**Fungi of Grapes**

**Hanns-Heinz Kassemeyer and Beate Berkelmann-Löhnertz**

**4.1 Introduction**

Grapevine can be attacked by a number of fungi and fungus-like organisms which

affect the berries and cause loss of quality and influence the taste of the wine. Due

to attack of the grapevine by pathogens’ the infected plant tissue is destroyed and

necrotization occurs. When large areas of the canopy are affected by grapevine diseases,

the assimilation capacity of the vine is reduced and as a result the berry

quality decreases. Aside from leaves, most grapevine pathogens also infect inflorescences,

clusters and berries so that the yield can be reduced. Berry infections

result in decay of fruit tissue, however specific effects on berry quality depend on

the ripening stage at which the infection occurs. Some pathogens directly destroy

the fruit tissue enzymtically; others impede ripening, and a number of fungi produce

off flavours or myctoxins. Grapevine diseases can spread rapidly under favourable

conditions and cause more or less severe epidemics. To avoid loss of quality and

yield, the pathogens have to be controlled by appropriated culture techniques and

targeted application of fungicides. Besides the pathogenic fungi causing grapevine

diseases, berries are also colonized by ubiquitous epiphytic fungi which use sugar

and amino acids leaking out of berries as nutrient source. In general grapevine

pathogens can be sub-divided into main pathogens of high economical importance

which are pre-dominant, like downy mildew (*Plasmopara viticola*), powdery mildew

(*Erysiphe necator*) and bunch rot (*Botrytis cinerea*) and those which occur only

locally or temporary. Moreover other important grapevine diseases are caused by

wood decaying fungi which pre-dominantly attack the trunk and canes (Fischer and

Kassemeyer 2003). In the present chapter such fungi and oomycetes are regarded

which colonize grapevine berries and consequently may influence the must and

wine. All fungi reported to colonize grapevine berries are listed in Table 4.1, however

some of them can be regarded as harmless epiphytes, others actually as antagonists

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62 H.-H. Kassemeyer

**Table 4.1** Fungi taxa detected on grapevine clusters and berries (Hall and Emmett 2001; Serra et

al. 2005; Serra et al. 2006; Uyovbisere et al. 2007; Whitelaw-Weckert et al. 2007); the taxonomy of

the Ascomycetes is according to Gams et al. 1998 and Mc Laughlin et al. 2001

Kindom Class Subclass Order Genus Species

Chromista Peronosporo

mycetes

Peronosporales *Plasmopara P. viticola*

(Berk. & Curt.)

Berl. & De

Toni

Mycota Zygomycetes Mucorales *Cunninghamella C. spec*. Matr.

*Mucor M. mucedo*

Fresen

*M. hiemalis*

Wehmer

*M. piriformis*

A. Fisch

*Rhizopus R. stolonifer*

(Ehrenb.)

Lind.

*Syncephalastrum S. spec*. J. Schröt.

Ascomycetes Plecto

mycetes

Eurotiales *Aspergillus A. aculeatus*

Iizuka

*A. alliaceus*

Thumb &

Church

*A. auricomus*

Saito

*A. candidus* Link

*A. carbonarius*

Bainier

*“A. ibericus”*

*A. carneus*

Blochwitz

*A. clavatus*

Desm.

*A. flavipes* Thom

& Church

*A. flavus* Link

*A. fumigatus*

Fresen

*A. japonicus*

Saito

*A.niger* aggregate

Tiegh.

*A. ochreaceus*

G. Wilh.

*A. ostinianus*

Wehmer

*A. terreus* Thom

*A. ustus* Thom &

Church

(continued)

4 Fungi of Grapes 63

**Table 4.1** (continued)

Kindom Class Subclass Order Genus Species

*A. versicolour*

Tirab.

*A. wentii* Wehmer

*Emericella E. spec*. Berk.

*Eurotium E. amstelodami*

L. Mangin

*E. chevalieri* L.

Mangin

*Paecilomyces Paecilomyces*

*variotii* Bain

*Penicillium P. aurantiogriseum*

Dierckx

*P. bilaiae*

Chalabuda

*P. brevicompactum*

Dierckx

*P. canescens*

Sopp

*P. chrysogenum*

Thom

*P. citrinum* Thom

*P. corylophilium*

Dierckx

*P. crustosum*

Thom

*P. echinulatum*

Fassatiova

*P. expansum* Link

*P. fellutanum*

Biourge

*P. funiculosum*

Thom

*P. glabrum/spinulosum*

*P. griseofulvum*

Dierckx

*P. implicatum*

Biourge

*P. janczeweskii*

K.M.

Zalessky

*P. miczynskii*

Zelaski

*P. minioluteum*

Dierckx

*P. novae-zeelandiae*

J.F.M.

Beyma

(continued)

64 H.-H. Kassemeyer

**Table 4.1** (continued)

Kindom Class Subclass Order Genus Species

*P. olsonii* Bainier

& Sartory

*P. oxalicum*

Currie &

Thom

*P. pinopilum*

Hedgcock

*P. purpurogenum*

Stoll

*P. raistrickii*

G. Sm.

*P. restrictum* J.C.

Gilman &

E.V. Abott

*P. roquefortii*

Thom

*P. rugulosum*

Thom

*P. sclerotiorum*

van Beyma

*P. simplicissimum*

Thom

*P. solitum*

Westling

*P. thomii* Maire

*P. variabile* Sopp

*P. verruculosum*

Peyrone*l*

*P. waksmannii*

Zalski

Onygenales *Histoplasma H. spec*. Darling

Pyreno

mycetes

Hypocreales *Acremonium*

*Beauveria*

*Fusarium*

*Gliocladium*

*Trichoderma*

*Trichoth ecium*

*A. spec*. Link

*B. spec*. Vuill.

*F. spec*. Link

*G. spec*. Corda

*T. spec*. Pers

*T. roseum* (Pers.)

Link

Diaporthales *Coniella C. petrakii*

B.Sutton

*Phomopsis Ph. viticola*

(Sacc.) Sacc

Sordariales *Arthrinium A. spec*.Kunze

*Chaetomium Ch. spec*. Kunze

*Chrysonilia C. spec*. Arx

*Neurospora N. spec*. Shear &

Dodge

(continued)

4 Fungi of Grapes 65

**Table 4.1** (continued)

Kindom Class Subclass Order Genus Species

Xylariales *Pestaltiopsis P. spec*. Steyeart

*Truncatella T. spec*. Steyeart

Erysiphales *Erysiphe Erysiphe necator*

Schwein.

Loculoasco

mycetes

Dothideales *Aureobasidium A. spec*. Viala &

Boyer

*Guignardia G. bidwellii*

(Ellis) Viala

& Ravaz

Pleosporales *Alternaria A. alternata* (Fr.:

Fr.) Keissler

*Curvularia C. spec*. Boedijn

*Drechslera D. spec*. S. Ito

*Epicoccum E. spec*. Link

*Periconia P. spec*. Tode ex

Fr.

*Phoma Ph. spec*. Sacc.

*Pithomyces P. spec*. Ellis

*Stemphylium S. spec*. Wallr.

*Ulocladium U. atrum* Pers.

Capnodiales *Cladosporium C. herbarum*

(Pers.) Link

Myrangiales *Elsinoë E. ampelina*

Shear

Disco

mycetes

Helotiales *Botrytis B. cinerea* Pers.:

Fr.

*Pseudopezicula P. tracheiphila*

(Müll.-

Thurg.) Korf

& Zhuang

**Table 4.2** Taxa producing mycotoxins considered

relevant for human health (Serra et al. 2005)

Taxon Mycotoxin

*Aspergillus ochraceus* Ochratoxins

*Aspergillus alliaceus*

*Aspergillus niger aggregate*

*Aspergillus carbonarius*

*Penicillium verucosum*

*Trichothecium roseum* Trichothecene

*Penicillium expansum* Patulin

of pathogenic fungi, e.g. *Trichoderma* and *Ulocladium* (Schoene and Köhl 1999; Li

et al. 2003). Some of the fungi produce mycotoxins (Table 4.2) which are more or

less human-toxic and some may release compounds which are toxic to yeasts. In

addition numerous fungi colonizing the berry surface during different stage of berry

development and ripening have been identified (Table 4.3).

66 H.-H. Kassemeyer

**Table 4.3** Taxa identified on grapes in Portugal at different phaenological stages

(Serra et al. 2005)

Taxon

Phenologial stage of the berry colonization

Pea size Veraison Harvest

*Acremoniella* Sacc. X X X

*Acremonium* Link X X X

*Arthrinium* Kunze. X X 0

*Aspergillus* Fr.:Fr. X X X

*Aureobasidium* Viala & Boyer X X X

*Beauveria* Vuill. X 0 0

*Chaetomium* Kunze X X 0

*Chrysonilia* Arx 0 X X

*Cunninghamella* Matr. X X X

*Curvularia* Boedijn X X X

*Drechslera* S. Ito X X X

*Emericella* Berk. X X X

*Epicoccum* Link X X X

*Eurotium* Link: Fr. X X X

*Fusarium* Link X X X

*Geotrichum* Link: Fr. X 0 0

*Gliocladium* Corda X X X

*Histoplasma* Darling X 0 0

*Neurospora* Shear & Dodge 0 X X

*Nigrospora* Zimm. 0 X X

*Periconia* Tode ex Fr. X 0 0

*Pestalotiopsis* Steyeart X X 0

*Phoma* Sacc. X X X

*Pithomyces* Ellis X X X

*Rhizopus* Ehrenb. X X X

*Scytalidium* Pesante X X 0

*Sremphylium* Wallr. X X X

*Syncephalastrum* J. Schröt. X 0 X

*Trichoderma* Pers. X X X

*Truncatella* Steyeart X 0 0

*Ulocladium* Preuss X X X

**4.2 Peronosporomycetes**

***4.2.1 Plasmopara viticola* (Berk. & Curt.) Berl.**

**& De Toni: Grapevine Downy Mildew**

*General aspects*. Downy mildew is the most serious disease of grapevine, particularly

in warm and humid climates. The pathogen is indigenous on wild grapevine

species, e.g. *Vitis aestivalis* in the south-east of USA. The European cultivars of

*Vitis vinifera* first came in contact with this pathogen roughly around 1878 where

4 Fungi of Grapes 67

first symptoms were found in the Bordeaux region. Due to high susceptibility of

European cultivars, grapevine downy mildews spread within a few years and caused

a pandemic in the viticultural regions of the whole of Europe. To date grape downy

mildew occurs in all viticultural regions that are warm and wet during the vegetative

growth of vine (e.g. Europe, Eastern part of North America, New Zealand,

China, and Japan). The absence of rainfall in spring and summer limits the spread

of the disease in certain areas (e.g. Australia, California, and Chile).

The disease affects all green parts of the vine, particularly leaves, inflorescences

and young berries. Depending on grape cultivar and leaf age, lesions are yellowish

and oily. After a damp night, oilspots and sporulation occurs on the lower leaf surface

visible as dense, white patches. Later on the sporulation sites become necrotic

and severely infected leaves generally drop. Such defoliation reduces sugar accumulation

in berries and decreases frost hardiness of shoots and over-wintering buds.

Inflorescences and clusters with young berries are highly susceptible which finally

turn brown, dry up, and drop. Although berries become less susceptible as they

mature, infection of the rachis can spread into older berries which turn into a dry

brown rot, without sporulation.

*Taxonomy*. The causal agent of grapevine downy mildew, *Plasmopara viticola*

(Berk. & Curt.) Berl. & De Toni, belongs to the Oomycetes, and according to current

taxonomy is a member of the family of Peronsporomycetes (Dick 2002). This

family is different from the kingdom of true fungi (Mycota) and is a part of

Chromista, a kingdom which comprises heterogeneous microorganisms among

others the autotrophic Chrysophyceae (golden algae) and Bacillariophyceae (diatoms).

Like all Chromista the cell wall of *P. viticola* consists of glucanes and

biflagellated zoospores are formed. Within the family of Peronosporomycetes

*P. viticola* shows some primary characteristic of Chromista; among others, parts of

the life cycle of the organism are bound to water. Like *P. viticola*, majority of

Peronosporomycetes are plant pathogens such as the causal agent of potato late

blight, *Phytophthora infestans*.

*Biology and Epidemiology. P. viticola* is a biotrophic pathogen strongly adapted

to members of the genus *Vitis*. It develops in the inter cellular space within the colonized

tissues of the vine in the form of tubular, coenocytic hyphae, developing

globular haustoria. The haustoria penetrate the cell wall and invaginate the outer

membrane to take nutrients from the host cell. Asexual reproduction occurs by formation

of lemon shaped sporangia formed on branched sporangiophores during

humid nights. Each sporangium gives rise to four to ten biflagellate zoospores

which are released as soon as the sporangium is incubated in water (Kiefer et al.

2002). Asexuell developed zoospores as well vegetative hyphae are diploid.

Sexual reproduction begins in the summer by developing of gametangia. In the

male antheridium as well as in the female oogonium, meiosis runs and the haploid

nucleus of the antheridium fuse with that of the oogonium forming a diploid

oospore. *P. viticola* is heterothallic and therefore fertilization occurs only between

two different mating types. The thick walled oospore over-winter in fallen leaves,

becomes mature in spring and germinates in free water forming a primary sporangium*,*

which produces 30–60 zoospores. Germination occurs during the vegetation

68 H.-H. Kassemeyer

period from spring to midsummer as soon as temperatures reach 10°C and rainfall

ensures required wetness (Hill 1989). From the primary sporangium the zoospores

are dispersed during intensive rainfall.

The released zoospores both from oospores and asexual sporangia swim within

a water film covering the surface of the host plant after precipitation and dew, and

attach around the stomata. They shed their flagella and encyst forming a cell wall

(Riemann et al. 2002). Subsequently an infection tube emerges from each encysted

spore (Fig. 4.1) which penetrates the stoma and forms a sub-stomatal vesicle in the

sub-stomatal cavity where it dilates into a primary hyphae (Kiefer et al. 2002).

Under optimal conditions, the period from the release of zoospores to penetration

is less than 90 min. From the sub-stomatal vesicle a hyphae grows in the intercellular

space of the host tissue. The hyphae branch and form a mycelium that colonizes

the host tissue (Unger et al. 2007). The period from infection to first

appearance of oilspots–the incubation period- depends on temperature and humidity.

In general sporulation takes place at the end of the incubation period, in the

first night when conditions for sporulation mentioned above occur (Rumbolz et al.

2002). Under favourable conditions, incubation period is very short and *P. viticola* is

able to sporulate three to four days after infection. As soon as the host tissue is

totally colonized by the mycelium of *P. viticola,* sporulation takes place.

Sporulation requires 95–100% relative humidity and at least 4 h of darkness at

temperatures >12.5°C; the optimal temperature for sporulation is 18–22°C and

therefore an outbreak of the disease, visible in the morning after a warm and damp

night yields maximum sporangia. Induction of sporulation is influenced by the

photoperiod and sporangiophores and sporangia differentiate only during the night

(Rumbolz et al. 2002). At the beginning of sporulation a secondary sub-stomatal

**Fig. 4.1** Encysted zoospores with a penetration peg from *Plasmopara viticola* attached at a

stoma; Low-Temperature-Scanning-Electron-Microscopy (Kassemeyer H.-H. and Düggelin M.,

University of Basel)

4 Fungi of Grapes 69

vesicle is formed from which hyphae grow out of the stoma. The emerging hyphae,

branch and form typical sporangiophores (Fig. 4.2). Finally, sporangia develop at

the tips of the branches and around seven hours after the beginning of sporulation,

mature sporangia are present. Immediately after formation, sporangia are detached

from sporangiophores and spread by wind. Successful infection conditions can be

calculated using the relation between temperature and duration of leaf wetness

(Huber et al. 2003).

**4.3 Ascomycetes**

***4.3.1 Erysiphe necator Schwein.* (emend. Uncinula necator**

**(Schw.) Burr) ( Erysiphales): Grapevine Powdery Mildew**

*General Aspects*. Grapevine powdery mildew occurs worldwide in all viticultural

regions and causes severe losses of yield and quality especially in warm and dry

weather conditions. This grapevine disease was introduced from North America

and detected first in Europe in the middle of the 19th century. The disease spread

within a short time in Europe and gave rise to economically relevant epidemics.

After bud burst, first symptoms are visible as white or grey powdery patches on

leaves and shoot tips between the three- and six-leaf stages on leaves. These “ flagshoots”

strike on susceptible cultivars such as Chardonnay, Cabernet Sauvignon,

Carignane, Portugieser, Vernatsch (Trollinger); they occur more or less pronounced

also on the vast majority of European cultivars. Young leaves, inflorescences and

young berries are highly susceptible immediately after the bloom; however older

**Fig. 4.2** Sporangiophore with sporangia from *Plasmopara viticola*; Low-Temperature-Scanning-

Electron-Microscopy (Kassemeyer H.-H., Boso S. and Düggelin M., University of Basel)

70 H.-H. Kassemeyer

leaves and berries up to the veraison are also infected (Ficke et al. 2002). Young

leaves and berries can be totally covered with white powdery patches, whereas

on older leaves small colonies occur on the upper leaf side. Infected leaves

remain green over a longer period but the assimilation efficiency of the leaves is

reduced. Shoot tips, inflorescences and young clusters are also covered with

whitish or greyish patches. Infected inflorescences become curled and necrotize.

Shoots become stunted and leaves appear yellowed. As a result of impeded

growth of the berry skin on infected berries from the pea-size stage, cracking and

splitting occurs. The splits are entrance ports for secondary invaders such as acetate

acid producing yeasts and bacteria (Fig. 4.3). Fully expanded berries can be colonized

by the pathogen up to the beginning of veraison. Berries with these late

infections ripen but the mycelium of powdery mildew can affect wine quality by

its mouldy taste. Additionally the pathogen penetrates the berry skin and facilitates

infections by bunch rot.

*Taxonomy*. The agent causing grapevine powdery mildew, *Erysiphe necator*

Schwein. (emend. *Uncinula necator* (Schw.) Burr) is an Ascomycete belonging to the

Erysiphales which comprise a broad range of plant pathogens (Bélanger et al. 2002).

In all Ascomycetes, the cell wall of *E. necator* consists of chitin, a polymere of

N-acetylglucosamine.

*Biology and Epidemiology***.** Like all powdery mildew fungi, *E. necator* is a

biotrophic fungus with limited spectrum of host plants, infecting only grapevine

(*Vitis*) species. The fungus grows epiphytically on the epidermis of green plant tissue

forming a dense white mycelium. *E. necator* over-winters as hyphae hidden

in the buds, or as ascospores in fruit bodies (Rügner et al. 2002; Rumbolz and

Gubler 2005). Both over-wintered hyphae and ascospores act as primary inoculum.

During the formation of winter buds in spring, hyphae colonize the inner bud

scales and remain dormant up to the following spring. After bud burst, over-wintered

hyphae colonize young leaves and shoots forming more or less striking

**Fig. 4.3** Berry infection by *Erysiphe necator* with splitting of the berry skin

4 Fungi of Grapes 71

“flagshoots”. The powdery cover of this “flagshoots” pre-dominatly consists of

conidiophores with chains of conidia (Pearson and Goheen 1988; Agrios 1997).

The ascospores are formed after karyogamie in an ascogenic hyphae, during dry

and warm weather in late summer and autumn. *E. necator* is heterothallic and,

two different mating types have to combine for sexual reproduction. The

ascospores are located in asci which are embedded in cleistothecia. These possess

hooked appendices responsible for the attachment of the fruit bodies at the bark

of canes and trunks. In the spring during rainfall, the cleistothecia open and by

mean of a special mechanism the ascospores are ejected out of the asci. Ascspores

as well as conidia attach actively on the surface of host plants and germinate

under optimal temperatures between 20 and 27°C within 4 h (Rumbolz et al. 2000).

No water is necessary for germination, but higher humidity favours this process. The

germ tube forms an apressorium which strengthens the attachment of the pathogen on

the host epidermis (Fig. 4.4). Beneath the apressorium, a penetration peg penetrates

encymatically the cuticle and epidermis cell wall (Rumbolz et al. 2000). At the tip

of the penetration peg, a lobed haustorium is formed which invaginates the epidermis

cell and deprive nutrients from the host. As soon as nutrient uptake is

ensured, a second hyphae emerges from the conidia and colonization of the host

surface commences. Temperatures ranging from 18 to 28°C promote hyphae

growth and mycelium formation. Within five to six days after infection, conidiophores

are formed projecting at a right angle from the host surface. From a basal

cell in the conidiophore, conidia develop and are cut off permanently. Conidia are

adapted to transport by wind and spread over long distances. High temperatures

and humid nights are favourable for the production of high amount of conidia. In

most cases, the epidemic starts in spring from ascospore infection or “flagshoots”

**Fig. 4.4** Germinated conidia from *Erysiphe necator* on the surface of a grapevine leaf; Low-

Temperature-Scanning-Electron-Microscopy (Rumbolz, J., Kassemeyer H.-H., Düggelin M. and

R. Guggenheim, University of Basel)

72 H.-H. Kassemeyer

when three to six leaves are unfolded. Under warm and dry conditions disease

incidence and severity increase up to berry set, due to high susceptibility of

young leaves, inflorescences and young berries (Ficke et al. 2002).

***4.3.2 Botrytis cinerea* Pers.:Fr. ( Helotiales): Botrytis Bunch Rot**

*General Aspects. Botrytis cinerea* is a plant pathogen of economical importance

causing rot in a broad range of crops, fruits and ornamental plants. In viticulture

*B. cinerea* may cause both serious loss and enhancement of quality. Injury and

profit, respectively, depends not only on the stage of ripening in which berries are

infected but also on weather conditions. Under dry and warm conditions infections

of ripe berries may raise the quality especially of white cultivars. In this case berry

ingredients are concentrated due to the perforation of the berry skin by the fungus.

In addition *B. cinerea* produces gluconic acid which confers a pronounced tastiness

to the wine. Consequently late infections of mature berries facilitate the production

of dessert wines like “Trockenbeerenauslesen”, “Sauternes” and “Tokay”. On the

other hand, berry infection at an early stage of ripening and during long lasting

wetness of the clusters reduces the quality due to berry decay. Infestation of clusters

with berry moth enhances bunch rot because the feedings sites of the larvae on berries

set entrance ports for *B. cinerea*. At the beginning of infection by *B, cinerea*

berries from white varieties become light-coloured from pinkish to light brown;

those from red variety changes from red to purple. Later on a light grey mycelium

occurs on the surface (Fig. 4.5), and in a proceeded infection stage berries become

brownish and rotten. On infected berries *B. cinerea* produces high amounts of laccases

which oxidase the anthocyanes and flavonoids to brown oxidation products.

Laccases are very stable and can pass over in must and wine and as a result, wine

becomes brownish and red wines especially lose their characteristic red colour.

**Fig. 4.5** Bunch rot caused by *Botrytis cinerea*; conidiophore emerge from pores and cracks in the

rotten berry skin and form a grey pad

4 Fungi of Grapes 73

*Taxonomy*. The teleomorph of *Botrytis cinerea* Pers.:Fr., *Botryotinia fuckeliana* (de

Bary) Whetzel is a member of the Heliotales (Ascomycetes). *B. cinerea* occurs mainly

in its anamorph form, whereas teleomorph *B. fuckeliana* is very rare (Gams et al. 1998;

Elad et al. 2004).

*Biology and Epidemiology. B. cinerea* is an ubiquitous fungus and has a broad

range of host plants. The fungus can live saprophytically on organic debris and

produce sclerotia as long-term survival form. *B. cinerea* over-winters both as

mycelium and as sclerotium on canes and leaf litter on the ground. The conidia

produced on sclerotia during periods with raising temperatures in the early spring

are considered the main source of primary inoculum. Conidia are short-lived

propagules during the season, and are spread by wind, rain and also insects. On

the host plant surface, the conidia germinate 1–3 h after inoculation forming various

penetration structures. In the presence of sugar the germ tubes of *B. cinerea*

forms a multilobed appressorium (Elad et al. 2004). To penetrate the host tissue

*B. cinerea* prefers wounds and natural openings, e.g. specialised structures of

flowers on which sugar and other nutrient are available (Keller et al. 2003; Viret

et al. 2004). When spores germinate on floral tissue of inflorescences or later in

the season on ripening berries, *B. cinerea* can change from saprophytic to necrotrophic

life style. The fungus expresses a set of enzymes such as lipases, cutinases

and pectinases that enables the pathogen to penetrate the epidermis of the

host tissue. The penetration of the host cuticle by *B. cinerea* mediated by cuteolytic

enzymes triggers a programmed cell death in the epidermis and the underlying

cells before they are invaded by hyphae. Effector proteins of *B. cinerea* acting

as pathogenicity factors and the induction of the programmed cell death facilitate

invasion and are essential for successful infection. So the pathogen is able to

complete its disease and life cycle (Elad et al. 2004). Flowers are susceptible to

infection because the receptacle constitutes natural openings and provides sugar

that facilitate flower colonization by the pathogen (Keller et al. 2003; Viret et al.

2004). Increasing susceptibility of ripening berries relies on several factors:

(1) host defence, e.g. expression of stilbenes, weakens with ongoing ripening,

(2) amount of fungistatic protoanthocyanidins reduces after veraison, (3) structure

of the cuticle and epidermis changes with advanced seed maturation and

micro cracks occur which allow the leakage of sugars (Kretschmer et al. 2007).

Conidia germination, germ tube growth, penetration and colonization of the host

tissue are crucial processes of the infection cycle. Conidia germination and infections

occur under high humidity (> 94% relative humidity) even on dry berries;

however long wetness period favours development of *B. cinerea* and increases

disease incidence. At 20–24°C and humid conditions, a germ tube arises within

four to eight hours and under this condition hyphae grow up to 4 mm per day (Fig. 4.6).

After penetration of the host tissue hyphae grow and after branching, a dense grey

mycelium is formed in which conidiophores with conidia develop (Person and

Goheen 1988; Agrios 1997). Conidia germination and growth of mycelium and

conidia formation also occur at lower temperatures up to 5°C; however infection

and development of the pathogen is delayed. Epidemics with severe infections and

high disease incidence arise under continuing rainfall after veraison.

74 H.-H. Kassemeyer

***4.3.3 Pseudopezicula tracheiphila* (Müll.- Thurg.)**

**Korf & Zhuang ( Helotiales): Rotbrenner**

*General Aspects*. Rotbrenner is locally confined and occurs primarily in warm vineyards

with stony soil. In some areas the disease results in severe losses annually, whereas in

others it occurs only sporadically or not at all. Lesions on leaves are initially yellow

on white and bright red to reddish brown on red cultivars. Subsequently a reddish

brown necrosis develops in the center of the lesion, leaving only a thin margin of

yellow or red tissue between the necrotic and green areas of the leaf. The lesions are

typically confined to the major veins and the edge of the leaf and are several centimeters

wide. Early infections occur on the first to the sixth leaf of young shoots, resulting in

minor losses. Later infections attack leaves up to the 10th or 12th position on the

shoot which result in severe defoliation. In addition, fungus attacks inflorescences

and berries causing them to rot and dry out (Mohr et al. 2005).

*Taxonomy*. The causing fungus of Rotbrenner, *Pseudopezicula tracheiphila*

(Müll.-Thurg.) Korf & Zhuang (syn. *Pseudopeziza tracheiphila* Müll.-Thurg.)

belongs to its teleomorph *Phialophora tracheiphila* (Sacc. & Sacc.) Korf to the

Helotiales (Ascomycetes) (Korf et al. 1986).

*Biology and Epidemiology*. The source of inoculum of the disease in spring is

ascospores which are formed sexually in asci. P. *tracheiphila* appears to be composed

of two mating types which exhibit a bipolare heterothallic mating system.

Apothecia formed primarily on fallen leaves in the spring, hold the asci with the

ascospore. Apothecia may also develop on current-season infected leaves in late

**Fig. 4.6** First stage of development of *Botrytis cinerea* 17 h after inoculation, Low-Temperature-

Scanning-Electron-Microscopy (Jäger, B., Jacków, J., Kassemeyer H.-H. and Düggelin M.,

University of Basel)

4 Fungi of Grapes 75

summer or fall. Depending on weather conditions, apothecia with mature ascospores

may be present throughout the season (Perarson et al. 1991). The primordia of the

apothecia mature as soon as the temperature rises at the end of winter. Apothecia

development requires sufficient wetness of fallen leaves. Under wet and warm conditions

ascospores are released already before bud burst. Heavy rainfall and prolonged

surface wetness favour infection and lead to severe disease. Young leaves

are susceptible after they reach a width of about 5 cm but the probability of infections

increases from the 6-leaf stage. After an incubation period of two to four

weeks, the fungus invades the vascular elements of infected leaves, causing symptom

development (Reiss et al. 1997). The fungus remains latent if it is unable to

invade the vessel elements, in which case it can be isolated from green leaves showing

no symptoms. Conditions required for fungus to invade the vascular system are

not well understood; however, soil conditions and water supply that place the vine

under temporary stress appear to be important factors. Disease incidence and severity

depend on the abundance of apothecia on fallen leaves on the ground of the

vineyards and on released ascospores. Monitoring of the ascospore release by

means of spore traps enables forecast of the disease situation. On malt agar, the

anamorph may be formed, with hyaline, septate, short conidiophores that are

coarser than vegetative hyphae. Conidiogenous cells are monophialidic and lageniform,

with well-defined but thin-walled collarettes. Conidia are ellipsoid, hyaline,

and unicellular. Hyphae grow in a characteristic sine-wave pattern that, when

observed in the vessel elements of diseased tissue, are considered diagnostic.

A disease very similar to Rotbrenner*,* called angular leaf scorch, has been

described in New York State (Person et al. 1988). The fungus causing angular leaf

scorch in North America produces smaller apothecia than *P. tracheiphila,* and its

broadly clavate asci has four spores in contrast to the eight-spored European fungus.

The American counterpart has been described as a distinct species, *P. tetraspora*

Korf, Pearson & Zhuang (anamorph *Phialophora*-type).

***4.3.4 Phomopsis viticola* (Sacc.) Sacc (Diaporthales):**

**Phomopsis Cane and Leaf Spot**

*General Aspect*. Phomopsis cane and leaf spot first observed in 1935 in California

is also widespread in Europe for more than 50 years. Actual loss of quality due to

the disease in most years is insignificant. However in rainy spring years, severe

infections occur and cause lesions on shoots. In addition shoot infections affect the

formation of basal buds and in consequence in the following year buds on the base

of the canes especially do not sprout. Repeated infections affect the fertility of the

basal parts of the canes and shorten life span of the vine. Under cool and rainy conditions

during berry ripening berry infections occasionally occur. The first symptoms

on shoots are dark brown to black spots on the shoot base visible from the

three- to six-leaf stage. The spots elongate and the cortex crack due to secondary

growth of the shoots. Large numbers of spots at the shoot base become scabby and

76 H.-H. Kassemeyer

black. Heavy infected shoots can be dwarfed and may die. During winter, infected

canes bleach and black pustules occur. Cluster infections show black spots on the

rachis. However these lesions become inactivate in the course of cluster development.

Rainfall in autumn rarely reactivate the lesions and cause berry infections.

Infected berries show brown spots which enlarge quickly and cause a bunch rot.

*Taxonomy. Phomopsis viticola* (Sacc.) Sacc. (*Sphaeropsis viticola* Cooke)

belongs to the Diaporthales ( Ascomycetes). The teleomorph *Diaporthe* according

to current knowledge is very rare in viticulture (Agrios 1997; Gams et al. 1998).

*Biology and Epidemiology. Ph. viticola* overwinters on infected canes and black

pustules on bleached canes occurring during dormancy are pycnidia (Fig. 4.7) where

pycnospores develop. Generally infections occur in spring as soon as pycnospores

mature in the pycnidia and green shoots sprout. During rainfall pycnospores emerge

in large quantities from the pycnidia embedded in vermiform cirri. Pycnospores (Fig.

4.8) are dispersed by splashing raindrops onto the sprouting shoots and infections

occur when water remains on the green host tissue for a longer time. Prolonged wetness

of sprouts and young shoots from bud break up to the six-leaf stage, favour

infections by *Ph. viticola*. The number of basal buds affected by fungus vary according

to frequency of rainfall and wetness of the host surface. After infection, mycelium

growth in the infected host tissue but mainly shoots and buds are colonized. During

summer *Ph. viticola* is less active, but in wet autumn mycelium may be reactivated

and berry infections may occur (Agrios 1997; Mohr et al. 2005).

***4.3.5 Elsinoë ampelina* Shear ( Myringiales): Anthracnose**

*General Aspects*. Anthracnose was widespread in earlier times in European viticulture

and before downy mildew was identified as the most dangerous of grapevine disease.

**Fig. 4.7** Cane with *Phomopsis viticola* pyknidia

4 Fungi of Grapes 77

Due to regular application of fungicides, anthracnose occurs only sporadically

under very humid conditions in untreated vineyards. Infected shoots show light

brown spots with black-violaceous edges. Black circular lesions occur on the leaves

that necrotize and over time gives rise to small holes like a shot gun effect. Affected

berries show sunken circular lesions with black-violaceous edges (“bird’s eyes”)

which crack and finally decay. Infections of the rachis cause necrosis of the cluster

with “bird’s eyes” on the stems (Mohr et al. 2005). Shoots and leaf infections

reduce the vigour of vine, yield and quality and shorten the life span of the plant.

Decayed berries have to be removed because they can influence the quality of must

and wine (Magarey et al. 1993; Sosnowski et al. 2007)).

*Taxonomy*. The causing fungus of the anthracnose, *Elsinoë ampelina* Shear (syn.

*Gloesporium ampelophagum* (Pass) Sacc., *Ramularia ampelophagum* Pass.,

*Sphaceloma ampelinum* de Bary) is a member of the Elsinoaceae family which

comprises ten genera (Gams et al. 1998). Elsinoaceae and Myrangiaceae belong

together to the order of Myrangiales which is a member of the larger class of the

Dothideomycetes (Ascomycetes).

*Biology and Epidemiology. E. ampelina* overwinters as sclerotia on the canes

which are formed in the autumn at lesions on shoots. The sclerotia develop stromata

on which under humid conditions shell-like acervuli with conidia emerge in the

spring (Agrios 1997). The conidia are covered with a gelatinous layer and provide

primary inoculum at the beginning of the vegetation period. Conidia propagation is

favoured by rainfall and for conidia germination, wetness of the host surface for 12 h

is necessary. At times fruiting bodies with asci and ascospores develop on the lesion.

The propagules are transported during rainfall over a short distance; thus the disease

initiates on more ore less widespread spots within the vineyards (Brook 1992).

**Fig. 4.8** Pycnospores from Phomopsis viticola and hyphae with characteristic septae; Differential

Interference Contrast (63x)

78 H.-H. Kassemeyer

***4.3.6 Guignardia bidwellii* (Ellis) Viala & Ravaz**

**( Dothideales): Black Rot**

*General Aspects*. Black rot originated from North America and has been in Europe

for nearly 30 years. The disease occurs particularly in abandoned vineyards and

also on resistant cultivars which are not treated with fungicides. To date black rot

is restricted to some viticultural regions but the disease is becoming more common.

Typical symptoms on leaves are light brown necrotic lesions with black edges up

to 10 mm in diameter. Within the necrotic spots black dots are barely visible to the

naked eye. On shoots, petiols and the rachis black sunken lesions appear. Infected

young berries primarily show pale spots which enlarge to concentric red-brown

lesions. Within a view days the affected berry gets blue-black and is covered with

black pustules (Fig. 4.9 ). The berries finally wrinkle and dry, but remain as mummies

fixed on the rachis. Frequently originated from some infected berries, the whole

cluster can be infected. High infestation of black rot defoliate the canopy and as a

result decrease the quality of grapes seriously. Cluster infections have an effect on

yield and berry quality and affected grapes are not suitable for wine production

(Pearson and Goheen 1988; Mohr et al. 2005).

*Taxonomy*. Black rot is caused by *Guignardia biwellii* (Ellis) Viala & Ravaz

(syn. *Greeneria uvicola* (Berk. & M.A. Curtis) Punith., *Botryosphaeria bidwellii*

(Ellis) Petr.) which belongs to the Dothideales an order within the Dothideomycetes

(Ascomycetes) comprising some other plant pathogens such as *Ascochyta*,

*Didymella*, *Botryosphaeria* and *Phoma* (Agrios 1997; Gams et al. 1998).

*Biology and Epidemiology. G. bidwellii* overwinters mainly in the mummified

clusters and berries remaining on the shoot and also on infected canes. Asci with

ascospore develop in perithecia on infected berries in spring . The ascospore are

**Fig. 4.9** Berry affected by black rot (*Guignardia bidwellii*)

4 Fungi of Grapes 79

ejected actively from the asci during low rainfall and spread by wind. For ascospore

germinate, prolonged wetness of host surface is necessary. All young green grapevine

tissue including shoots, inflorescences and berries may be infected. At the beginning,

infections are hard to detect but with progressed development of the fungus,

necrotic spots are visible, and finally necrotic lesions occur. Within necrotic lesions

on leaves, shoots and berries pyknidia with pyknospores develop during the season.

Pyknospores are released during rainfall and cause infections on berries. In late

summer the sexual cycle initiates on infected berries and perithecia are formed

which overwinter on the infected mummified berries and clusters (Jermini and

Gessler 1996; Hoffman et al. 2002; Longland and Sutton 2008).

***4.3.7 Penicillium expansum* Link ( Eurotiales): Green Mold**

*General Aspects*. Green mould is a secondary disease on mature berries, after

wounding or bunch rot infections. Green mould occurs in warm and humid years

when berries enter into ripening stage precociously. Recently incidence of green

mould increases may be due to high temperature in summer and frequent precipitation

during berry ripening. White pads occur on the edges of wounds and cracks

which enlarge and change to glaucous (Fig. 4.10). Infected berries soften and

change colour from olive-green to light-brown. In an advanced stage of infection

berries decay and shrink under dry conditions. Due to the squeezing of berries

and related wounds closed bunches are more frequently affected by green mould.

These clusters show nests inside with decayed berries (Mohr et al. 2005).

Therefore cultivars with close bunches are more susceptible than those with loose

bunches. Besides in years with high berry set resulting in dense clusters green

mould occurs more frequently. From single infected berries the whole cluster

**Fig. 4.10** Berry infected by *Penicillium expansum*, the fungus colonizes pores in the epidermis

on which nutrients leak from the berry

80 H.-H. Kassemeyer

may be affected causing mummified clusters covered with green mould. Green

mould produce mycotoxins (Abrunhosa et al. 2001; La Guerche et al. 2004;

Serra et al. 2006; Pardo et al. 2006) for example patuline which is however

degraded during fermentation and by sulphurization. Berries affected by green

mould have an off-flavor and even a small amount of infected berries add a

mouldy taste to the wine.

*Taxonomy*. The causal agent of green mould is mainly *Penicillium expansum*

Link; other species of *Penicillium* can also be detected on affected berries (Serra

and Peterson 2007). The genus *Penicillium* is regarded as a member of the

Deuteromycotina but according to current taxonomy it belongs to the Eurotiales

(Ascomycetes) (Agrios 1997; Gams et al. 1998; Mc Laughlin et al. 2001).

*Biology and Epidemiology. P. expansum* is ubiquitous and propagates by conidia

which are formed abundantly on conidiophores. The conidiophores of *P. expansum*

consist of two asymmetric branches with a number of flask-shaped phialides at the

tip of each branch. Phialides are conidiogenous cells which produce masses of

conidia in short intervals under humid and warm conditions. The phialides appear

as clusters on each tip of the conidiophore and the conidia are formed in chains on

each phialide. Conidiophores with the mass of conidia are visible as white to glaucous

pad on infected berries (Gams et al. 1998). *P. expansum* is a typical airborne

pathogen and the long-living conidia are transported by wind. In consequence

conidia are released even by a gentle movement while removing infected clusters.

Conidia germinate on wet surface of berries as soon as a sugary medium is available.

Possibly vigorous berry development due to high amount of water supply and

high temperatures causes micro cracks in the berry skin and consequently sugar

runs off the slow berries. The temperature range of *P. expansum* for conidia germination,

growth of the mycelium and sporulation is relatively broad, but optimal

development of the fungus occur at 25°C and high humidity. Under cool and dry

conditions *P. expansum* is rare even on berries whose skin is not intact. Slow and

consequently late ripening cultivars and those with a strong epidermis are less susceptible

to colonization by *P. expansum*.

***4.3.8 Aspergillus spec.* ( Eurotiales): Aspergillus Rot**

*General Aspects*. Aspergillus rot is widespread on substrates containing a disposable

source for carbohydrates such as mono- and polysaccharides. Rot is common in

crops and fruits and contaminate also sugary and starchy foods. At present aspergillus

rot occurs on grapevine particularly in warm climate (Leong et al. 2007). The

symptoms of aspergillus rot are visible as soon as sugar leaks from ripening berries

after the beginning of veraison. The surface of infected berries is covered by a black

mould and the berries decay. Aspergillus rot produces ochratoxins (Samson et al.

2004; Pardo et al. 2006) and contaminate must and wine with this mycotoxin suspected

to be carcinogenic. For this reason clusters affected with aspergillus rot have

to be sorted at harvest.

4 Fungi of Grapes 81

*Taxonomy*. Aspergillus rot is caused by different members of the genus

Aspergillus which are widely distributed worldwide. On grapes particularly *A. alliaceus*

Thom & Church, *A. carbonarius* (Bainier) Thom, *A. niger* aggregate Tiegh. and

*A. ochraceus* G. Wilh. occur. Totally the genus *Aspergillus* comprises more than

200 taxa including species with numerous sub-species and is a genetically heterogenous

group. Therefore the current taxonomy can change in the course of new

findings on the phylogeny of this group. Some teleomorphs associated with

*Aspergillus* are known for instance, *Emericella* Berk & Br. and *Eurotium* Link and

allow integration in the Eurotiales (Mc Laughlin et al. 2001).

*Biology and Epidemiology. Aspergillus* species sporulate asexually by forming

conidia without fruit bodies. Conidia develop on conidiophores which are sometimes

aggregated and visible as a black powdery pad. The unbranched conidiophores terminate

in vesicle on which phialides arise (Gams et al. 1998; Domsch et al. 2007 ). At the

tip of the flask-shaped phialides, conida develop in chains which are spread by

wind. The optimum temperature range for development of *Aspergillus* is 17–42°,

minimum temperature for growth is 11–13°C.

***4.3.9 Coniella petrakii* B. Sutton (Diaporthales): White Rot**

*General Aspects*. White rot occurs sporadically in southern viticultural regions

while in cool and moderate climate viticulture the disease appears very rarely.

Wounds, mainly from hail, favour infections by white rot. Above all damages

appear on affected rootstocks showing brown spots. Rootstocks infected by white

rot are not suitable for grafting and may disseminate the disease. Infected berries

become yellowish, shrink and have brown pustules. Due to the development of the

pustules on the berry skin the cuticle detaches from the epidermis and as a result

the berry becomes pale. Clusters affected by white rot should be sorted at harvest

because otherwise they may influence the quality of must and wine. In most cases

white rot is controlled by regular treatments against downy mildew and bunch rot.

*Taxonomy. Coniella petrakii* B.Sutton is the causal agent of white rot and

belongs to the order of Diaporthales (Ascomycetes) (Tiedemann 1985).

*Biology and Epidemiology*. The mycelium of *C. petrakii* is frequently septated and

abundant branched. Globose and ostiolate pycnidia are formed by a stroma below the

cuticle. The elliptical or ovate shaped pycnospores are single-celled and light brown and

arise from a basal stroma in the pycnidia from the pycnidial wall (Sutton and Waterston

1966; Locci and Quaroni 1972; Tiedemann 1985). *C. petrakii* is soil borne and splash

events are necessary to transport the propagules onto the host surface (Aragno 1973).

High temperatures between 24 and 27°C favour conidia development and infection.

After infection of wounded host tissue, the incubation period varies from 3 to 8 days

(Bisiach 1988). Masses of pycnidia forming pycnospores arise from the berry surface

and overwinter on the ground and are source of inoculum over years. High temperature

and simultaneous wetness of the host surface necessary for successful infections

exclude in most cases, infestation in cool or moderate climate viticulture.

82 H.-H. Kassemeyer

***4.3.10 Alternaria alternata* (Fr.: Fr.) Keissler**

**(Pleosporales): Alternaria Rot**

*General Aspects*. Alternaria rot is ubiquitous and distributed world-wide. A number of

fruit and crops may be affected; also foodstuffs and organic material like textiles,

leather and paper. Alternaria rot causes merely marginal losses of berry quality in viticulture

and colonize mainly ripe berries with leaked sugar. Colonized berries show a

black smut on the surface. Only occasionally injured berries are infected. In this case

Alternaria rot raises a mouldy taste of grapes and wine and produces mycotoxins.

Therefore infected clusters have to be sorted at harvest.

*Taxonomy*. To the taxon *Alternaria* belongs to numerous species of which

*Alternaria alternata* (Fr.: Fr.) Keissler is most common on grapevine. The genus

*Lewia* is described as teleomorph for *Alternaria* (Pleosporales, Ascomycetes)

(Gams et al. 1998; Mc Laughlin et al. 2001).

*Biology and Epidemiology*. The conidiophores of *Alternaria alternata* produce pale

to medium brown conidia in long, often branched chains. The brown to olive-green

conidia have transverse and longitudinal septae and a cylindrical or short conical beak

(Samson and Reenen-Hoekstra 1988). The fungus has a saprophytic lifestyle and prefers

a sugary substrate but occasionally it become parasitic. For setting an infection, high relative

humidity is necessary (98–100%). Under these conditions the germination peg of the

conidia is able to penetrate the epidermis directly. Therefore, frequent rain in late summer

and autumn is favourable for the infection process (Hewitt 1988; Valero et al. 2007).

***4.3.11 Cladosporium herbarum* (Pers.) Link**

**(Capnodiales): Cladosporium Rot**

*General Aspects*. Cladosporium rot is widespread and very common in temperate

regions on dead or dying plant substrates and other organic matter. Cladosporium

rot is typically a post-harvest disease on fruits and crops. Late harvested grapes and

table grapes may be infested by the rot and berries can decay. No major mycotoxins

of concern are produced (Frisvad 1988; Northolt and Soentoro 1988), however volatile

organic compounds are accumulate conferring a mouldy off-flavour to the

affected clusters.

*Taxonomy*. The genus *Cladosporium* comprises numerous species of which

some are the most common indoor and outdoor moulds. On grapevine clusters and

berries mainly *C. herbarum* (Pers.) Link occurs (Whitelaw-Weckert et al. 2007).

According to its teleomorph *Davidiella tassiana* (De Not.) Crous & U. Braun

(emend. *Mycosphaerella tassiana* (de Not.) Johanson) the fungus belongs to the

Capnodiales (Ascomycetes) (Gams et al. 1998; Mc Laughlin et al. 2001).

*Biology and Epidemiology*. Colonies of *C. herbarum* are velvety- powdery and

the colour is olivaceous to brown due to the abundant mass of conidia formed on the

mycelium. Smooth-walled conidiophores have terminal and intercalary swellings.

At the tip of the conidiophores one-, two- or more-celled conidia are formed in sim4

Fungi of Grapes 83

ple or sometimes branched chains. The conidia are ellipsoidal to cylindrical and

pigmented. They germinate on moist surfaces and lesions are black and circular

ranging from several millimeters in diameter to up to two-thirds of the berry surface.

If the lesion turns olivaceous, sporulation starts and conidiophores with numerous

conidia are present. The fungus has a broad temperature range (4–30°C), the optimum

lies between 20 and 24°C (Hewitt 1988; Whitelaw-Weckert et al. 2007).

***4.3.12 Trichothecium roseum* (Pers.) Link**

**(Hypocreales): Pink Rot**

*General Aspects*. Pink rot occurs under high relative humidity and rain on berries after

veraison. Normally the rot is associated with wounds and may occur on berries

infected by *Botrytis* cinerea which acts as a primary invader. Infected berries show

white to pink pads, shrink and decay. Expended infections cause mummified clusters

with pinkish covering. While harvesting, rotten parts of the clusters should be

sorted out as mycotoxins such as crotocin, trichothecin and roseotoxin may be produced

(Frisvad 1988). Additionally the rot causes an unsavoury, bitter taste in

affected parts of the cluster and can be responsible for off flavour in the wine.

*Taxonomy. Trichothecium roseum* (Pers.) Link belongs to the order of Hypocreales

(Ascomycetes); however the current Taxonomy is *incerta sedis* (Gams et al. 1998;

Mc Laughlin et al. 2001).

*Biology and Epidemiology*. On berry surfaces, pinkish erect, unbranched conidiophores

arise from the mycelium. Conidiophores are often septate near the base and

more or less rough-walled. At the apex, chains of ellipsoidal to pyriform conidia

develop by retrogressive division (Gams et al. 1998). Young conidia are aseptate,

and when they mature, one septum in the middle of the conidia is formed. After

conidia removal from the conidiophore an obliquely truncate basal scar is present

where the conidia have been inserted into the conidiophore. Fungus develop also at

lower temperature but optimal conidia germination and hyphae growth occurs at 25°C

(Samson and Reenen-Hoekstra 1988).

**4.4 Zygomycetes**

***4.4.1 Rhizopus stolonifer* (Ehrenb.) Lind.**

**(Mucorales): Rhizopus Rot**

*General Aspects*. Rhizopus rot is common on soft fruits, more abundant in warm,

humid climates than in cool climate viticulture. In several fruits and crops such as

strawberry, tomato, cucumber and table grapes Rhizopus rot causes a soft rot during

transport and storage (Hallmann et al. 2007). Rhizopus rot also affect bread and is

known as black bread mould.

84 H.-H. Kassemeyer

*Taxonomy*. The causal agent *Rhizopus stolonifer* (Ehrenb.) Lind is a member of

Mucorales which belong to the Zygomycetes a phylum of the kingdom of Mycota

distinct from the Ascomycetes (Mc Laughlin et al. 2001).

*Biology and Epidemiology. R. stolonifer* is heterothallic and sexual reproduction

occurs exclusively when opposite mating types fuse (Schipper and Stalpes 1980).

The young mycelium is whitish, becoming greyish-brown due to brownish sporangiophores

and brown-black sporangia. Sporangiophores stand alone or in groups of

usually 3–4. They are extremely tall, often over 20 mm high, erect and unbranched.

At the opposite side of the globose sporangia branched rhizoids are obvious, a typical

formation of *R. stolonifer* among most other *Rhizopus* species (e.g. *R. oryzae*).

The columella is of globose to ovoid shape. The sporangiospores are irregularly

formed (polygonal, globose, ovoid) with numerous striations on the spore surface

(Samson and Reenen-Hoekstra 1988). Rhizopus rot is not restricted to berry infection

alone. Under humid weather conditions, the fungus may spread to other berries

in a cluster, causing a bunch rot (Hewitt 1988).

***4.4.2 Mucor* Spp (Mucorales): Mucor Rot**

*General Aspects*. In general Mucor rot occurs as a post-harvest disease on fruits

including table grapes (Hallmann et al. 2007). The rot is very rare on grapes for

wine production.

*Taxonomy*. The three species *Mucor mucedo* Fresen, *M. hiemalis* Wehmer and

*M. piriformis* A. Fisch. are characteristic species of the Mucorales (Zygomycetes)

(Mc Laughlin et al. 2001).

*Biology and Epidemiology*. The thallus of *Mucor* spp. is white or coloured. The

sporangiophores end in a globose sporangium in which spores are formed. The wall

of the sporangiophore bursts for spore release (Gams et al. 1998). *Mucor* spp. can

even grow and develop at cool storage conditions, In the case of *M. hiemalis* the

optimum temperature for growth and sporulation is 5–25°C (Samson and Reenen-

Hoekstra 1988).

**4.5 Conclusions**

The majority of fungi causing berry rot need mono- and oligosaccharides for spore

germination and formation of a mycelium which is at disposal in the substrate.

Consequently berry rot is favoured by sugar leaking from the ripening berries. In

addition all these fungi develop at high temperature with an optimum from 22 to

24°C. Precocious veraison and consequently early onset of sugar import in the

berry under high temperature in the summer particularly provide optimal conditions

for berry infection. Susceptibility of berries to fungi is also triggered by the structure

of the berry skin. An intact berry skin with a dense layer of epicuticular waxes

4 Fungi of Grapes 85

and a compact cuticle provide a constitutive barrier against invaders. On the other

hand a weak skin is permeable for both the infection structures of the invading fungus

and sugars from inside the berries. The structure of the berry skin depends on

numerous factors- among others on the weather conditions, water supply and nitrogen

uptake of the vine. Warm and humid conditions favouring the development of

fungi may also reduce reinforcement of the berry skin. These interactions between

susceptibility of berries for fungi and the epidermal tissue including the cuticle

have to be noted for the control of berry rot. Another aspect of berry colonizing

fungi is the off-flavour and the production of mycotoxins which can devastate the

yield of the affected vineyard. To raise the quality and to avoid remarkable economical

loss all measures have to be taken to control fungi colonizing berries and

clusters. An important prerequisite for that is the knowledge of the biology, biochemistry

and epidemiology of fungi.