

# POSTHARVEST DECAY ON STONE FRUIT - WHAT, WHEN and HOW TO REDUCE

## Part 1: Understanding the infection process of the decay causing pathogens and factors affecting decay development

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### Introduction

Flavour, aroma, firmness (delayed softening) and eating quality after purchase, influences consumer preference and acceptance of stone fruit. These expectations demand sales of ripe, almost ready to eat fruit, with no decay. Decay development is potentially a major problem on stone fruit during storage. Losses of stone fruit during the postharvest phase can be rapid and severe, especially if fruit are of advanced maturity. Preharvest factors have a large impact on the level of postharvest decay. To control decay development, it is important to understand the causal organisms, the physiology of the fruit and the environmental conditions before, at and after potential infection periods. A general overview on 'Postharvest decay on stone fruit' has been compiled as a two part series, with Part 1 - **Understanding the infection process of the decay causing pathogens and factors affecting decay development** and Part 2 – **Pre- and postharvest control measures to reduce decay development**, to be published in the next South African Fruit Journal.

### Is decay on stone fruit increasing ?

No solid evidence is available to confirm a trend of increased decay levels on South African stone fruit over the years. However, what is incontestable is that the focus on postharvest decay has intensified, suggesting an increased awareness. It is highly likely that changes in weather conditions are increasing the decay status, for example, extended winter rain during flowering and onset of ripening, as well as higher temperatures and thunder storms during fruit development and harvesting. Climate affects many factors related to disease development, such as overwintering of the fungus, reproduction and new infections. The cultivar range of stone fruit has changed extensively in South Africa over the years, with very little information available on the susceptibility of new cultivars to decay. Furthermore, production practices have changed, with more emphasis on yield, putting pressure on many activities, such as handling, harvesting, packing, cooling, storage and marketing. Although the conditions under which stone fruit is stored directly affects disease development,

conditions under which the produce is grown, often determine its potential for successful storage and marketing (Conway, 1984).

### Infection process / Disease cycle

Causes of postharvest losses on stone fruit can be classified as parasitic, non-parasitic or physical (Cappellini & Ceponis, 1984). Attention is given in this overview to parasitic causes that are of microbiological origin, causing infection to stone fruit before or after harvest.

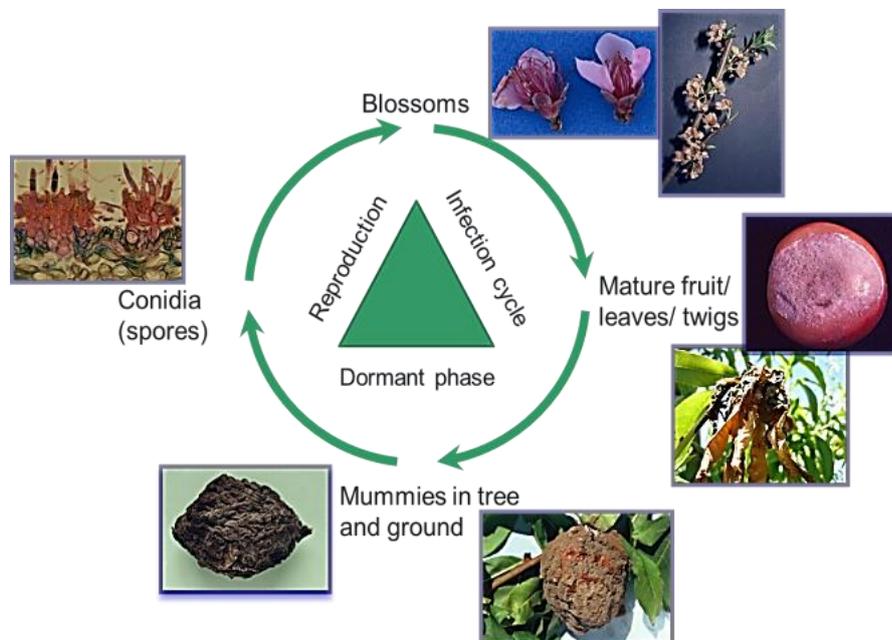
Postharvest decay on stone fruit is most frequently caused by fungi, rather than bacteria (Sholberg & Conway, 2004). *Botrytis cinerea* (grey mould), *Monilinia laxa* (brown rot), *Penicillium expansum* (blue mould) and *Rhizopus stolonifer* (bread mould, or whisker rot) are the most common decay causal pathogens on stone fruit (Adaskaveg, Förster, Gubler, Teviotdale & Thompson, 2005; Fourie & Holz, 1995; Snowdon, 1990). Other micro-organisms such as *Alternaria* and *Aspergillus*, as well as yeasts, may occasionally cause decay in the field and during storage.

Fruit rot micro-organisms either infect the produce while attached to the tree, or during subsequent harvest, handling and marketing. The fungal spore functions as the infection propagule for development of disease (Sommer, Fortlage & Edwards, 1984). The spore germinates under favourable conditions, of which moisture (high humidity or free water) and moderate temperature ( $\pm 15 - 25^{\circ}\text{C}$ ), are most important for infection and reproduction. Sufficient wetness is required to generate infection periods. Even dew can suffice as sufficient wetness to instigate the infection process. The pathogen enters the fruit either directly through the skin, or by gaining entrance through natural openings such as stomata, lenticels, or wounds such as abrasions, punctures, bruises and growth cracks (Figure 1). Even microscopic damage is sufficient for the spores to gain access to the fruit. Insect damage is often instrumental in stone fruit infections.



**Figure 1:** Decay development on stone fruit associated with insect and stem puncture wounds, and natural cracks

The germ tube, which emerges from the spore, grows on the fruit surface, or into the fruit tissue, where it colonises the flesh. Direct penetration of the fruit is in some instances aided by special attachment structures (appressoria), which affix to the fruit surface. These structures are not readily removed by general washing processes. A thin infection peg emerges from the appressorium or germ tube, capable of penetrating the skin and fruit tissue by enzymatic action (Fourie, 1992). After penetration, the infection peg regains its original size, with mycelia invading and colonising the fruit flesh. In some instances infection from direct penetration is interrupted, due to resistance of the host, with the infection peg unable to develop further and hence invade the fruit. In this instance, the infection process is said to remain quiescent. Quiescent infections can be defined as visible or non-visible (latent) infections that could potentially develop at a later stage, under conducive environment conditions, when host resistance is reduced (Adaskaveg, Förster & Thompson, 2000; Verhoeff, 1974). Each pathogen has a distinct infection method and disease cycle, which is followed through the season, and into to the next (Figure 2).



**Figure 2:** Disease cycle of *Monilinia laxa* in stone fruit

Susceptibility of stone fruit to decay is related to the development stage at which the host material is challenged by the pathogen. Blossoms and mature fruit are by far the most susceptible. Immature stone fruit often have natural ability to overcome infection by *Botrytis* and *Monilinia* (Fourie, 1992). Thinned fruit on the orchard floor can be infected under favourable climatic conditions. Infection and sporulation of *Monilinia* decreases if the water content of thinned fruit is reduced by natural drying. Fast drying of thinned fruit is essential, and if this cannot be achieved, such fruit should be removed, since they are significant sources of later infections.

## Decay causing pathogens

### Brown rot (*M. laxa*)

#### *Infection and appearance*

Different *Monilinia* species may cause decay on stone and pome fruit, with *M. fructicola* (American brown rot) and *M. laxa* (European brown rot) causing brown rot on stone fruit (Byrde & Willets, 1977; Jones & Sutton, 1996). In studies conducted from 1984 onwards, only *M. laxa* has been isolated from decayed stone fruit in South Africa (Fourie, 1984; Fourie, 2001; Carstens, van Niekerk & Laubscher, 2010). Brown rot can reduce profitability by rotting fruit on the tree and during storage. *M. laxa* is very active early spring, during blossoming, as well as after the onset of fruit ripening. The fungus may infect blossoms, or fruit, and can spread into twigs, branches or leaves (Figure 3). It can cause spurs and shoots to wither and die, with occurrence of typical blossom blight symptoms. Gummosis may appear on spurs, often accompanying blossom blight symptoms. Canker formation occasionally occurs. Brown rot infected blossoms and spurs are important inoculum sources for fruit infections (Adaskaveg *et al.*, 2005).



**Figure 3:** *Monilinia* decay, spreading from an infected fruit to leaves and twigs

Fruit rot occurs by direct penetration, or through wounds. Quiescent *Monilinia* infections may develop when the fruit ripens, becoming visible during storage. *M. laxa* over-winters in mummified fruit remaining in the tree after harvest, or which have fallen to ground (Figure 4). Diseased leaves and twigs can also be a source for new infections the following season. Conidia (known as spores) are produced when conditions become suitable. *M. laxa* will grow slowly at temperatures near 0°C, becoming more active if the temperature increases.



**Figure 4:** *Monilinia* fruit mummies in the tree or on the orchard floor

*Monilinia* fruit rot symptoms initially appear as small, circular, light brown spots on the fruit surface, expanding rapidly under favourable weather conditions. Under wet and humid conditions, tan-coloured tufts of sporodochia, fungal structures bearing conidia, appear on the infected tissue (Figure 5). Under conditions of extended storage, the skin of infected stone fruit may darken (Figure 6). Wetting hours required for *M. laxa* infections are reduced from 18h at 10°C to 5h at 25°C. Fruit cracked from rain are particularly susceptible to infection.



**Figure 5:** Tufts of tan coloured spore bearing structures of *Monilinia* on plum and peach fruit



**Figure 6:** Darkening of the skin of peach and plums associated with *Monilinia* decay

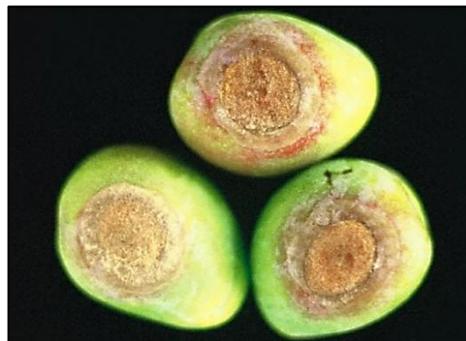
#### *When and how to reduce:*

Brown rot is best controlled by a combination of sanitation practices and applying a protective fungicide programme. Disease resistant cultivars are not readily available. However, less susceptible cultivars can be selected for high, decay risk production areas. Blossom infections and subsequent fruit rot can be controlled by applying fungicide sprays during bloom, followed by pre-harvest fungicide sprays when the fruit start to colour or soften ( $\pm$  3w before harvest). Control of insects, especially those that directly injure the fruit, will assist in preventing infections. Fruit should be picked and handled with care to avoid injuries. Periodic removal of overripe and rotten fruit from the orchard will assist in reducing losses from decay. Postharvest fungicide applications and cleaning of pack lines will also assist in controlling brown rot.

Grey mould (*B. cinerea*)

*Infection and appearance*

Grey mould, caused by *Botrytis cinerea*, is most active on fruit after onset of ripening (Coley-Smith, Verhoeff & Jarvis, 1980). However, *B. cinerea*, similar to *Monilinia*, can infect blossoms, causing blossom blight. Blossom blight caused by *Botrytis* is often thought to be less problematic than *Monilinia*, but the impact of *Botrytis* blossom infections is often underestimated. Rotting of green fruit by *B. cinerea* (Figure 7) is more common than with *M. laxa*. Infection of immature fruit occurs under conducive weather conditions of extended wetting, in the presence of a high inoculum load. Fruit rot may occur as the fruit matures, either by direct penetration, or through wounds. Similar to *Monilinia*, most infections of stone fruit by *Botrytis* occur through wounds inflicted during harvesting and handling. Split pip in peaches or nectarines may also suffice as point of entrance for *Botrytis*. This decay is often not visible from the outside of the fruit (Figure 8). *Botrytis* overwinters in rotten fruit which have fallen to ground, and in diseased, pruned twigs, which act as a source for new infections the following season. *B. cinerea* overwinters on decayed plant material in the soil, by producing specialised, hardened structures (sclerotia). Conidia are produced on the diseased material and from sclerotia when conditions become suitable. *B. cinerea* grows at temperatures near 0°C, but becomes more active at higher temperatures.



**Figure 7:** Fruit rot on immature green fruit caused by *Botrytis cinerea*



**Figure 8:** *Botrytis* split-pip decay occurring around the stone of peaches (left), is often not visible on the outside of the fruit (right)

*Botrytis* fruit rot symptoms initially appear as small, circular, light brown spots on the fruit surface. Under wet and humid conditions, ash-grey spores are produced on the infected tissue (Figure 9). Spread of *Botrytis* decay from an infected to an adjacent sound fruit is common, on the tree and during storage.



**Figure 9:** *Botrytis* decay on the surface of peach and nectarine fruit

*Botrytis* and *Monilinia* decay on stone fruit are often, incorrectly identified, since symptoms are very similar. *Botrytis* growth on the fruit surface is much more even, whereas *Monilinia* bears tuft-like spore structures (Figure 10). Growth of the two fungi on fruit in the orchard also differs distinctly, with *Botrytis* bearing smoother, grey-toned fungal matter (Figure 11).



**Figure 10:** Symptoms of *Botrytis* (left) and *Monilinia* (right) on a decayed, inoculated fruit, distinguished by the even growth of *Botrytis* and tuft-like appearance of *Monilinia*



**Figure 11:** Smoother fungal growth appearance of *Botrytis* (left) compared to tufts of *Monilinia* (right), on decayed fruit in the orchard

*When and how to reduce:*

Control measures for *Botrytis* decay are similar to *Monilinia*. Blossom infections should be controlled to avoid subsequent fruit rot, especially at the ripening phase. Sanitation, by removal available sources for infections, such as thinned fruit on the ground and rotten fruit in the tree or on the orchard floor, is essential for decay reduction. Practices which enhance drying of thinned fruit, may further assist in decreasing the source of infections. Chemical and other control strategies should be utilised to counter infections and decay development by *B. cinerea* in orchards, as well as during storage.

*Rhizopus* and *Mucor* rot (*R. stolonifer* and *M. piriformis*, respectively)

*Infection and appearance*

*Rhizopus* rot, caused by *Rhizopus stolonifer* and *Mucor* rot, caused by *Mucor piriformis*, are most problematic on ripe, or near ripe fruit. *Rhizopus* rot can be a serious postharvest disease on stone fruit, particularly peaches (Snowdon, 1990). *Rhizopus* and *Mucor* are very similar in appearance in culture and on decayed fruit, with the difference that the sporangiophores (spore bearing structures) of *Rhizopus* are longer than that of *Mucor* (Figure 12), with *Rhizopus* exhibiting an interwoven web of fungal matter, especially on stored fruit (Figure 13). In the field, under warm, humid conditions, both organisms at first produce short sporangiophores with white fruiting bodies appearing at the edges of the decayed area. The white spore bearing structures (sporangia) turn black as they mature.

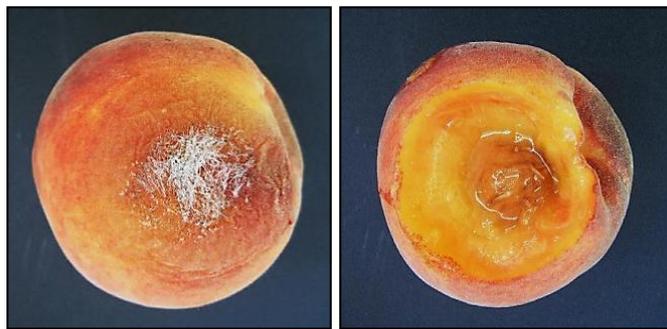


**Figure 12:** Symptoms of *Mucor* (left) bearing short spore bearing structures, compared to longer sporangiophores of *Rhizopus* (right)



**Figure 13:** *Rhizopus* rot on peaches, exhibiting an interwoven mat of fungal matter, spreading to adjacent fruit

Rotting of blossoms, or immature fruit by *R. stolonifer* and *M. piriformis* is uncommon. Fruit rot occurs mainly by wounds inflicted at harvest, or by spread to adjacent fruit by direct contact. The fungus over-winters on discarded fruit in the orchard, or on organic plant matter in the soil. *R. stolonifer* and *M. piriformis* seldom grow at temperatures of 0°C or less, but grow comfortably above 4°C. Both these fungi are rated as fast growers, macerating fruit tissue rapidly after infection. The rotted material separates easily from the sound area (Figure 14). Infected areas are soft, watery and covered with whisker-like growth. The black, spherical structures (sporangia), contain thousands of small sporangiospores (Jones & Sutton, 1996). The outer wall of the sporangia ruptures, freeing the spores, which are readily disseminated by air currents, but are also splash-dispersed by water from rotting fruit.



**Figure 14:** Whisker-like growth of *Rhizopus* rot on peaches, with the rotten material separating easily from the sound area

#### *When and how to reduce:*

*Rhizopus* and *Mucor* are most effectively controlled by storing fruit below 4°C. Careful handling of fruit, avoiding injuries, is of utmost importance. Likewise, picking and storage containers need to be clean, free from soil particles and organic matter, to avoid injury during transport and storage. Injuries from insects need to be minimised. Sanitation, by removal of decayed fruit from the orchard is of high importance to reduce inoculum and hence decay. Only a few fungicides are effective in controlling *Rhizopus* and *Mucor* rot, of which not many are registered for use. Fungicides used for the control of brown rot are normally not effective for the control of *Rhizopus*.

#### Blue mould (*P. expansum*)

##### *Infection and appearance*

Blue mould, caused by *Penicillium expansum*, is most active on fruit during storage. Infections and decay may occur in the field, especially if moist, humid weather precedes harvest. Wounding is perceived to be a prerequisite for infection. Fruit rot occurs mainly as fruit ripen, through wounds, or by spread through direct contact between sound and decayed fruit. The fungus over winters in rotten fruit and various forms of organic, decomposing plant matter. Large quantities of small spores are produced on spore bearing structures, which are readily spread by air movement.

Once an infection has established on fruit, lesion growth of *Penicillium* rot is fairly rapid, although not as fast as *Rhizopus*. Soft, watery lesions are formed. *P. expansum* grows readily at 0°C, becoming more active at higher temperatures. The fungus produces blue-green, cushion-like structures of conidia on infected fruit (Figure 15). *Penicillium* conidia are extremely resistant to drying, surviving for long periods (months) on picking and packing equipment, and in the orchard on rotting organic material. *Penicillium* spores often build up in water which is used for bulk drenching or transport of fruit in flumes. *Penicillium* rarely occurs on immature fruit in the orchard, except on fallen and damaged fruit.



**Figure 15:** Blue-green fungal growth occurring on peach and plums, associated with *Penicillium* decay

#### *When and how to reduce:*

*Penicillium* rot is reduced most effectively by harvesting fruit at optimum maturity, and by avoiding over ripeness. Stone fruit should be carefully handled to prevent injuries and potential infections. Effective cooling and storage at low temperature (< 3°C) will inhibit disease development. The main aim should be to inhibit infections, growth and spread of decay. Only a few chemicals are registered for the control of blue mould. Fungicide resistance against *Penicillium* frequently occurs. Hence, fungicide selection and the control programme, is important. Seek advice from chemical suppliers on appropriate chemicals and actions to avoid resistance. The use of alternative chemicals, or biological control agents, is likely to become more important in the future.

#### **Summary**

It is apparent from the above that many fungi could cause pre- and post-harvest fruit decay of stone fruit, with possible severe losses if not controlled. Although the disease symptoms finally differ between pathogens, there are distinct commonalities in the infection process. It is furthermore evident that pre-harvest infections, as much as infections during harvest, handling and storage, determines the development and occurrence of post-harvest decay on stone fruit, along with environmental conditions. It is essential to understand the infection process and factors affecting establishment of decay, as indicated in Part 1, related to control measures. Correct identification of the pathogen causing postharvest decay, and further understanding of the infection process, is related to the selection of an appropriate disease control strategy. Decay control and measures to counter

postharvest decay will be discussed in depth in Part 2 of the publication '[postharvest decay on stone fruit - what, when and how to reduce](#)'.

## REFERENCES

ADASKAVEG, J.E., FÖRSTER, H. and THOMPSON, D.F., 2000. Identification and etiology of visible quiescent infections of *Monilinia fructicola* and *Botrytis cinerea* in sweet cherry fruit. *Plant Disease* 84: 328-333.

ADASKAVEG, J.E., FÖRSTER, H., GUBLER, W.D., TEVIOTDALE, B.L. and THOMPSON, D.F., 2005. Reduced risk fungicides help manage brown rot and other fungal diseases of stone fruit. *California Agriculture* 59(2): 109-114.

BYRDE, R.J.W. and WILLETS, H.J., 1977. *The Brown Rot Fungi of Fruit: Their Biology and Control*. Pergamon Press, Oxford.

CAPPELLINI, R.A. and CEPONIS, M.J., 1984. Postharvest losses in fresh fruits and vegetables. Chapter 4, p24-30, in: *Postharvest Pathology of Fruits and Vegetables: Postharvest Losses of Perishable Crops*, Publication NE-87, Univ. Calif., Bulletin 1914, Berkley Agricultural Experimental Station.

CARSTENS, E., VAN NIEKERK, J.M., LAUBSCHER, W. and FOURIE P.H., 2010. Resolving the status of *Monilinia spp.* in South African stone fruit orchards. *Journal of Plant Pathology* 92: 35-41.

COLEY-SMITH, J.R., VERHOEFF, K. and JARVIS, W.R., 1980. *Biology of Botrytis*. Academic Press, Cornell University.

CONWAY, W.S., 1984. Preharvest factors affecting postharvest losses from disease. Chapter 2, p11-16, in: *Postharvest Pathology of Fruits and Vegetables: Postharvest Losses of Perishable Crops*, Publication NE-87, Univ. California, Bulletin 1914, Berkley Agricultural Experimental Station.

FOURIE, J.F., 1984. Postharvest fungal decay of stone fruit. M.Sc., University of Stellenbosch.

FOURIE, J.F., 1992. Postharvest decay of stone fruit: Infection and latency of *Botrytis cinerea*. Ph.D., University of Stellenbosch.

FOURIE, J.F. and HOLZ, G, 1995. Initial infection process by *Botrytis cinerea* on nectarine and plum fruit and the development of decay. *Phytopathology* 85: 82-87.

FOURIE, P.H., 2001. Epidemiology of *Monilinia laxa* on nectarine and plum: Infection of fruits by conidia. Ph.D., University of Stellenbosch.

JONES, A.L. and SUTTON, T.B., 1996. Diseases of Tree Fruits in the east. Michigan State University Extension.

SHOLBERG, P.L. and CONWAY, W.S., 2004. Postharvest pathology In: GROSS, K.C., WANG, C.Y. and SALTVEIT, M. (Eds.) Agriculture Handbook Number 66: The Commercial Storage of Fruits, Vegetables, and Florist and Nursery Stocks. Published online (<http://usna.usda.gov/hb66/contents.html>) by the U.S. Department of Agriculture (USDA), Agricultural Research Service (ARS), Washington DC, USA.

SNOWDON, A.L., 1990. A Colour Atlas of Post-harvest Diseases and Disorders of Fruits and Vegetables, Volumes 1, General introduction and fruits. CRC Press Inc., Boca Raton, Florida.

SOMMER, N.L., FORTLAGE, R.J. and EDWARDS, D.C., 1984. Postharvest diseases of selected commodities. Chapter 15, p117-160, in: Postharvest Pathology of Fruits and Vegetables: Postharvest Losses of Perishable Crops, Publication NE-87, Univ. Calif., Bulletin 1914, Berkley Agricultural Experimental Station.

VERHOEFF, K., 1974. Latent infections by fungi. Annual Review of Phytopathology. 12: 99-110.