destined for human consumption (De Iongh et al., 1964; Dorner et al., 1994; Kipper et al., 1991; Lusky et al., 1998; Micco et al., 1988; Prelusky, 1994). Humans themselves can transfer mycotoxins to their fellow beings, e.g. aflatoxin via mother's milk (Coulter et al., 1984) or through blood transfusion (Onyemelukwe and Ogbadu, 1981). Particularly perturbing are the many instances where animals themselves are affected by mycotoxins, often fatally. In this regard the increasing number of reports referring to real, suspected or potential mycotoxicoses in wildlife (Abbas and Bosch, 1990; Cole et al., 1988; Galash and Marchenko, 1991; Howerth et al., 1989; Huff et al., 1992; Lee et al., 1991; Li et al., 1994; Morrell and Adams, 1993; Neiger et al., 1995; Ruff et al., 1992; Sauviat et al., 1991) are of considerable concern.

Two well-known "mycotoxicoses" associated with vegetable diseases are celery photodermatitis and moldy sweet potato toxicosis. Celery photodermatitis is a contact dermatitis occurring among celery harvesters and results from sensitization of the skin to sunlight by the handling of celery plants infected by *Sclerotinia sclerotiorum*. Exposure of pigs to infected material and to ultraviolet (UV) light induces symptoms resembling foot-and-mouth disease (Montgomery et al., 1987). The active compounds with phototoxic activity formed in infected celery tissue have been identified as the furocoumarins (psoralens) xanthotoxin and bergapten (Kadis et al., 1972). These compounds are not formed when other vegetables are infected by *S. sclerotiorum*, nor are they induced in celery infected by other decay fungi (Wu et al., 1972). However, furocoumarin production in celery can be triggered by infection with the soft-rot bacterium *Erwinia carotovora* (Karasawa et al., 1990), insect feeding (Trumble et al., 1994), and even by spraying of celery with fungicides such as chlorothalonil, mancozeb, or copper oxychloride (Nigg, 1997). Evidence indicates that carrots also produce photoactive furocoumarins in response to disease (Ceska et al., 1986), although the disease was not specified.

Moldy sweet potato toxicosis is a fatal respiratory disease of cattle attributed to the ingestion of mold-damaged sweet potatoes. Pathological lesions are restricted to the lungs and include edema, congestion and hemorrhage. These symptoms are induced by the pulmonary toxins 4-ipomeanol and ipomeanine, produced by sweet potatoes in response to invasion by certain fungi that are not otherwise toxic. Although chemical or physical

damage as well as various invading organisms can stimulate sweet potato to produce furanoterpenoid stress metabolites such as ipomeamerone, the above pulmonary toxins are formed only in the presence of specific fungi, particularly *Fusarium solani* (Burka et al., 1977).

In the above two examples, toxicity is induced by stress metabolites or phytoalexins produced by the host plant in response to fungal infection, rather than by the fungus itself. Although plant stress metabolites have received considerable attention in phytopathological literature, their effects on human and animal health have mostly been neglected. However, based on epidemiological grounds, the congenital birth defect spina bifida has been linked with the consumption of *Phytophthora infestans*–blighted potatoes by pregnant women (Renwick, 1972). This hypothesis was supported by experimental evidence that blighted potatoes could induce fetal malformations involving osseous defects of the cranial vault in marmosets (Poswillo et al., 1972). Subsequent studies showed that extracts of healthy potatoes could be teratogenic, and ascribed the toxicity to solanine, a steroid glycoside of the saponin group present in many solanaceous plants (Jelinek et al., 1976). Solanine levels increase when tubers are attacked by an incompatible race of *P. infestans* (host resistance), but not when tubers are infected by a compatible race (host susceptibility) (Kadis et al., 1972). Toxic glycoalkaloids such as α-solanine and α-chaconine are natural constituents of healthy potato tubers (Morgan and Coxon, 1987). The concentration of such chemicals in uninfected tubers is affected by the genetic constitution of the plant (Sanford and Sinden, 1972), conditions of cultivation, and postharvest treatment of tubers (Jadhav et al., 1981).

Numerous reports refer to the isolation of toxigenic fungi from vegetables, whereas some describe the production of mycotoxins in artificially infected produce (Blumenthal-Yonassi et al., 1988; Bosch et al., 1992; Desjardins and Plattner, 1989; El-Banna et al., 1984; Latus-Zietkiewicz et al., 1995). Mycotoxins that have been detected as natural contaminants in vegetables and related crops include aflatoxin in cassava (Gerona, 1986; Marasas and Van Rensburg, 1979), cytochalasin B, trichothecenes, and sambutoxin in potatoes (Kim et al., 1995; Lafont et al., 1983; Scott et al., 1975), zearalenone in sugar beet and celery (Bosch and Mirocha, 1992; Li and Meng, 1989), T-2 toxin and fusarenon-X in *Cucurbita ficifolia* (Carrillo, 1990), diacetoxyscirpenol, T-2 toxin and zearalenone in mustard seed (Chakrabarti and Ghosal, 1987), and aflatoxin B_1, zearalenone, ochratoxin A and citrinin in root drugs from *Achyranthes aspera*, *Acorus calamus*, *Adhatoda vasica*, *Clerodendrum serratum*, and *Picrorhiza kurroa* (Roy and Chourasia, 1990). The presence of aflatoxin B_1 in any commodity is of particular concern, as the compound is not only extremely toxic but also one of the most potent naturally occurring carcinogens known (Eaton and Groopman, 1994). Citrinin is a known cause of porcine nephrotoxicosis (Scott, 1977), whereas zearalenone can cause porcine hyperestrogenism and infertility in sheep. Trichothecenes such as deoxynivalenol, fusarenon-X and T-2 toxin may cause feed refusal, emesis and internal hemorrhage in animals (Marasas et al., 1984; Towers and Sprosen, 1992). Available evidence furthermore implicates trichothecene mycotoxins, particularly T-2 toxin, in outbreaks of alimentary toxic aleukia, a fatal human disease characterized by necrotic stomatitis, exhaustion of the bone marrow, extreme leukopenia, and multiple hemorrhages. Since the beginning of the nineteenth century this disease has been recorded in the former U.S.S.R. (Lutsky et al., 1978). Cytochalasin B has toxic effects on membrane function and the contractile mechanisms of cell movement and division (Griffin, 1994), whereas tenuazonic acid has been implicated in thrombocytopenic purpura, an acute bleeding disease endemic to Africa (Rabie et al., 1975; Steyn and Rabie, 1976). Ochratoxin A

can cause porcine nephropathy and is possibly also involved in Balkan nephropathy, a fatal chronic kidney disease that occurs in some rural populations in Bulgaria, Rumania, and Yugoslavia (Kuiper-Goodman and Scott, 1989). There is, furthermore, mounting evidence that ochratoxin A is a genotoxic carcinogen (Dirheimer, 1996). Sambutoxin has been considered as a possible source of mycotoxicosis in experimental and farm animals, but its toxicity is relatively low (Kim et al., 1995).

A class of mycotoxins currently receiving considerable attention are the fumonisins, a group of related polar metabolites that have been implicated in mycotoxicoses such as porcine pulmonary edema (Osweiller et al., 1992), equine leukoencephalomalacia (Wilson et al., 1990), and human esophageal cancer (Chu and Li, 1992, 1994; Marasas et al., 1993; Rheeder et al., 1992; Yoshizawa et al., 1994). Fumonisins have also been reported to increase serum cholesterol levels and to induce chronic hepatotoxicity in vervet monkeys (Fincham et al., 1992). The above toxicoses have mostly been attributed to, or resulted from, the ingestion of corn contaminated with *Fusarium moniliforme*, the species from which fumonisins originally were isolated (Bezuidenhout et al., 1988). However, an increasing number of species are reported to produce fumonisins—e.g., isolates of *Fusarium anthophilum* (A. Braun) Wollenw.; *F. dlamini* Marasas, Nelson and Tousson; *F. globosum* Rheeder, Marasas and Nelson; *F. napiforme* Marasas, Nelson, and Rabie; *F. nygamai* Burgess and Trimboli; *F. oxysporum*, and *F. proliferatum* (Matsushima) Nirenberg (Abbas et al., 1995; Nelson et al., 1992; Seo et al., 1996; Sydenham et al., 1997; Thiel et al., 1991). Of the above species, *F. moniliforme* and *F. oxysporum* are important postharvest pathogens on various vegetable crops (Table 1), whereas species such as *F. proliferatum* and *F. nygamai*, which were described relatively recently (Burgess and Trimboli, 1986; Nirenberg, 1976), could show an increase in significance in future. Indeed, a recent report (Theron, 1999) has shown the last two species to be nonpathogenically associated with dry rot and stem-end rot of potato tubers in South Africa. It is furthermore noteworthy that fumonisins are structurally closely related to phytotoxic AAL-toxins produced by strains of *Alternaria alternata* (Mirocha et al., 1992) and that some isolates of *A. alternata* actually produce fumonisins (Abbas and Riley, 1996; Chen et al., 1992).

VI. DISEASE MANAGEMENT

Most plant disease control programs rely on the use of agrochemicals, and vegetable diseases are no exception to the rule. Control of postharvest diseases with chemicals often depends on an integrated approach, combining application of fungicides in the field to prevent preharvest infection with packinghouse or storage applications to control postharvest infection. The latter treatment also prevents or reduces spread of decay from infected to sound fruit or vegetables (Harvey, 1978). The use of fungicides for the control of postharvest diseases has previously been extensively reviewed (e.g., Dennis, 1987; Dixon, 1981; Eckert and Sommer, 1967; Sherf and MacNab, 1986; Chap. 18), and information regarding new compounds appearing on the market is readily available from agrochemical companies. A growing international concern over possible detrimental effect of fungicides on human health and the environment along with the development of pathogen resistance to fungicides have resulted in a greater need for development of alternative control measures. Furthermore, naturally occurring epiphytic inhabitants of aerial plant parts often influence phytopathogenic diseases (Andrews, 1992; Blakeman and Fokkema, 1982; Fokkema, 1993), and elimination of these natural inhabitants by fungicides through the "nontarget"effect can lead to the development of iatrogenic diseases (Griffiths, 1981). Due to

these concerns, considerable effort has been devoted to the discovery of alternative measures for protecting vegetables from disease. As is evident from Chapter 23, biological control shows great potential to be an effective and environmentally compatible option. Other alternative control strategies—such as inoculum reduction through basic sanitation practices, prevention of wounding through safer harvesting and handling, and maintenance of an effective cold chain to prevent disease development—are discussed in Chapters 24, 16, and 17 respectively, while the innovative application of biotechnology and genetic engineering to improve resistance of plants to disease is dealt with in Chapter 19.

Except for disease resistance, the above control strategies primarily concern the postharvest milieu. However, prevention of disease should preferably start at preharvest level. For this purpose various options are available, broadly classifiable as cultural, ecological, physical, or novel. Cultural measures comprise pathogen exclusion, field hygiene, crop rotation, tillage, fertilization, irrigation, and plant spacing, about which more information is available in reviews by Gurkin and Jenkins (1985), Kahn (1991), Leach et al. (1993), Palti (1981), Punja et al. (1986), Scholte (1992), Steadman et al. (1973), Snowdon (1992), Sumner et al. (1981), and others. Ecological aspects pertinent to preharvest management of vegetable diseases include classical biological control (Cook, 1993), mycorrhizae (Schenck, 1987), exploitation of naturally disease-suppressive soils and induction of soil suppressiveness (Hornby, 1983; Ko, 1982; Schneider, 1982; Schroth and Hancock, 1982), exclusion of insect vectors and damage (Gadoury et al., 1989), effective control of weed hosts (Raid and Pennypacker, 1987), antagonistic plants (Cook and Baker, 1983), decoy crops (White, 1954; Winter and Winiger, 1983), and organic soil amendment (Hoitink et al., 1991).

The utilization of composted organic material to control plant diseases may involve the development of systemic acquired resistance in the host. Soil incorporation of mature composts increases yield and plant and fruit size (Bryan et al., 1995; Obreza and Reeder, 1994; Roe et al., 1993), and it can suppress plant diseases caused by soil-borne organisms (Boehm and Hoitink, 1992; De Brito Alvarez et al., 1995; Hardy and Sivasithamparam, 1991; Schueler et al., 1989; Widmer et al., 1998). Disease-suppressive composts possess high microbial activity (Chen et al., 1988), and various organisms antagonistic to soil-borne pathogens have been isolated from them (Kuter et al., 1983; Kwok et al., 1987; Nelson and Hoitink, 1983). These findings suggest that suppressive organisms may at least partly be responsible for the decrease in disease observed in plants grown in compost-amended substrates. However, several reports indicate that composts may increase resistance of plants to disease (Roe et al., 1993; Tränkner, 1992; Zhang et al., 1996). It is unclear how increased resistance is brought about, but plants in compost mixes are colonized by various bacterial taxa, some of which are capable of inducing systemic resistance in plants (Liu et al., 1995; Maurhofer et al., 1994; Van Peer et al., 1991; Wei et al., 1991), possibly due to enhanced activity or accumulation of pathogenesis-related proteins (Maurhofer et al., 1994; Zhang et al., 1996, 1998). Effective control of foliar diseases has also been achieved with compost water extracts applied as topical sprays (Elad and Shtienberg, 1994; McQuilken et al., 1994; Stindt and Weltzien, 1990; Weltzien, 1992; Yohalem et al., 1994). Direct antagonism to and competition with the pathogen by the biota in the compost extract obviously play a role in disease suppression. However, filter-sterilized extracts have also been shown to exhibit activity (Cronin et al., 1996; Stindt and Weltzien, 1990; Weltzien, 1992; Yohalem et al., 1994), and it has been proposed that the protective effect of the extracts is due, at least in part, to the induction of systemic resistance in plants (Weltzien, 1992).

Classical biological control—i.e., the utilization of introduced antagonists—has been evaluated successfully against preharvest infections that cause postharvest losses (see Chap. 22). Potential antagonists can be applied to soil before or after planting (Strashnov et al., 1985; Velvis and Jager, 1983) or to seed (Elad et al., 1986), seedlings (Thirumalachar and O'Brien, 1977), foliage (Baker et al., 1985), or flowers (Nelson and Powelson, 1988).

Physical methods of disease control rely mainly on incineration or thermal inactivation or destruction of the pathogen. Flame and fire have been used for ages to destroy infected plant residues and to disinfest equipment (Hardison, 1976). Hot water treatment, although not applicable to the preharvest situation, is a well-known means of controlling fungal infections, particularly if the commodity is to be sold soon after harvesting (Barkai-Golan and Phillips, 1991). Combining hot water with fungicide treatment can improve control of diseases such as scurf on sweet potato caused by *Monilochaetes infuscans* and *Fusarium* rot of cucurbits (Clark and Moyer, 1988); Zitter et al., 1996), probably due to an increase in chemical activity or deposition and penetration of the fungicides (Barkai-Golan and Phillips, 1991). Exposure of some storage organs to warm air (curing) removes excess moisture from their surfaces and promotes the healing of wounds through enhanced formation of periderm, thus restricting infection of the organs by weak pathogens (Kushman, 1959).

Soil solarization—i.e., inactivation of pathogens through hydrothermal heating of soil accomplished by covering moist soil with transparent polyethylene sheeting in summer (Katan et al., 1976)—has been used successfully for controlling various soil-borne pathogens. There are, however, indications that soil solarization also reduces diseases caused by foliar pathogens by eradicating infected plant residues and/or by affecting plant resistance through changes in the mineral content of the soil (Katan and DeVay, 1991). Fungal genera associated with postharvest vegetable disease that are suppressed by soil solarization include *Alternaria* (Abu-Blan and Abu-Gharbieh, 1994), *Botrytis*, *Cladosporium* (Garibaldi and Gullino, 1991), *Colletotrichum* (Davis and Sorensen, 1986), *Fusarium* (Katan et al., 1980; Raj and Kapoor, 1993), *Macrophomina* (Dubey, 1992), *Phoma* (Katan et al., 1980), *Phytophthora* (Chellemi et al., 1994; Yücel, 1995), *Pythium* (Pullman et al., 1981), *Rhizoctonia* (Keinath, 1995; Osman and Saheb, 1983), *Sclerotinia* (Phillips, 1990; Porter and Merriman, 1983) and *Sclerotium* (Basallote-Ureba and Melero-Vara, 1993; Duff and Connelly, 1993). Unlike fumigation with, for instance, methyl bromide, soil solarization does not create a biological vacuum, which renders soil conducive to invasion by pathogens. Indeed, it often induces suppressiveness that protects the soil from reinfestation (DeVay and Katan, 1991). Furthermore, soil solarization affects not only the biotic composition, but also the structure of the soil and soluble minerals available for plant and microbial growth (Chen and Katan, 1980; Stapleton and DeVay, 1986). It is therefore not surprising that plants often grow better in solarized soil even where no pathogen is involved (Stapleton et al., 1985). This improvement in growth response is ascribed to increased nutrient levels in the soil solution, stimulation of beneficial rhizosphere microorganisms, including mycorrhizal symbionts and nitrogen-fixing bacteria, and enhanced physiological processes in the plant (Chen et al., 1991; Gamliel and Katan, 1991; Gruenzweig et al., 1993; Nair et al., 1990). Soil solarization has also found application in the treatment of fresh (Duff and Barnaart, 1992) and recycled (Gamliel et al., 1989) container media and in the reclamation of organic soils (Meron et al., 1989). Efficacy of the process can be enhanced by supplementation with introduced antagonists (Ristaino et al., 1991; Sivan and Chet, 1993), reduced dosages of methyl bromide (Yücel, 1995), solar-heated water (Abu-Gharbieh, 1991), irrigation (Lodha, 1995), fertilization or organic soil amend-

ment (Gamliel and Stapleton, 1993a), particularly with cabbage residues (Gamliel and Stapleton, 1993b).

Novel approaches that have been described for the control of postharvest vegetable diseases include the use of ultraviolet (UV)-irradiation or ozone for decontaminating irrigation water (Runia, 1990; Stangellini et al., 1984), inhibition of pathogens with surfactants (Stanghellini et al., 1996), radurization of produce (Barkai-Golan et al., 1968), and storage and transport under hypobaric atmospheres instead of an atmosphere low in oxygen and high in carbon dioxide or monoxide (Apelbaum and Barkai-Golan, 1977; Sommer, 1982). Other recent developments in disease control are the coating of fruit and vegetables with antitranspirant compounds that form a protective film on the plant surface (Han, 1990; Ziv and Zitter, 1992), spraying of plants with bicarbonates (Ziv and Zitter, 1992), and prevention of sporulation in pathogenic fungi that require light in the ultraviolet range for sporulation by equipping greenhouses with a special UV-absorbing vinyl film (Jarvis, 1989).

Systemic resistance to infection can be directly or indirectly induced in plants by exposing them to chemical compounds such as salicylic acid (Schneider and Ullrich, 1994), oxalate (Doubrava et al., 1988), phosphates (Gottstein and Kuc, 1989), unsaturated fatty acids (Cohen et al., 1991), jasmonic acid (Cohen et al., 1993), DL-3-amino-*N*-butanoic acid (Cohen, 1994); silicon (Chérif et al., 1992) and chitosan (Walker-Simmons et al., 1983). Besides inducing resistance in plants, chitosan is also known to initiate the formation of structural barriers in host tissue (El Ghaouth et al., 1994), cause morphological and cytological alterations in the pathogen (Benhamou, 1992; El Ghaouth et al., 1992) and, when applied as a coating to strawberries, to delay ripening of the berries by decreasing their respiration rate (El Ghoath et al., 1991).

Plants contain a variety of antifungal compounds (Wanger, 1985; Grayer and Harborne, 1994), many of which have been implicated in the natural resistance of the plant to infection. These compounds, in pure or crude form, have potential as natural fungicides, and some of them are known to induce systemic plant defense mechanisms—e.g., extracts from giant knotweed [*Reynoutria sachalinensis* F. Schmidt (Nakai)] (Daayf et al., 1997), spinach and rhubarb (Doubrava et al., 1988). Plants can also be "immunized" against disease by prior inoculation with the particular pathogen (Dalisay and Kuc, 1995), a different pathogen (Stroember and Brishammer, 1991), or with extracts of pathogenic organisms (Ricci et al., 1989). As indicated previously, even plant growth-promoting rhizobacteria have been shown to protect plants against foliar diseases (Wei et al., 1991).

ACKNOWLEDGMENT

The authors are greatly indebted to Elsie van Eeden, Amelita Lombard, Wilma Havenga, and Colette Cronjé for spending many hours retrieving information for this chapter.

REFERENCES

Abbas, H.K., and Bosch, U. 1990. Evaluation of trichothecene and nontrichothecene mycotoxins produced by *Fusarium* in soybeans. Mycotox. Res. 6:13–20.

Abbas, H.K., Ocamb, C.M., Xie, W., Mirocha, C.J., and Shier, W.T. 1995. First report of fumonisin B_1, B_2 and B_3 production by *Fusarium oxysporum* var. *redolens*. Plant Dis. 79:968.

Abbas, H.K., and Riley, R.T. 1996. The presence and phytotoxicity of fumonisins and AAL-toxin in *Alternaria alternata*. Toxicon Oxf. 34:133–136.

Abu-Blan, H.A., and Abu-Gharbieh, W.I. 1994. Effect of soil solarization on winter planting of potato, cauliflower and cucumber in the central Jordan Valley. Dirisat. Series B, Pure Appl. Sci. 21:203–213.

Abu-Gharbieh, W.I., Saleh, W.I.H., and Al-Banna, L. 1991. Application of solar-heated water for soil solarization. FAO Plant Prod. Prot. Paper 109:69–77.

Adair, C.N. 1971. Influence of controlled-atmosphere storage conditions on cabbage postharvest decay. Plant Dis. Reptr. 55:864–868.

Agrios, G.N. 1997. Plant pathology. Academic Press, London.

Alexopoulos, C.J., Mims, C.W., and Blackwell, M. 1996. Introductory mycology. Wiley, New York.

Andrews, J.H. 1992. Biological control in the phyllosphere. Annu. Rev. Phytopathol. 30:603–635.

Apelbaum, A., and Barkai-Golan, R. 1977. Spore germination and mycelial growth of postharvest pathogens under hypobaric pressure. Phytopathology 67:400–403.

APSnet. 1996. http://www.SCISOC.ORG/resource/common/tofc.htm.

Ashworth, L.J., Schroeder, H.W., and Langley, B.C. 1965. Aflatoxin: Environmental factors governing occurrences in Spanish peanuts. Science 148:1228–1229.

Baker, C.J., and Stavely, J.R. 1985. Biocontrol of bean rust by *Bacillus subtilis* under field conditions. Plant Dis. 69:770–772.

Barkai-Golan, R., and Phillips, D.J. 1991. Postharvest heat treatment of fresh fruits and vegetables for decay control. Plant Dis. 75:1085–1089.

Barkai-Golan, R., Kahan, R.S., and Temkin-Gorodeiski, N. 1968. Sensitivity of stored melon fruit fungi to gamma irradiation. Int J. Appl. Radiat. Isotopes 19:579–583.

Basallote-Ureba, M.J., and Melero-Vara, J.M. 1993. Control of garlic white rot by soil solarization. Crop Prot. 12:219–223.

Bateman, D.F., and Basham, H.G. 1976. Degradation of plant cell walls and membranes by microbial enzymes, p. 316–355. In: R. Heitefuss and P.H. Williams (eds). Physiological plant pathology. Springer-Verlag, Berlin.

Benhamou, N. 1992. Ultrastructural and cytochemical aspects of chitosan on *Fusarium oxysporum* f. sp. *radicis-lycopersici*, agent of tomato crown and root rot. Phytopathology 82:1185–1193.

Besri, M. 1983. Lettuce contre le chancre a Didymella lycopersicidela tomato parchauffage solaire (solarisation) des tuteurs. Phytopathol Zeitschr 108:333–340.

Bezuidenhout, S.C., Gelderblom, W.C.A., Gorst-Allman, C.P., Horak, R.M., Marasas, W.F.O., Spiteller, G., and Vleggaar, R. 1988. Structure elucidation of fumonisins, mycotoxins from *Fusarium moniliforme*. J. Chem. Soc. Commun. 1988:743–745.

Blakeman, J.P., and Fokkema, N.J. 1982. Potential for biological control of plant diseases on the phylloplane. Annu. Rev. Phytopathol. 20:167–192.

Blumenthal-Yonassi, J., Paster, N., Barkai-Golan, R. 1988. Differences in zearalenone production by *Fusarium equiseti* strains in vitro and in fruits. Proc. Jpn. Assoc. Mycotoxicol. Suppl. 1: 232–233.

Boehm, M.J., and Hoitink, H.A.J. 1992. Sustenance of microbial activity in potting mixes and its impact on severity of Pythium root rot poinsettia. Phytopathology 82:259–264.

Bohling H., and Hansen, H. 1977. Storage of white cabbage (*Brassica oleracea* var. *capitata*) in controlled atmospheres. Acta Hort. 62:49–54.

Bosch, U., and Mirocha, C.J. 1992. Toxin production by *Fusarium* species from sugar beets and natural occurrence of zearalenone in beets and beet fibers. Appl. Environ. Microbiol. 58:3233–3239.

Bosch, U., Mirocha, C.J., and Wen, Y. 1992. Production of zearalenone, moniliformin and trichothecenes in intact sugar beets under laboratory conditions. Mycopathologia 119:167–173.

Brown, E.J., and Howard, R.J. 1994. Adhesion of fungal spores and germlings to host plant surfaces. Protoplasma 181:202–212.

Bruton, B.D. 1994. Mechanical injury and latent infections leading to postharvest decay. HortScience 29:747–748.

Bryan, H., Schaffer, B., and Crane, J.H. 1995. Solid waste compost for improved water conservation

and production of vegetable crops (tomatoes/squash)—Homestead. Florida Water Conservation/ Compost Utilization Program Final Report. Center for Biomass Programs, UF/IFAS, Gainesville, FL.

Burgess, L.W., and Trimboli, D. 1986. Characterization and distribution of *Fusarium nygamai*, sp. nov. Mycologia 78:223–229.

Burka, L.T., Kuhnert, L., Wilson, B.J., and Harris, T.M. 1977. Biogenesis of lung-toxic furans produced during microbial infection of sweet potatoes (*Ipomoea batatas*). J. Am. Chem. Soc. 99:2302–2305.

Butler, E.E. 1959. Fungi and rots in Californian canning tomatoes. Plant Dis. Rep. 43:187–192.

Butler, E.E. 1961. Transmission of *Geotrichum* rot of tomato fruit by *Drosphila melanogaster*. Phytopathology 51:250–255.

Butler, E.E., and Bracker, C.R. 1963. The role of *Drosophila melanogaster* in the epiphytology of *Geotrichum*, *Rhizopus* and other fruit rots of tomato. Phytopathology 53:1016–1020.

Carrillo, L. 1990. Micotoxinas de *Fusarium* spp. en frutos deteriorados de *Cucurbita ficifolia*. Rev. Argentina Microbiol. 22:212–215.

Ceska, O., Chaudhary, S.K., Warrington, P.J., and Ashwood-Smith, M.J. 1986. Furocoumarins in the cultivated carrot, *Daucus carota*. Phytochemistry 25:81–83.

Chakrabarti, D.K., and Ghosal, S. 1987. Mycotoxins produced by *Fusarium oxysporum* in the seeds of *Brassica campestris* during storage. Mycopathologia 97:69–75.

Chellemi, D.O., Olson, S.M., and Mitchell, D.J. 1994. Effects of soil solarization and fumigation on survival of soilborne pathogens of tomato in northern Florida. Plant Dis. 78:1167–1172.

Chen, J., Mirocha, C.J., Xie, W., Hogge, L., and Olson, D. 1992. Production of the mycotoxin fumonisin B_1 by *Alternaria alternata* f. sp. *lycopersici*. Appl. Environ. Microbiol. 58:3928–3931.

Chen, W., Hoitink, H.A.J., and Madden, L.V. 1988. Microbial activity and biomass in container media for predicting suppressiveness to damping-off caused by *Pythium ultimum*. Phytopathology 78:1447–1450.

Chen, Y., Gamliel, A., Stapleton, J.J., and Aviad, T. 1991. Chemical, physical, and microbial changes related to plant growth in disinfested soil, p. 103–129. In: Katan, J., and DeVay, J.E. (eds.). Soil solarization. CRC Press, Boca Raton, FL.

Chen, Y., and Katan, J. 1980. Effect of solar heating of soils by transparent polyethylene mulching on their chemical properties. Soil Sci. 130:271–277.

Chérif, M., Benhamou, N., Menzies, J.G., and Bélanger, R.R. 1992. Silicon induced resistance in cucumber plants against *Pythium ultimum*. Physiol. Mol. Plant Pathol. 41:411–425.

Chesson, A. 1980. Maceration in relation to the post-harvest handling and processing of plant material. J. Appl. Bacteriol. 48:1–45.

Chu, F.S., and Li, G.Y. 1994. Simultaneous occurrence of fumonisin B_1 and other mycotoxins in moldy corn from the People's Republic in China in regions with high incidence of oesophageal cancer. Appl. Environ. Microbiol. 60:847–852.

Clark, C.A., and Moyer, J.W. (eds). 1988. Compendium of sweet potato diseases. APS Press, St. Paul, MN.

Cohen, Y. 1994. Local and systemic protection against *Phytophthora infestans* induced in potato and tomato plants by DL-3-amino-n-butanoic acids. Phytopathology 84:55–59.

Cohen, Y., Gisi, U., and Mosinger, E. 1991. Systemic resistance of potato plants against *Phytophthora infestans* induced by unsaturated fatty acids. Physiol. Mol. Plant Pathol. 38:255–263.

Cohen, Y., Gisi, U., and Niderman, T. 1993. Local and systemic protection against *Phytophthora infestans* induced in potato and tomato plants by jasmonic acid and jasmonic acid methyl ester. Phytopathology 83:1054–1062.

Cole, R.J., and Cox, R.H. 1981. Handbook of toxic fungal metabolites. Academic Press, New York.

Cole, R.J., Dorner, J.W., Gilbert, J., Mortimer, D.N., Crews, C., Mitchell, J.C., Windingstad, R.M.,

Nelson, P.E., and Cutler, H.G. 1988. Isolation and identification of trichothecenes from *Fusarium compactum* suspected in the etiology of a major intoxication of sandhill cranes. J. Agr. Food Chem. 36:1163–1167.

Cook, R.J., 1993. Making greater use of introduced microorganisms for biological control of plant pathogens. Annu. Rev. Phytopathol. 31:53–80.

Cook, R.J., and Baker, K.F. 1983. The nature and practice of biological control of plant pathogens. APS Press, St. Paul, MN.

Coulter, J.B.S., Lamplugh, S.M., Suliman, G.I., Omer, M.I.A., and Hendrickse, R.G. 1984. Aflatoxins in human breast milk. Ann. Trop. Paediatr: 6:61–66.

Cronin, M.J., Yohalem, D.S., Harris, R.F. and Andrews, J.H. 1996. Putative mechanism and dynamics of inhibition of the apple scab pathogen *Venturia inaequalis* by compost extracts. Soil. Biol. Biochem. 28:1241–1249.

Crossan, D.F. 1954. *Cercosporella* leafspot of crucifers. Technical Bulletin of North Carolina Agricultural Experiment Station No. 109.

Daayf, F., Schmitt, A., and Bélanger, R.R. 1997. Evidence of phytoalexins in cucumber leaves infected with powdery mildew following treatment with leaf extracts of *Reynoutria sachalinensis*. Plant Physiol. 113:719–727.

Dalisay, R.F., and Kuc, J.A. 1995. Persistence of induced resistance and enhanced peroxidase and chitinase activities in cucumber plants. Physiol. Mol. Plant Pathol. 47:315–327.

Davis, J.R., and Sorensen, L.H. 1986. Influence of soil solarization at moderate temperatures on potato genotypes with resistance to *Verticillium dahliae*. Phytopathology 76:1021–1026.

Day, J.R., Lewis, B.G., and Martin, S. 1972. Infection of stored celery by *Centrospora acerina*. Ann. Appl. Biol. 71:201–210.

De Brito Alvarez, M.A., Gagné, S., and Antoun, H. 1995. Effect of compost on rhizosphere microflora of the tomato and on the incidence of plant growth-promoting rhizobacteria. Appl. Environ. Microbiol. 61:194–199.

De Iongh, H., Vles, R.O., and Van Pelt, J.G. 1964. Milk of mammals fed an aflatoxin-containing diet. Nature 202:466–467.

Dennis, C. 1983. Salad crops, p. 157–177. In: C. Dennis (ed.). Post-harvest pathology of fruits and vegetables. Academic Press, London.

Dennis, C. 1987. Fungi, p. 377–411. In: J. Weichmann (ed.). Postharvest physiology of vegetables. Marcel Dekker, New York.

Dennis, C., and Cohen, E. 1976. The effect of temperature on strains of soft fruit spoilage fungi. Ann. Appl. Biol. 82:51–56.

Derbyshire, D.M. 1973. Post harvest deterioration of vegetables. Chem. Ind. 22:1052–1054.

Desjardins, A.E., and Plattner, R.D. 1989. Trichothecene toxin production by strains of *Gibberella pulicaris (Fusarium sambucinum)* in liquid culture and in potato tubers. J. Agr. Food Chem. 37:388–392.

DeVay, J.E., and Katan, J. 1991. Mechanisms of pathogen control in solarized soils, p. 87–101. In: Katan, J., and DeVay, J.E. Soil solarization. CRC Press, Boca Raton, FL.

Dirheimer, G. 1996. Mechanistic approaches to ochratoxin toxicity. Food Addit. Contam. 13 (suppl.):45–48.

Dixon, G.R. 1981. Vegetable crop diseases. AVI, Westport, CT.

Domsch, K.H., Gams, W., and Anderson, T.-H., 1980. Compendium of soil fungi. Academic Press, London.

Dorner, J.W., Cole, R.J., Erlington, D.J., Suksupath, S., McDowell, G.H., and Bryden, W.L. 1994. Cyclopiazonic acid residues in milk and eggs. J. Agr. Food Chem. 42:1516–1518.

Doubrava, N.S., Dean, R.A., and Kuc, J. 1988. Induction of systemic resistance to anthracnose caused by *Colletotrichum lagenarium* in cucumber by oxalate and extracts from spinach and rhubarb leaves. Physiol. Mol. Plant Pathol. 33:69–79.

Dubey, R.C. 1992. Effects of soil solarization on the survival of *Macrophomina phaseolina* in fungicide-amended and unamended soils. Trop. Sci. 32:275–279.

Duff, J.D., and Barnaart, A. 1992. Solarisation controls soilborne fungal pathogens in nursery potting mixes. Aust. Plant Pathol. 21:20–23.

Duff, J.D., and Connelly, M.I. 1993. Effect of solarisation using single and double layers of clear plastic mulch on *Pythium*, *Phytophthora* and *Sclerotium* species in a nursery potting mix. Aust. Plant Pathol. 22:28–35.

Durbin, R.D. 1955. Straight-line function of growth of micro-organisms at toxic levels of carbon dioxide. Science 121:734–735.

Eaton, D.L., and Groopman, J.D. 1994. The toxicology of aflatoxins: Human health, veterinary, and agricultural significance. Academic Press, San Diego, CA.

Eckert, J.W. and Sommer, N.F. 1967. Control of diseases of fruits and vegetables by postharvest treatment. Annu. Rev. Phytopathol. 11:391–432.

Elad, Y., and Shtienberg, D. 1994. Effect of compost water extracts on grey mold (*Botrytis cinerea*). Crop Prot. 13:109–114.

Elad, Y., Zviely, Y., and Chet, I. 1986. Biological control of *Macrophomina phaseolina* (Tassi) Goid. by *Trichoderma harzianum*. Crop Protect. 5:288–292.

El-Banna, A.A., Scott, P.M., Lau, P.-Y., Sakuma, T., Platt, H., and Campbell, V. 1984. Formation of trichothecenes by *Fusarium solani* var. *coeruleum* and *Fusarium sambucinum* in potatoes. Appl. Environ. Microbiol. 47:1169–1171.

El Ghaouth, A., Arul, J., Asselin, A., and Benhamou, N. 1992. Antifungal activity of chitosan on post-harvest pathogens: Induction of morphological and cytological alterations in *Rhizopus stolonifer*. Mycol. Res. 96:769–779.

El Ghaouth, A., Arul, J., Grenier, J., Benhamou, N., Asselin, A., and Bélanger, R. 1994. Effect of chitosan on cucumber plants: Suppression of *Pythium aphanidermatum* and induction of defense reactions. Phytopathology 84:313–320.

El Ghaouth, A., Arul, J., Ponnampalam, R., and Boulet, M. 1991. Chitosan coating effect on storability and quality of fresh strawberries. J. Food Sci. 56:1618–1620, 1631.

El-Goorani, M.A., and Sommer, N.F. 1979. Suppression of postharvest plant pathogenic fungi by carbon monoxide. Phytopathology 69:834–838.

Ellis, M.B., and Ellis, J.P., 1985. Microfungi on land plants. Macmillan, New York.

Fincham, J.E., Marasas, W.F.O., Taljaard, J.J.F., Kriek, N.P.J., Badenhorst, C.J., Gelderblom, W.C.A., Seier, J.V., Smuts, C.M., Faber, M. Weight, M.J., Slazus, W., Woodroof, C.W., Van Wyk, M.J., Kruger, M., and Thiel, P.G. 1992. Artherogenetic effects in a nonhuman primate of *Fusarium moniliforme* cultures added to a carbohydrate diet. Artherosclerosis 94:13–25.

Fokkema, N.J. 1993. Opportunities and problems of control of foliar pathogens with microorganisms. Pestic. Sci. 37:411–416.

Follstad, M.N. 1966. Mycelial growth rate and sporulation of *Alternaria tenuis*, *Botrytis cinerea*, *Cladosporium herbarum*, and *Rhizopus stolonifer* in low-oxygen atmospheres. Phytopathology 56:1098–1099.

Friedman, B.A. 1960. Market diseases of fresh fruits and vegetables. Econ. Bot. 14:145–156.

Gadoury, D.M., McHardy, W.E., and Rosenburg, D.A. 1989. Integration of pesticide application schedules for disease and insect control in apple orchards of the northern United States. Plant Dis. 73:98–105.

Geeson, J.D. 1976. Storage rot of white cabbage caused by *Phytopthora porri*. Plant Pathol. 25: 115–116.

Galash, V.T., and Marchenko, A.M. 1991. The effect of trichothecene mycotoxins on carp. Bull. Eur. Assoc. Fish Pathol. 11:63–64.

Gamliel, A., and Katan, J. 1991. Involvement of fluorescent pseudomonads and other microorganisms in increased growth response of plants in solarized soils. Phytopathology 81:494–502.

Gamliel, A., Katan, J., Chen, Y., and Grinstein, A. 1989. Solarization for the recycling of container media. Acta Hort. 255:181–188.

Gamliel, A., and Stapleton, J.J. 1993a. Effect of chicken compost or ammonium phosphate and

solarization on pathogen control, rhizosphere microorganisms, and lettuce growth. Plant Dis. 77:886–891.

Gamliel, A., and Stapleton, J.J. 1993b. Characterization of antifungal compounds evolved from solarized soil amended with cabbage residues. Phytopathology 83:899–905.

Garibaldi, A., and Gullino, M.L. 1991. Soil solarization in southern European countries, with emphasis on soilborne disease control of protected crops, p. 227–235. In: Katan, J., and DeVay, J.E., eds. Soil solarization. CRC Press, Boca Raton, FL.

Geeson, J.D. 1976. Storage rot of white cabbage caused by *Phytophthora porri*. Plant Pathol. 25: 115–116.

Geeson, J.D., and Browne, K.M. 1979. Effect of post-harvest fungicide drenches on stored winter white cabbage. Plant Pathol. 28:161–168.

Geeson, J.D., and Browne, K.M. 1980. Controlled atmosphere storage of winter white cabbage. Ann. Appl. Biol. 95:267–272.

Gerona, G.R. 1986. Technology keeps cassava free of aflatoxin for four months. Cassava Newsl. 10:8.

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

Gottstein, H.D., and Kuc, J. 1989. Induction of systemic resistance to anthracnose in cucumber by phosphates. Phytopathology 79:176–179.

Grayer, R.J., and Harborne, J.B. 1994. A survey of antifungal compounds from higher plants, 1982–1993. Phytochemistry 37:19–42.

Griffin, D.H. 1994. Fungal physiology. Wiley-Liss, New York.

Griffiths, E. 1981. Iatrogenic plant diseases. Annu. Rev. Phytopathol. 19:69–82.

Gruenzweig, J.M., Rabinowitch, H.D., and Katan, J. 1993. Physiological and developmental aspects of increased plant growth in solarized soils. Ann. Appl. Biol. 122:579–591.

Gurkin, R.S., and Jenkins, S.F. 1985. Influence of cultural practices, fungicides and inoculum placement in southern blight and Rhizoctonia crown rot of carrot. Plant Dis. 69:477–481.

Han, J.-S. 1990. Use of antitranspirant epidermal coatings for plant protection in China. Plant Dis. 74:263–266.

Hancock, J.G., Millar, R.L., and Lorbeer, J.W. 1964. Pectolytic and cellulolytic enzymes produced by *Botrytis allii*, *B. cinerea* and *B. squamosa* in vitro and in vivo. Phytopathology 54:928–931.

Hardison, J.R. 1976. Fire and flame for plant disease control. Annu. Rev. Phytopathol. 14:355–379.

Hardy, G.E. St. J., and Sivasithamparam, K. 1991. Sporangial responses do not reflect microbial suppression of *Phytophthora drechsleri* in composted eucalyptus bark mix. Soil Biol. Biochem. 23:756–765.

Harrison, J.G., Searle, R.J., and Williams, N.A., 1997. Powdery scab disease of potato—A review. Plant Pathol. 46:1–25.

Hawksworth, D.L., Kirk, P.M., Sutton, B.C., and Pegler, D.N. 1995. Ainsworth & Bisby's dictionary of the fungi. CAB International, Wallingford, UK.

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

Henze, J. 1977. Influence of CA-storage on fermentation of white cabbage (*Brassica oleracea* L.). Acta Hort. 62:71–77.

Hoitink, H.A.J., Inbar, Y., and Boehm, M.J. 1991. Status of compost-amended potting mixes naturally suppressive to soilborne diseases of floricultural crops. Plant Dis. 75:869–873.

Hooker, W.J. 1983. Compendium of potato diseases. APS Press, St. Paul, MN.

Hornby, D. 1983. Suppressive soils. Annu. Rev. Phytopathol. 21:65–85.

Howerth, E.W., Wyatt, R.D., and Hayes, D.A. 1989. Equine leukoencephalomalacia in white tailed deer from North Carolina. J. Wildl. Dis. 25:384–387.

Huff, W.E., Ruff, M.D., and Chute, M.B. 1992. Characterization of the toxicity of the mycotoxins aflatoxin, ochratoxin, and T-2 toxin in game birds. II. Ringneck pheasant. Avian Dis. 36:30–33.

Jackson, O.A., and Taylor, C.B. 1996. Plant-microbe interactions: Life and death at the interface. Plant Cell 8:1651–1668.

Jadhav, S.J., Sharma, R.P., and Salunkhe, D.K. 1981. Naturally occurring toxic alkaloids in foods. CRC Crit. Rev. Toxicol. 9:21–104.

Jarvis, W.R. 1977. *Botryotinia* and *Botrytis* species. Taxonomy, physiology, and pathogenicity. Res. Stn. Can. Dep. Agric. Harrow, Monogr. 15.

Jarvis, W.R. 1989. Managing diseases in greenhouse crops. Plant Dis. 73:190–194.

Jelinek, R., Kyzlink, V., and Blattny, C. 1976. An evaluation of the embryotoxic effects of blighted potatoes on chicken embryos. Teratology 14:335–342.

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

Jones, J.B., Jones, J.P., Stall, R.E., and Zitter, T.A. 1991. Compendium of tomato diseases. APS Press, St. Paul, MN.

Kadis, S., Ciegler, A., and Ajl, S.J. (eds.). 1972. Microbial toxins. Vol. VIII. Academic Press, New York.

Kahn, R.P. 1991. Exclusion as a plant disease control strategy. Annu. Rev. Phytopathol. 29:219–246.

Karasawa, D., Shibata, H., Horiuchi, N. Andou, Y., and Simada, M. 1990. Photoactive furocoumarins in diseased celery (*Apium graveolens*). Agr. Biol. Chem. 54:2141–2142.

Katan, J., and DeVay, J.E. 1991. Soil solarization: historical perspectives, principles, and uses, p. 23–37. In: Katan, J., and DeVay, J.E., eds. Soil solarization. CRC Press, Boca Raton, FL.

Katan, J., Greenberger, A., Alon, H., and Grinstein, A. 1976. Solar heating by polyethylene mulching for the control of diseases caused by soil-borne pathogens. Phytopathology 66:683–688.

Katan, J., Rotem, I., Finkel, Y., and Daniel, J. 1980. Solar heating of the soil for the control of pink root and other soilborne diseases in onions. Phytoparasitica 8:39–50.

Keinath, A.P. 1995. Reduction in inoculum density of *Rhizoctonia solani* and control of belly rot on pickling cucumber with solarization. Plant Dis. 79:1213–1219.

Kim, J., Lee, Y., and Yu, S. 1995. Sambutoxin-producing isolates of *Fusarium* species and occurrence of sambutoxin in rotten potato tubers. Appl. Environ. Microbiol. 61:3750–3751.

Kipper, L., Schweigmann, A., and Vater, N. 1991. Untersuchungen zum Carry-over von Ochratoxin A in der Nahrungskette Futtermittel-Schlachtschwein-Fleischerzeugnis. Tierarzt.-Unschau 46: 538–542.

Ko, W.-H. 1982. Biological control of *Phytophthora* root rot of papaya with virgin soil. Plant Dis. 66:446–448.

Kolattukudy, P.E. 1985. Enzymatic penetration of the plant cuticle by fungal pathogens. Annu. Rev. Phytopathol. 23:233–250.

Kolattukudy, P.E., Rogers, L.M., Li, D., Hwang, C., and Flaishman, M.A. 1995. Surface signaling in pathogenesis. Proc. Natl. Acad. Sci. USA 92:4080–4087.

Kuiper-Goodman, T., and Scott, P.M. 1989. Risk assessment of the mycotoxin ochratoxin A. Biomed. Environ. Sci. 2:179–248.

Kushman, L.J. 1959. Curing of Porto Rico sweetpotatoes at 95°F for prevention of black rot in storage. Proc. Amer. Soc. Hort. Sci. 73:467–472.

Kuter, G.A., Nelson, E.B., Hoitink, H.A.J., and Madden, L.V. 1983. Fungal populations in container media amended with composted hardwood bark suppressive and conducive to Rhizoctonia damping-off. Phytopathology 73:1450–1456.

Kwok, O.C.H., Fahy, P.C., Hoitink, H.A.J. and Kuter, G.A. 1987. Interactions between bacteria and *Trichoderma hamatum* in suppression of Rhizoctonia damping-off in bark compost media. Phytopathology 77:1206–1212.

Kwon-Chung, K.J., and Bennett, J.E. 1992. Medical mycology. Lea & Febiger, Philadelphia.

Lacey, J. 1991. Aerobiology and health: The role of airborne fungal spores in respiratory disease, p. 157–185. In: Hawksworth, D.L., ed. Frontiers in mycology. CAB International, Wallingford, UK.

Lafont, P., Girard, T., Payen, J., Sarfati, J., and Gaillardin, M. 1983. Contamination de pommes de terre se consommation par des fusariotrichothecenes. Microbiol. Alim. Nutr. 1:147–152.

Larone, D.H. 1995. Medically important fungi. American Society for Microbiology. ASM Press, Washington, DC.

Latus-Zietkiewicz, D., Perkowski, J., and Chelkowski, J. 1995. Mycotoxins production, pathogenicity and toxicity of *Fusarium* species isolated from potato tubers with dry rot injuries. Microbiol. Alim. Nutr. 13:87–100.

Leach, S.S., Porter, G.A., Rourke, R.V., and Clapham, W.M. 1993. Effects of moldboard plowing, chisel plowing and rotation crops on the Rhizoctonia disease of white potato. Am. Pot. J. 70: 329–337.

Lee, B.C., Hendricks, J.D., and Bailey, G.S. 1991. Toxicity of mycotoxins to fish, p. 607–626. In:Smith, J.E., and Henderson, R.S. (eds). Mycotoxins and animal foods. CRC Press, Boca Raton, FL.

Li, H.X., and Meng, F.J. 1989. The isolation and identification of zearalenone from *Apium graveolens*. Acta Phytophysiol. Sinica 15:211–215 (in Chinese).

Li, M.H., Raverty, S.A., and Robinson, E.H. 1994. Effects of dietary mycotoxins produced by the mold *Fusarium moniliforme* on channel catfish *Ictalurus punctatus*. J. World Aquacult. Soc. 25:512–516.

Liu, L., Kloepper, J.W., and Tuzun, S. 1995. Induction of systemic resistance in cucumber by plant growth-promoting rhizobacteria: Duration of protection and effect of host resistance on protection and root colonization. Phytopathology 85:1064–1068.

Lodha, S. 1995. Soil solarization, summer irrigation and amendments for the control of *Fusarium oxysporum* f. sp. *cumini* and *Macrophomina phaseolina* in arid soils. Crop Protect. 14:215–219.

Lowings, P.H. 1955. Some storage rots of celery caused by *Centrospora acerina* and other fungi. Plant Pathol. 4:106–107.

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

Lusky, K., Gobel, R., Tesch, D., Tenner, G., Haider, W., Kruger, M., and Lippert, A. 1998. Tiergesundheit und Ruckstandsverhalten beim Schwein bei gleichzeitiger Aufnahme der Mykotoxine Ochratoxin A (OTA) und Deoxynivalenol (DON) über das Futter im 90-Tage-Test. Tierarzt. Umschau 53:623–630.

Lutsky, I., Mor, N., Yagen, B., and Joffe, A.Z. 1978. The role of T-2 toxin in experimental alimentary toxic aleukia: A toxic study in cats. Toxicol. Appl. Pharmacol. 43:111–124.

Luttrell, E.S. 1947. *Diaporthe phaseolum* var. *sojae* on crop plants. Phytopathology 37:445–465.

Lyons, J.M. 1973. Chilling injury in plants. Annu. Rev. Plant Physiol. 24:445–466.

Marasas, W.F.O., Nelson, P.E., and Toussoun, T.A. 1984. Toxigenic *Fusarium* species. Pennsylvania State University Press, University Park, PA.

Marasas, W.F.O., Thiel, P.G., Gelderblom, W.C.A., Shephard, G.S., Sydenham, E.W., and Rheeder, J.P. 1993. Fumonisins produced by *Fusarium moniliforme* in maize: Foodborne carcinogens of pan African importance. Afr. Newslett. Occup. Health Saf. Suppl. 2:11–18.

Marasas, W.F.O., and Van Rensburg, S.J. 1979. Mycotoxins and their medical and veterinary effects, p. 357–378. In: Horsfall, J.G. and Cowling, E.B. (eds.). Plant disease. Vol. 4. Academic Press, London.

Maurhofer, M. Hase, C., Meuwly, P., Métraux, J.-P., and Défago, G. 1994. Induction of systemic resistance of tobacco to tobacco necrosis virus by the root-colonizing *Pseudomonas fluorescens* strain CHAO: Influence of the *gacA* gene and pyoverdine production. Phytopathology 84:139–146.

McQuilken, M.P., Whipps, J.M., and Lynch, J.M. 1994. Effects of compost extracts of a composted manure-straw mixture on the plant pathogen *Botrytis cinerea*. World J. Microbiol. Biotechnol. 10:20–26.

Meron, M., Filler, Y., Cohen, Y., Grinstein, A., Gamliel, A., Chen, Y., and Katan, J. 1989. Solarization and fumigation for reclamation of organic soils. Acta Hort. 255:117–124.

Micco, C., Miraglia, M., Benelli, L., Onori, R., Ioppolo, A., and Mantovani, A. 1988. Long term administration of low doses of mycotoxins in poultry. 2. Residues of ochratoxin A and aflatoxins in broilers and laying hens after combined administration of ochratoxin A and aflatoxin B_1. Food Add. Contam. 5:309–314.

Mills, J., and Masur, H. 1990. AIDS-related infections. Sci. Am. 263:50–57.

Mirocha, C.J., Gilchrist, D.G., Shier, W.T., Abbas, H.K., Wen, Y., and Vesonder, R.F. 1992. AAL toxins, fumonisins (biology and chemistry) and host-specificity concepts. Mycopathologia 117: 47–56.

Mitchell, D.J., and Mitchell, J.E. 1973. Oxygen and carbon dioxide concentration effects on the growth and reproduction of *Aphanomyces euteiches* and certain other soil-borne plant pathogens. Phytopathology 63:1053–1059.

Montgomery, J.F., Oliver, R.E., Poole, W.S.H., and Julien, A.F. 1987. A vesiculo-bullous disease in pigs resembling foot and mouth disease. II. Experimental reproduction of the lesion. N. Z. Vet. J. 35:27–30.

Morgan, M.R.A., and Coxon, D.T. 1987. Tolerances: glycoalkaloids in potatoes, p. 221–230. In: Watson, D. H. (ed.). Natural toxicants in food. Progress and prospects. Ellis Horwood, Chichester, UK.

Morrell, C.M., and Adams, J.A. 1993. Toxicity of verrucarin A to gametes and embryos of the purple sea urchin (*Arbacia punctulata*). Bull. Environ. Contam. Toxicol. 51:889–894.

Morse, W.J. 1914. Powdery scab of potatoes. Maine Agr. Exp. Sta. Bull. 227:89–104.

Nair, S.K., Peethambaran, C.K., Geetha, D., Nayar, K., and Wilson, K.I. 1990. Effect of soil solarization on nodulation, infection by mycorrhizal fungi and yield of cowpea. Plant Soil 125:153–154.

Neiger, R.D., Johnson, T.J., Hurley, D.J., Higgins, K.F., Rottinghaus, G.E., and Stahr, H. 1994. The short-term effect of low concentrations of dietary aflatoxin and T-2 toxin on mallard ducklings. Avian Dis. 38:738–743.

Nelson, E.B., and Hoitink, H.A.J. 1983. The role of microorganisms in the suppression of *Rhizoctonia solani* in container media amended with composted, hardwood bark. Phytopathology 73: 274–278.

Nelson, M.E., and Powelson, M.L. 1988. Biological control of gray mold of snap beans by *Trichoderma hamatum*. Plant Dis. 72:727–729.

Nelson, P.E., Plattner, R.D., Shackelford, D.D., and Desjardins, A.E. 1992. Fumonisin B_1 production by *Fusarium* species other than *F. moniliforme* in section *Liseola* and by some related species. Appl. Environ. Microbiol. 58:984–989.

Nigg, H.N., Strandberg, J.O., Beier, R.C., Petersen, H.D., and Harrison, J.M. 1997. Furanocoumarins in Florida celery varieties increased by fungicide treatment. J. Agr. Food. Chem. 45:1430–1436.

Nirenberg, H. 1976. Untersuchungen über die morphologische und biologische Differenzierung in der Fusarium-Section Liseola. Mitt. Biol. Bundesanst. Land-Forstwirtsch. Berlin-Dahlem 169: 1–117.

Obreza, T.A., and Reeder, R.K. 1994. Municipal solid waste compost use in tomato/watermelon successional cropping. Soil Crop Sci. Soc. Fla. Proc. 53:13–19.

Onyemelukwe, G.C., and Ogbadu, G. 1981. Aflatoxin levels in sera of healthy first time rural blood donors: preliminary report. Trans. Roy. Soc. Trop. Med. Hyg. 75:780–782.

Osman, A.R., and Saheb, A.F. 1983. Control of *Rhizoctonia solani* by soil solarization. Acta. Hort. 152:245–246.

Osweiller, G.D., Ross, P.F., Wilson, T.M., Nelson, P.E. Witte, S.T., Carson, T.L., Rice, L.G., and Nelson, H.A. 1992. Characterization of an epizootic of edema in swine associated with fumonisin in corn screenings. J. Vet. Diagn. Invest. 4:53–59.

Paharia, K.D. 1960. Charcoal rot of potatoes in India. Indian Potato J. 2:1–11.

Palti, J. 1981. Cultural practices and infectious crop diseases. Springer-Verlag, Berlin.

Phillips, A.J.L. 1990. The effects of soil solarization on sclerotial populations of *Sclerotinia sclerotiorum*. Plant Pathol. 39:38–43.

Pierson, C.F. 1966. Effects of temperature on the growth of *Rhizopus stolonifer* on peaches and on agar. Phytopathology 56:276–278.

Podilia, G.K., Rogers, L.M., and Kolatukkudy, P.E. 1993. Chemical signals from avocado surface wax trigger germination and appressorium formation in *Colletotrichum gloeosporioides*. Plant Physiol. 103:267–272.

Porter, I.J., and Merriman, L.R. 1983. Effects of solarization of soil on nematode and fungal pathogens at two sites in Victoria. Soil Biol. Biochem. 15:39–44.

Poswillo, D.E., Sopher, D., and Mitchell, S. 1972. Experimental induction of fetal malformation with "blighted" potatoes: A preliminary report. Nature (London). 239:462–464.

Prelusky, D.B. 1994. Residues in food products of animal origin, 405–419. In: Miller, J.D. and Trenholm, H.L. (eds.). Mycotoxins in grain: Compounds other than aflatoxin. Eagan Press, St. Paul, MN.

Prescott, L.M., Harley, J.P., and Klein, D.A. 1999. Microbiology. WCB McGraw-Hill, Boston.

Prusky, D. 1996. Pathogen quiescence in postharvest diseases. Annu. Rev. Phytopathol. 34:413–434.

Prusky, D., and Plumbley, R.A. 1992. Quiescent infections of *Colletotrichum* in tropical and subtropical fruits, 289–307. In: J.A. Bailey and M.J. Jeger (eds.). *Colletotrichum*: Biology, pathology and control. CAB International, Wallingford, UK.

Pullman, G.S., DeVay, J.E., Garber, R.H., and Weinhold, A.R. 1981. Soil solarization: effects on Verticillium wilt of cotton and soilborne populations of *Verticillium dahliae*, *Pythium* spp., *Rhizoctonia solani*, and *Thielaviopsis basicola*. Phytopathology 71:954–959.

Punja, Z.A., Carter, J.D., Campbell, G.M., and Rosselli, E.L. 1986. Effects of calcium and nitrogen fertilizers, fungicides and tillage practices on incidence of *Sclerotium rolfsii* on processing carrots. Plant Dis. 70:819–824.

Rabie, C.J., Van Rensburg, S.J., Van der Watt, J.J., and Lübben, A. 1975. Onyalai—The possible involvement of a mycotoxin produced by *Phoma sorghina* in the aetiology. S. Afr. Med. J. 49:1647–1650.

Raid, R.N., and Pennypacker, S.P. 1987. Weeds as hosts for Colletotrichum coccodes. Plant Dis. 71:643–646.

Raj, H., and Kapoor, I.J. 1993. Soil solarization for the control of tomato wilt pathogen (*Fusarium oxysporum* Schl.). Zeitschr. Pflanzenkrankh. Pflanzensch. 100:652–661.

Raj, S.K., Thakur, R.N., Singh, P., and Sastry, K.S.M. 1986. Tuber rot-a new disease of *Dioscorea composita* and its control. Curr. Sci. 55:411–412.

Renwick, J.H. 1972. Hypothesis: Anencephaly and spina bifida are usually preventable by avoidance of a specific but unidentified substance present in certain potato tubers. Br. J. Prev. Soc. Med. 26:67–88.

Rheeder, J.P., Marasas, W.F.O., Thiel, P.G., Sydenham, E.W., Shephard, G.S., and Van Schalkwyk, D.J. 1992. *Fusarium moniliforme* and fumonisins in corn in relation to human esophageal cancer in Transkei. Phytopathology 82:353–357.

Ricci, P., Bonnett, P., Huet, J.-C., Sallantin, M., Beauvais-Cante, F., Bruneteau, M., Billard, V., Michel, G., and Pernollet, J.-C. 1989. Structure and activity of proteins from pathogenic fungi *Phytophthora* eliciting necrosis and acquired resistance in tobacco. Eur. J. Biochem. 183:555–563.

Ristaino, J.B., Perry, K.B., and Lumsden, R.D. 1991. Effect of solarization and *Gliocladium virens* on sclerotia of *Sclerotium rolfsii*, soil microbiota, and the incidence of southern blight of tomato. Phytopathology 81:1117–1124.

Robinson, J.E., Browne, K.M., and Burton, W.G. 1975. Storage characteristics of some vegetables and soft fruits. Ann. Appl. Biol. 81:399–408.

Roe, N.E., Stoffella, P.J., and Bryan, H.H. 1993. Utilization of MSW compost and other organic mulches on commercial vegetable crops. Compost Sci. Util. 1:73–84.

Roy, A.K., and Chourasia, H.K. 1990. Mycotoxin incidence in root drugs. Int. J. Crude Drug Res. 28:157–160.

Ruff, M.D., Huff, W.E., and Wilkins, G.C. 1992. Characterization of the toxicity of the mycotoxins aflatoxin, ochratoxin, and T-2 toxin in game birds. III. Bobwhite and Japanese quail. Avian Dis. 36:34–39.

Runia W.TH. 1990. Ontsmetting van drainwater en gietwater met ozon: Betrouwbaar resultaat bij voldoende behandelingstijd. Weekblad Groenten en Fruit 9 November 1990:42–43.

Sanford, L.L., and Sinden, S.L. 1972. Inheritance of potato glycoalkaloids. Amer. Potato J. 49:209–217.

Sarantinos, J., Hung, T.V., Black, R.G., and Brouwer, J.B. 1996. The effects of fungal infection on the chemical and functional properties of chickpeas (*Cicer arietum*) and faba beans (*Vicia faba*). J. Sci. Food Agr. 70:197–203.

Sauviat, M.P., Laurent, D., Kohler, F., and Pellegrin, F. 1991. Fumonisin, a toxin from the fungus *Fusarium moniliforme* Sheld, blocks both the calcium current and the mechanical activity in frog atrial muscle. Toxicon Oxf. 29:1025–1031.

Schäfer, W. 1994. Molecular mechanisms of fungal pathogenicity to plants. Annu. Rev. Phytopathol. 32:461–477.

Schenck, N.C. 1987. Vesicular-arbuscular mycorrhizal fungi and the control of fungal root diseases, p. 179–191. In: Chet, I. (ed.). Innovative approaches to plant disease control. Wiley, New York.

Schneider, R.W. (ed.). 1982. Suppressive soils and plant disease. APS Press, St. Paul, MN.

Schneider, S., and Ullrich, W.R. 1994. Differential induction of resistance and enhanced enzyme activities in cucumber and tobacco caused by treatment with various abiotic and biotic inducers. Physiol. Mol. Plant Pathol. 45:291–304.

Scholte, K. 1992. Effect of crop rotation on the incidence of soil-borne fungal diseases of potato. Netherlands J. Plant Pathol. 98:93–101.

Schueler, C., Biala, J., and Vogtmann, H. 1989. Antiphytopathogenic properties of biogenic waste compost. Agr. Ecosyst. Environ. 27:477–482.

Schroth, M.N., and Hancock, J.G. 1982. Disease-suppressive soil and root-colonizing bacteria. Science 216:1376–1381.

Scott, P.M. 1977. *Penicillium* mycotoxins, p. 283–356. In: Wyllie, T.D. and Morehouse, L.G. (eds.). Mycotoxic fungi, mycotoxins, mycotoxicoses: An encyclopedic handbook. Vol. 1. Marcel Dekker, New York.

Scudamore, K.A. 1998. Mycotoxins, p. 147–181. In: Watson, D.H. (ed.). Natural toxicants in food. Sheffield Academic Press, Sheffield, U.K.

Semb, L. 1971. A rot of stored cabbage by *Phytophthora* sp. Acta Hort. 20:32–35.

Seo, J.A., Kim, J.C., and Lee, Y.W. 1996. Isolation and characterization of two new type C fumonisins produced by *Fusarium oxysporum*. J. Nat. Prod. 59:1003–1005.

Sherf, A.F., and MacNab, A.A. 1986. Vegetable diseases and their control. Wiley, New York.

Sherwood, R.T. 1966. Pectin lyase and polygalacturonase production by *Rhizoctonia solani* and other fungi. Phytopathology 56:279–286.

Simmonds, J.H. 1963. Studies in the latent phase of *Colletotrichum* species causing ripe rots of tropical fruits. Qd. J. Agr. Sci., 20:373.

Sivan, A., and Chet, I. 1993. Integrated control of *Fusarium* crown and root rot of tomato with *Trichoderma harzianum* in combination with methyl bromide or soil solarization. Crop Protect. 12:380–386.

Snowdon, A.L. 1992. Color atlas of post-harvest diseases and disorders of fruits and vegetables. Vol. 2. Vegetables. CRC Press, Boca Raton, FL.

Sommer, N.F. 1982. Postharvest handling practices and postharvest diseases of fruit. Plant Dis. 66: 357–364.

Stahl, D.J., and Schäfer, W. 1992. Cutinase is not required for fungal pathogenicity on pea. Plant Cell 4:621–629.

Stanghellini, M.E., Rasmussen, S.L., Kim, D.H., and Rorabaugh, P.A. 1996. Efficacy of nonionic surfactants in the control of zoospore spread of *Pythium aphanidermatum* in a recirculating hydroponic system. Plant Dis. 80:422–428.

Stanghellini, M.E., Stowell, L.J., and Bates, M.L. 1984. Control of root rot of spinach caused by *Pythium aphanidermatum* in a recirculating hydroponic system by ultraviolet irradiation. Plant Dis. 68:1075–1076.

Stapleton, J.J., and DeVay, J.E. 1986. Soil solarization: A non-chemical approach for management of plant pathogens and pests. Crop Protect. 5:190–198.

Stapleton, J.J., Quick, J., and DeVay, J.E. 1985. Soil solarization: effects on soil properties, crop fertilization and plant growth. Soil Biol. Biochem. 17:369–373.

Steadman, J.R., Coyne, D.P., and Cook, G.E. 1973. Reduction of severity of white mold disease on Great Northern beans by wider row spacing and determinate plant growth habit. Plant Dis. Rep. 57:1070–1071.

Steyn, P.S., and Rabie, C.J. 1976. Characterization of magnesium and calcium tenuazoate from *Phoma sorghina*. Phytochemistry 15:1977–1979.

Stindt, A., and Weltzien, H.C. 1990. Untersuchungen zur Wirkung und zu den Wirkungsmechanismen von Kompost Extrackten auf *Botrytis cinerea* Pers. Ex. Nocca & Balb an Erdbeeren, Kopfsalat und Buschbohnen. Erwerbstobstbau 33:28–29.

Strashnov, Y., Elad, Y., Sivan, A., Rudich, Y., and Chet, I. 1885. Control of *Rhizoctonia solani* fruit rot of tomatoes by *Trichoderma harzianum* Rifai. Crop Protection 4:359–364.

Stroember, A., and Brishammer, S. 1991. Induction of systemic resistance in potato (*Solanum tuberosum* L.) plants to late blight by local treatment with *Phytophthora infestans* (Mont.) de Bary, *Phytophthora cryptogaea* Pathyb, Laff. or dipotassium phosphate. Potato Res. 34:219–225.

Sumner, D.R., Doupnik, B. Jr., and Boosalis, M.G. 1981. Effect of reduced tillage and multiple cropping on plant diseases. Annu. Rev. Phytopathol. 19:167–187.

Swinburne, T.R. 1983. Quiescent infections in post-harvest diseases, p. 1–21. In: C. Dennis (ed.). Post-harvest pathology of fruits and vegetables. Academic Press, London.

Swinburne, T.R. 1993. Quiescent infections in post-harvest diseases. p. 1–20. In: Dennis, C. (ed.). Postharvest pathology of fruits and vegetables. Academic Press, London.

Sydenham, E.W., Shephard, G.S., Stockenstrom, S., Rheeder, J.P., Marasas, W.F.O., and Van der Merwe, M.J. 1997. Production of fumonisin B analogues and related compounds by *Fusarium globosum*, a newly described species from corn. J. Agr. Food Chem. 45:4004–4010.

Theron, D.J. 1999. Fusarium dry rot of potatoes: etiology, epidemiology, toxicity and control. PhD thesis. University of the Orange Free State, Bloemfontein, South Africa.

Thiel, P.G., Marasas, W.F.O., Sydenham, E.W., Shephard, G.S., Gelderblom, W.C.A., and Nieuwenhuis, J.J. 1991. Survey of fumonisin production by *Fusarium* species. Appl. Environ. Microbiol. 57:1089–1093.

Thirumalachar, M.J., and O'Brien, M.J. 1977. Suppression of charcoal rot in potato with a bacteria antagonist. Plant Disease Reptr. 61:543–546.

Thorne, S.M. 1972. Studies on the behavior of stored carrots with respect to their invasion by *Rhizopus stolonifer* Lind. J. Food Tech. 7:139–151.

Tomkins, R.G. 1963. The effects of temperature, extent of evaporation and restriction of ventilation on the storage life of tomatoes. J. Hort. Sci. 38:335–347.

Towers, N.R., and Sprosen, J.M. 1992. *Fusarium* mycotoxins in pastoral farming: zearalenone induced infertility in ewes, p. 272–284. In: Gopalakrishnakone, P. and Tan, C.K. (eds.). Recent advances in toxicology research. Vol 1. National University of Singapore, Singapore.

Trail, F., and Koller, W. 1990. Diversity of cutinases from plant pathogenic fungi: Evidence for a relationship between enzyme properties and tissue specificity. Physiol. Mol. Plant Pathol. 36: 495–508.

Tränkner, A. 1992. Use of agricultural and municipal organic wastes to develop suppressiveness to plant pathogens, p. 35–42. In: Tjamos, E.C., Papavizas, G.C., and Cook, R.J. (eds.). Biological control of plant diseases. Plenum Press, New York.

Trumble, J.T., Diawara, M.M., Millar, J.G., Ott, D.E., and Carson, W.C. 1994. Implications of changing patterns of linear furanocoumarins in celery with plant growth, pesticide applications, and insect feeding. Acta Hort. 381:569–599.

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

Van Peer, R., Niemann, G.J., and Schippers, B. 1991. Induced resistance and phytoalexin accumulation in biological control of Fusarium wilt of carnation by *Pseudomonas* sp. strain WCS417r. Phytopathology 81:728–734.

Van Steekelenburg, N.A.M. 1982. Factors influencing external fruit rot of cucumbers caused by *Didymella bryoniae*. Neth. J. Plant Pathol. 88:47–56.

Velvis, H., and Jager, G. 1983. Suppression of *Rhizoctonia solani* in potato fields. II. Effect of origin and degree of infection with *Rhizoctonia solani* of seed potatoes on subsequent infestations and on formation of sclerotia. Netherlands J. Plant Pathol. 89:141.

Verhoeff, K. 1974. Latent infections by fungi. Annu. Rev. Phytopathol. 12:99–100.

Verhoeff, K. and Warren, J.M. 1972. In vivo and in vitro production of cell wall degrading enzymes by *Botrytis cinerea* from tomato. Netherlands J. Plant Pathol. 78:179–185.

Walker-Simmons, M., Hadwiger, L.A. and Ryan, C.A. 1983. Chitosan and pectic polysaccharides both induce the accumulation of the antifungal phytoalexin pisatin in pea pods and antinutrient proteinase inhibitors in tomato leaves. Biochem. Biophys. Res. Commun. 110:194–199.

Wanger, H. 1985. Plant drug analysis. Springer-Verlag, Berlin.

Wei, G., Kloepper, J.W., and Tuzun, S. 1991. Induction of systemic resistance of cucumber to *Colletotrichum orbicularae* by select strains of plant-growth-promoting rhizobacteria. Phytopathology 81:1508–1512.

Welch, A.W., Jenkins, S.F., and Averre, C.W. 1975. *Trichothecium* fruit rot on greenhouse tomatoes in North Carolina. Plant Dis. Rep. 59:255–257.

Weltzien, H.C. 1992. Biocontrol of foliar fungal diseases with compost extracts, p. 430–450. In: Andrews, J.H. and Hirano, S.S. (eds.). Microbial ecology of leaves. Springer-Verlag, New York.

White, N.H. 1954. Decoy crops. The use of decoy crops in the eradication of certain soil-borne plant diseases. Aust. J. Sci. 17:18–19.

Whittaker, R.H. 1969. New concepts of kingdoms of organisms. Science 163:150–160.

Widmer, T.L., Graham, J.H., and Mitchell, D.J. 1998. Composted municipal waste reduces infection of citrus seedlings by *Phytophthora nicotianae*. Plant Dis. 82:683–688.

Wilson, T.M., Ross, P.F., Rice, L.G., Osweiller, G.D., Nelson, H.A., Owens, D.L., Plattner, R.D., Reggiardo, C., Noon, T.H., and Pickrell, J.W. 1990. Fumonisin B_1 levels associated with an epizootic of equine leucoencephalomalacia. J. Vet. Diagn. Invest. 2:213–216.

Winter, W., and Winiger, F.A. 1983. Einfluss verschiedener Fangpflanzen sowie von Kalk und Kalkstickstoff auf die Bodenverseuchung mit *Spongospora subterranea*, dem Erreger des Pulverschorfes bei Kartoffeln. Mitt. Schweizer. Landwirtsch. 31:190–206.

Wu, C.M., Koehler, P.E., and Ayres, J.C. 1972. Isolation and identification of xanthotoxin (8-methoxypsoralen) and bergapten (5-methoxypsoralen) from celery infected with *Sclerotinia sclerotiorum*. Appl. Microbiol. 23:852–856.

Wyllie, T.D., and Morehouse. L.G. (eds). 1977/8. Mycotoxic fungi, mycotoxins, mycotoxicoses: An encyclopedic handbook. Vols. 1–3. Marcel Dekker, New York.

Yoder, O.C., and Whalen, M.L. 1975. Factors affecting post-harvest infection of stored cabbage tissue by *Botrytis cinerea*. Can. J. Bot. 53:691–699.

Yohalem, D.S., Harris, R.F., and Andrews, J.H. 1994. Aqueous extracts of spent mushroom substrate for foliar disease control. Compost Sci. Util. 2:67–74.

Yoshizawa, T., Yamashita, A., and Luo, Y. 1994. Fumonisin occurrence in corn from high and low risk areas of human esophageal cancer in China. Appl. Environ. Microbiol. 60:1626–1629.

Yücel, S. 1995. A study on soil solarization and combined with fumigant application to control

Phytophthora crown blight (*Phytophthora capsici* Leonian) on peppers in the East Mediterranean region of Turkey. Crop Protect. 14:653–655.

Zhang, W., Hoitink, H.A.J., and Dick, W.A. 1996. Compost-induced systemic acquired resistance in cucumber to Pythium root rot and anthracnose. Phytopathology 86:1066–1070.

Zhang, W., Han, D.Y., Dick, W.A., Davis, K.R., and Hoitink, H.A.J. 1998. Compost and compost water extract-induced acquired resistance in cucumber and Arabidopsis. Phytopathology 88: 450–455.

Zitter, T.A., Hopkins, D.L., and Thomas, C.E., eds. 1996. Compendium of cucurbit diseases. APS Press, St. Paul, MN.

Ziv, O., and Zitter, T.A. 1992. Effects of bicarbonates and film-forming polymers on cucurbit foliar diseases. Plant Dis. 76:513–517.