REVIEW ARTICLE

Stem-end rot in major tropical and sub-tropical fruit species

K.O.L.C. Karunanayake and N.K.B. Adikaram

Stem-end rot (SER)

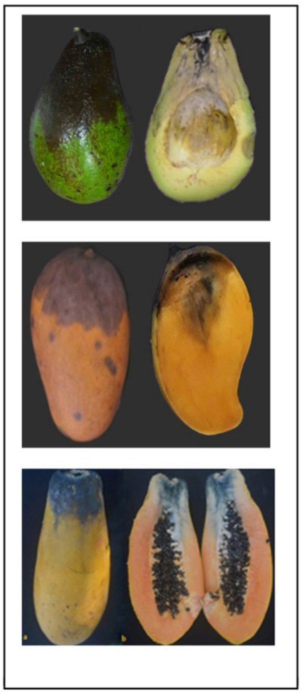
A most destructive postharvest disease affecting tropical and sub-tropical fruits during ripening.

Symptoms

A watery decay appears at the stem-end which rapidly expands through fruit pulp rotting the entire upper half of fruit.

Causal agents

SER is caused by several fungi, *Lasiodiplodia theobromae* being the most common.



Infection

In most fruit species, infection takes place in the field during flowering, fruit-set or during fruit development.

Control

Preventing field infections through cultural practices.

Protective fungicide sprays, commencing at flowering and continuing till 2 weeks prior to harvest.

Postharvest treatment with a safe fungicide, essential oil or biological control agent.

Highlights

- Stem-end rot (SER) is a most destructive postharvest disease in tropical and sub-tropical fruits.
- SER can be caused by several fungal pathogens, *Lasiodiplodia theobromae* being the most common.
- Infection occurs at flowering or fruit development. Fungi may become endophytic in the inflorescence.
- Many control options are available where reduction of field infections is key to SER control.

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REVIEW ARTICLE

Stem-end rot in major tropical and sub-tropical fruit species

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Abstract: Stem-end rot (SER) is one of the most destructive postharvest diseases affecting some tropical and sub-tropical fruits. The disease is caused by several fungal pathogens, including Botryosphaeriaceae species. In mango, *Dothiorella dominicana, D. mangiferae, Lasiodiplodia theobromae, Phomopsis mangiferae, Cytosphaera mangiferae, Pestalotiopsis* sp. endophytically colonize the inflorescence in the orchard or field. Fungi may occur endophytically in the stem tissue of the trees prior to inflorescence emergence. During the endophytic stage, the fungi colonize the phloem and xylem at stem-end and, after fruit ripening, some fungi become necrotrophic and colonize the fruit pulp causing SER. The SER pathogens in other fruit species, including ripe avocado or papaya, are not known to undergo an endophytic phase. Stem-end rot is a disease that is difficult to control. Preharvest cultural practices and selection of resistant varieties are beneficial to reduce field infections. Studies have shown that numerous control options, involving cultural, chemical (fungicides, essential oils) and biological approaches, are available to manage SER through either direct inhibition of fungi or induction of host defenses. These may be used individually or integrated manner. The most common and effective practice for controlling the SER is pre- and postharvest application of synthetic fungicides. With the new tools that are currently available, and expected to emerge, there is a possibility of a better understanding the host–pathogen or host-microbiota interactions within the stem-end region, leading to newer technologies as alternatives to toxic synthetic fungicides for the management of SER in fruits.

Keywords: Postharvest disease, *Lasiodiplodia theobromae, Neofusicoccum parvum,* Endophytic fungi, host-microbiota.

INTRODUCTION

Production of fruits for human consumption is an important part of the market economy, requiring many investments. Any waste in the field or fruits after harvest, due to physical damage, disease or disorders incurs monetary losses. In fact, the monetary loss due to damage or loss of fruits after harvest is greater than losses in the field because the cost of harvesting, storage and transport etc. must be added to the cost of production.

Reduction of postharvest losses increases fruit availability and represent a critical component of ensuring food security. Most tropical fruits are highly susceptible to postharvest diseases. A larger portion of the postharvest fruit loss could be accounted for disease caused by fungal pathogens. Fruit susceptibility to postharvest diseases increases during ripening due to physiological changes and senescence favoring disease development (Prusky *et al*., 2002). Diseases of harvested fruits and mycotoxin contamination contribute to major economic losses to grower, wholesaler, retailer and consumer. Management of postharvest diseases is needed to avoid losses and increase revenues.

Anthracnose disease, caused by *Colletotrichum* species, and the stem-end rots often incited by a complex of fungi are considered most common and important postharvest diseases that limit the long-term storage of fruit commodities. The stem-end rots are affecting mostly the tropical and sub-tropical fruits. Mango (*Mangifera indica* L.), avocado (*Persea americana*), papaya (*Carica papaya*), citrus (*Citrus sinensis*), mangosteen (*Garcinia mangostana*), rambutan (*Nephelium lappaceum* L.) and carambola are fruits often affected by the stem-end rot disease.

Stem-end rots have been investigated extensively for understanding the cause/s, the process or the mode of infection and for formulation of disease management strategies. In most fruit species, fungal infection takes place in the field during flowering, fruit-set or at an early stage of fruit maturity. Understanding of the field infection process of stem-end rot and perhaps the epidemiology also is essential to help predict the disease risk during the fruit season. Predicting plant disease is not only challenging but requires major research efforts. In a complex handling system, cultural practices, safer fungicides and biological antagonists are integrated to manage postharvest diseases. The greatest challenge is to substitute synthetic fungicides with safer alternatives to sell a highest quality, residue-free produce to the consumer.

The review will focus on the research findings so far accumulated on the stem-end rot disease in tropical and sub-tropical fruit species, with emphasis on mango, avocado, papaya, banana and rambutan, including disease management strategies.

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SER in mango

Mango (*Mangifera indica* L.) belongs to the dicotyledonous family Anacardiaceae. The genus *Mangifera* comprises of 62 species of which 15 are edible fruits. Mango originated in the Indo-Burma region and has been cultivated for at least 4,000 years in the Indian subcontinent and was spread from India to Asia, Africa, Australia, and other parts of the world by traders and missionaries (Kwee and Chang, 1985). Currently, mango is recognized as the most important fruit crop cultivated in 90 tropical and subtropical countries of the world.

Postharvest diseases of mango reduce fruit quality and cause severe losses resulting in completely unmarketable fruits. Stem-end rot is one of the most important diseases of mango posing a major threat to the industry, causing economic losses. The disease is a serious threat to the mango industry because it is difficult to control (de Oliveira Costa *et al.,* 2010). It is more prevalent in fruit harvested from older trees (Coates *et al*., 1993). In Israel, SER causes 30–40% loss of harvested mango fruit during 2014 (Diskin *et al.,* 2017a). In Sri Lanka postharvest losses of mango exceed 30 - 40% of which a larger proportion of losses is due to extensive rotting of harvested fruits. Certain mango varieties show resistance to the stem-end rot (Karunanayake *et al*., 2014). The importance of the different fungi in causing mango stem-end rot is influenced by prevailing climatic conditions in the location.

SER pathogens of mango

The predominant pathogens causing this disease are *Neofusicoccum parvum* (formerly known as *Dothiorella domanicana*), *N. mangiferae* (formerly known as *Dothiorella mangiferae*), *Lasiodiplodia theobromae* (Syn. *Botryodiplodia theobromae*), *Phomopsis mangiferae*, *Pestalotiopsis mangiferae* (Johnson *et al*., 1992) and *Cytosphaera mangiferae* (Muller and Burt, 1989; Johnson *et al*., 1991a; Johnson, 1992; Ko *et al*., 2009; Takushi *et al*., 2017; Hong *et al*., 2012; Karunanayake *et al*., 2014) and other fungi. *Botryosphaeria dothidea* is the major pathogen causing stem-end rot of mango in Brazil, which brings about significant losses during transportation and storage (Terao *et al*., 2018). *Colletotrichum gloeosporioides, Alternaria alternata, Fusicoccum aesculi, Nattrassia mangiferae, Botryosphaeria* spp. and *Botrytis cinerea* were linked to SER in mangoes (Ploetz *et al*., 1994; Abdalla *et al*., 2003; Slippers *et al.,* 2005; de Oliveira Costa *et al*., 2010). Using morphological characteristics and DNA sequence data, the presence of *Lasiodiplodia theobromae* (Fig, 2) *Fusicoccum aesculi* and *Neofusicoccum parvum*. *L. theobromae* were confirmed in the Assú Valley, Brazil. *F. aesculi* and *N. parvum* were in the São Francisco Valley. In fruit inoculations, *L. theobromae* and *N. parvum* were more virulent than *F. aesculin* (de Oliveira Costa *et al*., 2010).

Lasiodiplodia theobromae, *Dothiorella* spp. *C. gloeosporioides*, *Phomopsis mangiferae* and *Pestalotiopsis mangiferae* were identified as stem-end rot (SER) pathogens in mango in Sri Lanka with two more Ascomycota species namely *Xylaria* sp. and *Nodulisporium* sp. from the cv. ‘Karuthacolomban’ (Figure 1) in a more recent study using ITS sequence data (Karunanayake *et al*., 2014; Ekanayake *et al*., 2019). Botryosphaeriaceae and Diaporthaceae species, have been reported as pathogens of stem-end rot disease around the world, including *Botryosphaeria dothidea*, *Diaporthe pseudomangiferae*, *Neoscytalidium dimidiatum* (Marques *et al*., 2013; Serrato-Diaz *et al*., 2014). *Diaporthe* species were consistently isolated from mangoes showing stem-end rot in Japan (Ajitomi *et al*., 2020). The isolates reproduced the original symptoms in healthy mango fruits after inoculation and were re-isolated from the inoculated fruits. Based on morphology and a phylogenetic analysis of rDNA-ITS, CAL, H3, EF1-α, and TUB, the isolates were classified into two *Diaporthe* species (Clade I and Clade II) (Ajitomi *et al*., 2020).

SER in avocado, papaya and rambutan

The stem-end rot is recognized as a major postharvest disease in ripe avocado (*Persea americana* Mill.) fruit. Unripe fruits are usually free from visible symptoms or characteristic decay lesions develop during fruit ripening.

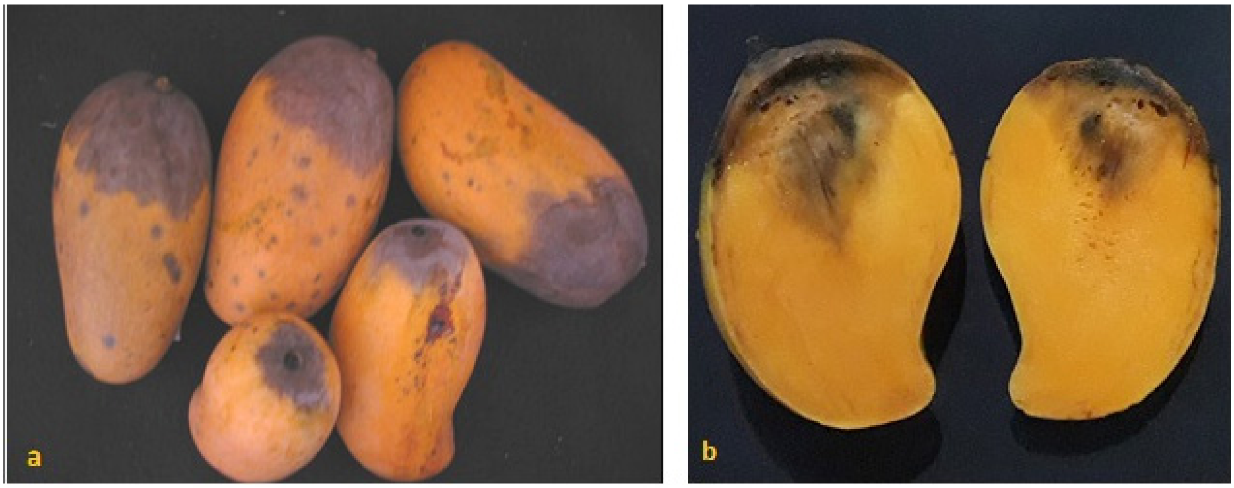

Figure 1: (a) External symptoms of SER on ripe mango fruit, and (b). A vertically halved mango fruit showing internal pulp symptoms due to SER.

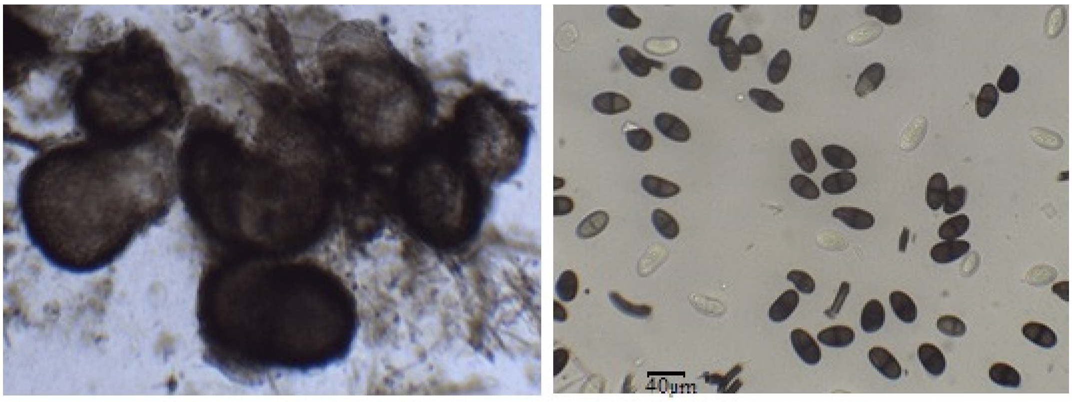

Figure 2: *Lasiodiplodia theobromae*, a major SER pathogen, pycnidia (left) produced in 4-week old PDA medium, and conidia (right), mature conidia black in color with a septum, and younger conidia, hyaline and aseptate.

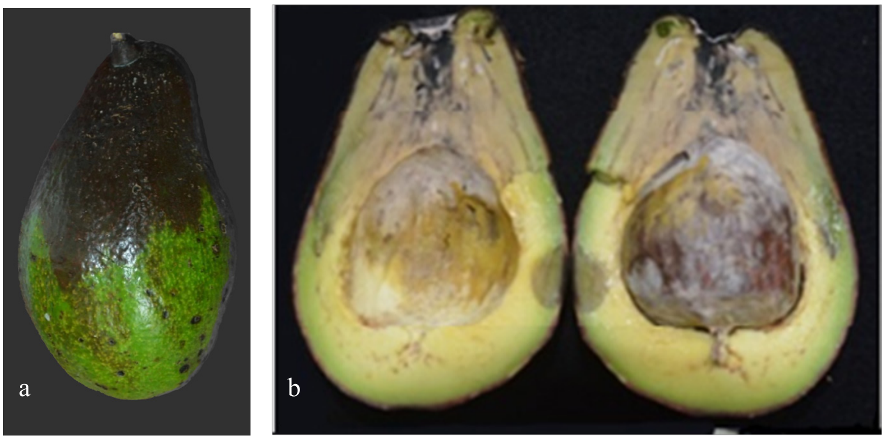

Figure 3: (a) External symptoms of SER on ripe avocado fruit, and (b). A vertically halved avocado showing internal pulp symptoms due to SER.

Figure 4: (a) Ripe papaya fruit showing SER symptoms due to *L. theobromae* infection, shrunk stem-end with black color outer mycelial growth, and (b) Vertically halved papaya fruit showing a large area of softened pulp and blackish fungal growth.

Stem-end rot in avocado (Fig, 3) is caused by several fungi including *Lasiodiplodia theobromae* (Madhupani and Adikaram, 2017), *Pestalotiopsis* sp., *Phomopsis perseae* and *Phoma* spp. (Adikaram and Karunarathne, 1998) and occasionally by *Colletotrichum acutatum* as reported in New Zealand (Everett *et al*., 2005). The disease can result in heavy postharvest losses. Different causative agents are also reported to be associated with the disease. The fungus most commonly associated with SER in `Fuerte' avocado was *Thyronectria pseudotrichia* whereas in the variety `Edranol', it was *Dothiorella aromatica*. However, *D. aromatica* was found as the major causative agent of avocado SER in Australia (Darvas, 1981). SER of 'Hass' avocados in New Zealand is caused by *Botryosphaeria* *parva* and *Phomopsis* spp. (Hartill and Everett, 2002). However, the pathogen most predominantly found causing the disease is *L. theobromae*.

Stem-end rot of papaya (*Carica papaya* L.) was initially attributed to *Ascochya* sp. (Hine *et al*., 1965). Later certain other genera including *Botryodiplodia* (Hunter and Buddenhagen, 1972; Abeywickrama *et al*., 2012), *Phomopsis* and occasionally *Fusarium* were found in diseased fruits (Hunter and Buddenhagen 1972). A stem-end disease of papaya (*Carica papaya* L.) was reported to be caused by *Mycosphaerella* sp., the perfect stage of *Ascochyta caricae-papayae* (Chau *et al*., 1979). Initial symptom was a translucent zone that appears around the peduncle which follows slight browning of the region. When the disease advances the infected tissues become black, wrinkled and dry. White mycelium forms at the stem end.

Botryodiplodia theobromae also causes stem-end rot disease in papaya. The pathogen produces a wider margin of water-soaked tissue (Figure 4) and a rough surface caused by an irregular pattern of erumpent pycnidia (Hunter and Buddenhagen, 1972). Later, the affected portion becomes shriveled and turns dark brown. The disease is found in certain papaya varieties in Sri Lanka. Stem-end rots of papaya occur when fungi invade the severed peduncle after harvest. Spores may invade through crevices between the peduncle and the papaya flesh or invade through small wounds that occur at harvest.

Rambutan (*Nephelium lappacium* Linn.) is a fruit native to South East Asia. The fruits are affected by postharvest stem-end rot caused by *Gliocephalotrichum microchlamydosporum.* The disease is reported from countries in Asia including Philippines, Thailand and Sri Lanka (Sivakumar *et al*., 1997). A dark brown lesion appears in the entire fruit 4-5 days after harvest. During latter stages of the disease, a dark color hyphal mass is developed on diseased tissues. The symptoms are most often observed in the stem-end region of the fruit. The flesh becomes soft, brown and semi-liquid (Sivakumar *et al*., 1997). The skin is softened, wrinkled and black in color with encrusted pycnidia of the fungus. *G. microchlamydosporum* infections take place only when a wound is present. Harvesting fruits with the stalk and avoidance of wounds on the fruit surface during harvest or handling could prevent the disease (Sivakumar *et al*., 1997).

Stem-end rot in ripe banana fruits is a minor disease and has not been reported from many regions in the world. The disease was recorded in Bangladesh (Rashid *et al*., 1999) and Thailand (Rattanakreetakul 2013) and the causal agent was identified as *Lasiodiplodia theobromae* (Syn. *Botryodiplodia theobromae*) in both the countries (Rashid *et al*., 1999; Rattanakreetakul, 2013). There is not much published information available on the disease. Synthetic volatile oils, linalool and eugenol, were found to be very effective in reducing the stem-end rot development (Rattanakreetakul, 2013) than the essential oils extracted from finger root (*Bosenbergia rotunda*) and lemon grass (*Cymbopogon nardus*) (Rattanakreetakul, 2013).

A fungal rot, observed in the rhizome of banana, caused by *Ceratocystis paradoxa*, was also described as a stem-end rot. The rot forms brown color or water-soaked patches in the rhizome. The fruit stalks decay rapidly, the tissues become soft and blackened and have a characteristic sweet smell. The pathogen spreads to the fruit where it causes uneven black discoloration of the skin. The pulp is reduced to a dark brown soft wet mass and premature ripening of fruits.

Endophytic colonization by SER pathogens

SER in mango is caused by diverse pathogenic fungi that endophytically colonize the inflorescence in the orchard or field. *Dothiorella dominicana, Dothiorella mangiferae, Lasiodiplodia theobromae* (Syn. *Diplodia natalensis*, *Phomopsis mangiferae, Cytosphaera mangiferae, Pestalotiopsis* sp. and *Dothiorella* 'long'), as well as other fungi, including *Alternaria alternata*, associated with the stem-end rot disease in mango, were found to occur endophytically in the stem tissue of mango trees prior to inflorescence emergence (Johnson *et al*., 1992). Endophytic colonization of inflorescence and pedicel tissue was found to be a primary route of infection for fruit which develops stem-end rot during ripening (Johnson *et al*., 2008). They enter the stem tissues through natural openings and wounds, mainly during inflorescence and flowering stages.

During the endophytic stage, the fungus colonizes the phloem and xylem of the fruit at stem-end and after fruit ripening, the fungus turns in to a necrotrophic lifestyle and starts colonizing the fruit parenchyma causing SER. Not all fungi present in the stem-end make the transition from endophytic to necrotrophic lifestyle and become pathogenic during fruit ripening. However, little is known about the fruit stem-end endophytic microbiome, which could contain new and existing biocontrol agents (Galsurker *et al*., 2018). At a plantation with a history of high stem-end rot levels, sequential monitoring of inflorescence tissue between flowering and harvest by plating out tissue pieces revealed that some of the pathogens, *Dothiorella* spp., *P. mangiferae, Pestalotiopsis* sp. and *C. mangiferae,* gradually colonised the inflorescence, reaching pedicel tissue of young fruit 8 weeks after flowering (Johnson *et al*., 1992).

During flowering and fruit set, colonization by *A. alternata, Cladosporium cladosporioides, D. dominicana, D. mangiferae, Epicoccum purpurascens*, *Dothiorella* sp. and *Pestalotiopsis* sp. is increased in Australia as the flowers undergoing senescence and young fruits are formed (Johnson *et al*., 1991a). *Lasiodiplodia theobromae* was isolated at high rates from flowers, peduncles, fruit-bearing shoots, and fruiting mother shoots in Japan (Takushi *et al*., 2019)

In the third week after flowering, the incidence of *D. dominicana* and *D. mangiferae* with the fruit-pedicel connection tissue of mango declined in coincidence with early fruit-fall, suggesting that early infections by *Dothiorella* spp. may cause fruitlet abortion (Johnson *et al*., 1991b). After remaining low levels for the next six weeks, the incidence of *Dothiorella* spp. at 16 weeks after flowering, determined by isolation from fruit-pedicel connection tissue, was similar to the incidence of stem-end rot, caused by *Dothiorella* spp., which developed in fruit harvested at that time. The results suggested that in fruit and fruit pedicel tissue, colonization might arise from *Dothiorella* spp. occurring endophytically in the peduncle. Using larger tissue pieces (1–2 mm thick TS or tissue 25 mm^2 x 3 mm) in isolations, endophytic colonization by *Dothiorella* spp. and *P. mangiferae* was detected in stem, inflorescence and pedicel tissues of mature fruit specimens from two different sites, one unsprayed, and the other regularly sprayed with copper. The fungi were detected more frequently in the samples from unsprayed trees (Johnson *et al*., 1992).

The earliest indicator of stem-end rot incidence at harvest was the infection level in peduncle tissue sampled 11 weeks after flowering (Johnson *et al*., 1991b). As the fruit ripens, some pathogenic fungi switch from endophytic colonization to necrotrophic stage and cause stem-end rot (Galsurker *et al*., 2020). Stem-end rot fungi are also found as endophytes in host tissue and can cause twig and branch dieback, cankers and fleck-spots on inflorescence and stem tissues.

Hartill and Everett (2002) isolated SER pathogens from dead twigs and branches of avocado but appeared to be present as saprophytes and there was no evidence that they had killed the tissues. There are no records of *B. theobromae* to be an endophyte on avocado or papaya fruit.

Symptoms

The development of watery decay in ripe mangoes begins to appear around the stem-end which would rapidly expand through the pulp tissues, softening the entire upper half of the fruit (Figure 1), depending on the cultivar. The colonization extends ahead of symptoms along vascular tissue while colonization progresses into the fruit flesh and the seed coat. The symptoms produced during infection also vary with the pathogen. Fast moving dark lesions are produced by *L. theobromae* and Botryosphaeraceaeous fungi. Steel grey mycelium can develop over the surface on fruit affected by *Neofusicoccum* spp. Pycnidia may develop around the stem end particularly with infections caused by *N. parvum* (formerly *D. dominicana*) and (Johnson *et al*., 1991a). Symptom development is slower with *Phomopsis mangiferae* or *Cytosphaera mangiferae* and a dark lesion of more uniform radius develops at the stem end. Superficial mycelium does not usually develop (Johnson *et al*., 1991a). The occurrence of SER caused by *P. mangiferae* can be more frequent in fruit from dry areas.

In avocado also the SER symptoms develop only after harvest when the fruit starts to ripen. The earliest sign of infection is a slight shriveling around the stem button in the ripening fruit, and fungal mycelium may often be visible on the abscission scar when the button is removed. Conspicuous dark rot with a well-defined margin develops downwards from the stem-end. Rotting extends further with ripening covering most of the fruit which becomes shriveled. Affected fruit flesh becomes soft and pale in color (Figure 3), and the vascular strands running vertically turn brown to black.

Subsequently, rot develops from the stem-end of the fruit. Externally, the affected tissue becomes darkened and the area of rot has a well-defined and advancing margin. Internally, the flesh becomes darker, soften and the vascular strands may discolor in advance of the rotting flesh (Darvas, 1981).

Stem-end microbiome

Traditionally, Plant Pathologists have focused on the species of the pathogen and its function at a time. In recent times, studies have been broadened to the concept of 'microbiome', the entire community of microorganisms within a habitat and its surrounding environmental conditions. A holistic approach is endorsed to explore how microbiomes influence the ecosystem and pathosystem function and their dependence on each other because of its complex nature.

The microbiome consists of all the microorganisms (fungi, yeasts and bacteria) inhabiting the plant tissue. The advances in high-throughput sequencing of DNA have been applied to study the plant microbiome. In apples, variety and rootstock modulate the endophytic microbiome, suggesting coevolution of a specific genotype with its microbiome (Liu *et al*., 2018). When these methods were used to study the mango stem-end microbiome during postharvest storage and disease development, healthier stem-ends (with a lower incidence of SER) showed more diverse microbial communities (Diskin *et al*., 2017b). In contrast, an increase in SER incidence was correlated with a reduction in microbiome diversity and an expansion of one or several fungal pathogen families, such as Pleosporaceae and Botryosphaeriaceae. In addition, the increase in fungal abundance and SER was correlated with an increase in the chitin-degrading, Chitinophagaceae bacteria and a reduction in biocontrol agents (Diskin *et al*., 2017b). This implies that the stem-end microbiota is a dynamic system that can be modified to control SER-causing pathogens.

Today, the genomes of most of the hosts and SER pathogens are available, which will enable transcriptome analysis that could open new insights for better understanding the host defense response and the switching of pathogenic fungi from endophytic to pathogenic stage. This transcriptome analysis could lead to the development of new control methods.

Management of SER

SER can be controlled by good field sanitation, optimal cultural and harvesting practices coupled with prompt cold storage after harvest. In warm and humid tropics, however, the use of refrigeration is costly. Most of the commercially available synthetic fungicides are toxic and can bring about adverse effects on human health and environment. Therefore, due to consumer concerns, alternative methods aimed at preventing, suppressing, or delaying disease symptoms have been investigated (Sommers, 1985). More attention is therefore required to be focused on other control methods. In situations where the SER is a serious problem, the use of synthetic fungicides may be necessary.

Chemical control

Control of SER has been traditionally achieved using synthetic fungicides (Singh and Sharma, 2007) which can be applied preharvest or at postharvest stage. Considering the fact that most SER pathogens infect the flower first (Johnson *et al*., 1992; Diskin *et al*., 2017a) and later invade the fruit pedicel residing endophytically, targeting of the flowering stage for application of fungicides seems appropriate. Although environmentally unsound, the more common practice for controlling SER is by postharvest fungicidal treatments.

Good control of avocado stem-end rot was achieved with the fungicides that contain Ethyl hydrogen phosphonate and Copper oxychloride as active ingredients (Darvas, 1981). Preharvest spray application of copper oxychloride, combined with commercial dicarbamic fungicides, containing manganese and zinc, from flowering until harvest, controls most postharvest diseases of mango (Lonsdale and Kotze, 1993). Diskin *et al*. (2017) also report that, application of the chemical fungicide that contain the active ingredients fluopyram and pyrimethanil or fludioxonil and cyprodinil during flowering, significantly reduces the incidence and severity of postharvest SER and side decay in mango fruits as it reduces early colonization by pathogenic fungi.

However, Peterson *et al*. (1991) state that a significant reduction in SER was not achieved by preharvest fungicide treatment. Benzimidazole fungicides, are effective against SER caused by *Lasiodiplodia theobromae* on mango, whereas imidazoles are not (Estrada *et al*., 1996, Plan *et al*., 2002). Dipping mango fruits in warm carboxamide fungicides has been found effective (Johnson *et al.,* 1990; Sangchote, 1991; Swart *et al*., 2006) or partially effective (Muirhead *et al*., 1982) in the past. In a comparative study, the efficacy of six commercial fungicides was assayed by artificially inoculation of mango with *L. theobromae*. This study showed that benzimidazoles fungicides were the most effective in inhibiting the mycelial growth of *L. theobromae* (Syed *et al*., 2014).

The use of some of the fungicides mentioned above in early literature was banned from use globally in 2001 (List of banned pesticides and pesticide watch list, 2015) or are to be banned from Europe at the end of 2021 (Shimshoni *et al*., 2019). Recent work suggests that azoxystrobin fungicides or electrolyzed oxidizing water instead of fungicides are effective in controlling SER of avocado (Hassan and Dann, 2019). Prochloraz fungicides are to be banned at the end of 2021 from Europe due to their unfavourable toxicological properties and Fludioxonil, a relatively safer, wide spectrum fungicide has been found to be a suitable alternative for avocado (Shimshoni *et al*., 2019) and as a postharvest treatment for mango.

Essential oils with fungicidal properties

Plant extracts have gained much importance in recent years as safe and effective alternatives to synthetic fungicides. *Moringa oleifera*, *Syzygium aromaticum* and *Cinnamomum zeylanicum* (Alam *et al*., 2017) extracts of *Datura stramonium* and *Eucalyptus camaldulensis* (Ullah *et al*., 2017), basil oil (*Ocimum basilicum*), cardamom oil (*Elettaria cardamomum*) and citronella oil (*Cymbopogon nardus* L.) (Kulasinghe *et al*., 2019) have shown significant antifungal activity against mycelial growth of SER pathogens and also a reduction of SER development in mango. Dip treatment of mango fruits cv ‘Karuthacolomban’ *in vivo* with cardamom oil in warm water (45 ˚C) significantly reduced SER development with little effect on sensory properties (Kulasinghe *et al*., 2019). Similarly, spray and fumigation treatments using basil, clove, cinnamon leaf and cinnamon bark oils, followed by storage at 12 – 14 ºC for 8 days, enabled effective control of the SER of mango cv ‘Karuthacolomban’ (Kodituwakku *et al*., 2020). Similarly, another study involving fumigation treatment with thyme oil, clove oil and cinnamon oil vapors resulted in inhibition of *Colletotrichum gloeosporioides* and *L. theobromae* growth *in vitro* (Perumal *et al*., 2016). Papaya cultivars, ‘Rathna’ and ‘Red Lady’, stored at 12-14 ˚C following treatment with 1% (w/v) alum (sodium aluminium sulphate) or 1% alum and 0.16% (v/v) basil oil and enclosed in styrofoam sleeves were found free of postharvest diseases including SER (Abeywickrama *et al*., 2012). No residues of basil oil could be detected on fruit peel by gas chromatography at the end of the experimental period of 14 days (Abeywickrama *et al*., 2012).

Biological control

Biocontrol is another less common yet environmentally friendly method of controlling SER. Application of a cell suspension of *Aureobasidium pullulans* at the stem-end of harvested unripe avocado fruit delayed incidence of stem-end rot by two days compared to controls which had either stalk intact or not. Retention of stalk reduced the progression of symptoms in both treated and control avocado fruits but did not significantly affect disease incidence. *Aureobasidium pullulans* reduced conidia germination and mycelial growth of *L. theobromae*, (Madhupani and Adikaram, 2017). Normally, chitinase, β-1,3-glucanase and antifungal activity of the peel of avocado fruit (control) declined during ripening. In *A. pullulans*-treated fruits, however, the activity of chitinase and β -1,3-glucanase was increased when fruits were inoculated with *L. theobromae* after treatment with *A. pullulans*. β-1,3-glucanase activity increased only slightly in fruits that was inoculated with *L. theobromae* without treatment. Greater preformed antifungal activity was retained in *A. pullulans*-treated fruits during ripening. Enhanced activity of chitinase and β-1,3-glucanase and greater retention of preformed antifungal activity may have contributed to the delayed stem-end rot incidence in *A. pullulans*-treated avocados. Application of *A. pullulans*, two days prior to inoculation and retention of stalk at harvest, allowed better establishment of *A. pullulans* on the treated site (Madhupani and Adikaram, 2017).

Pestalotia neglecta, a non-pathogenic surface-inhabitant, most commonly encountered on the avocado fruit, exhibited strong antagonistic properties against

Phoma species, a major stem-end rot pathogen in Sri Lanka. Application of conidia of *P. neglecta* on to the stem-end region of harvested avocados, pre-inoculated with *Phoma* species, delayed stem-end rot development by four days, compared to the control. A similar effect was not observed with *B. theobromae. In vitro* studies confirmed that *P. neglecta* was antagonistic to *Phoma* species, but not *B. theobromae*. The antagonism appears to be due to a heat-labile extracellular factor. Dipping avocado fruits in a suspension of conidia of *P. neglecta* delayed both anthracnose and stem-end rot development (Adikaram and Karunaratne, 1998).

Bacillus subtilis (isolate B246), *Bacillus cereus* and *Bacillus licheniformis* (isolate B248) were inhibitory to avocado postharvest pathogens. *B. cereus* isolate B249 actually stimulated development of *Lasiodiplodia theobromae*. Diffusible metabolites from the four antagonists impeded growth of *C. gloeosporioides, D.aromática, P. perseae, L. theobromae*, but not of *T. pseudotrichia. B. subtilis* (B246) mode of action was determined using an *in vitro* spore-germination assay. *In vitro* the antagonist effectively reduced percentage spore germination of *C. gloeosporioides, D. aromática, P. perseae, L. theobromae, F. solani, P. versicolor* and *D. setariae*. In addition, bulb formation and lysis of hyphae were observed, as well as bacterial movement towards germinating spores and subsequent attachment to the spores (Korsten and Jager, 1995; Korsten *et al*. 1988; 1997).

When applied in the field, *B. subtilis* was found to get attached to and colonize avocado flowers and interfere with SER-causing pathogen colonization (Demoz and Korsten, 2006). Postharvest application of *B. licheniformis* reduced mango anthracnose and SER (Govender *et al.,* 2005) and *Trichoderma viride* was reported to control SER caused by *L. theobromae* in mango fruit (Kota *et al*., 2006).

Trichoderma harzianum isolated from soil of Rambutan orchards were found to be effective biocontrol agents against *Botryodiplodia, Colletotrichum* and *Gliocephalotrichum* spp. causing postharvest diseases of Rambutan in Sri Lanka (Sivakumar *et al*., 2012).

Field management and postharvest practices

There is a variation in susceptibility to SER among cultivated mango varieties. Studies done in Thailand reveal that cultivars ‘Okrong’ and ‘Tong dum’ had higher disease incidence compared to ‘Khaew’ (Sangchote, 1991). This variation in susceptibility is due to the higher sucrose content in susceptible cultivars (Sangchote, 1991). Karunanayake *et al*. (2014) report that there is a correlation with antifungal compounds; especially those in mango latex that contribute to cultivar differences in susceptibility to SER. Chitinase activity was highest in cultivars ‘Rata’ and ‘Kohu’ that are resistant to SER and lowest in cultivars ‘Gira’ and ‘Karuthacolomban’ that are susceptible to SER (Karunanayake *et al*., 2014). Further, this is possibly the underlying reason for reduced SER in fruits that are not de-sapped after harvest. The simple practice of harvesting fruits with intact pedicels as opposed to detaching the fruit pedicel at its base which the common practice had a major and significant impact on reducing SER incidence in mango and avocado fruit (Diskin *et al*., 2017a; Sangchote, 1991). A negative correlation was observed between pedicel length and SER development in ‘Karuthacolomban’ mangoes (Karunanayake *et al*., 2015). Fruits harvested with pedicel, thereby retaining the latex, developed SER later compared to the fruits which were detached from the pedicel. Sangchote (1991) reported similar findings where fruits with longer pedicels developed SER later and those with longer pedicels had the lowest disease incidence opposed to those without pedicel that had the highest disease incidence. The fruit peel in which latex was retained had greater chitinase activity. The reduction of anthracnose and SER could be due to the greater antifungal resorcinols and chitinase activity respectively in latex-retained fruit (Karunanayake *et al*., 2015). Similarly, ‘Kensington Pride’ mango fruit stored with 2- to 3-cm long stems had significantly more resorcinol in their peel and smaller anthracnose lesions than de-sapped fruit (Hassan *et al*., 2007). Therefore, it seems that when harvesting fruit with stems, more sap that contains antifungal compounds is left in the fruit stem and peel, leading to decreased postharvest side decay and SER (Galsurker *et al*., 2018).

Applying high doses of potassium fertilizer to the soil, during fruit development significantly reduced SER development in mango fruit cv. ‘Karuthacolomban’ at the postharvest stage. The effective dose of fertilizer was 2055 g x 3/ tree/ year which was three times the recommended dose (Karunanayake *et al*., 2016). The elicitors Salicylic acid (Karunanayake, 2008) and Kasil (Sinniah, 2010) were more effective in controlling anthracnose than SER of mango fruit.

A combination of hot water with gamma irradiation at the postharvest stage significantly reduced mango SER and anthracnose (Spalding and Reeder, 1986). UV-C controls fungal pathogens, including SER-causing pathogens (Stevens *et al*., 1996) by inducing fruit resistance (Romanazzi *et al*., 2016). Similarly, red mango fruit that was exposed to sunlight in the orchard accumulated anthocyanin and was more resistant to SER than fruit that developed within the tree canopy (Diskin *et al*., 2017a). Therefore, pruning and exposure of fruit to sunlight could be a good method for reducing postharvest SER. Hot-water dips and different heat-treatment such as rinsing, hot vapor and dry-air treatments have been suggested to reduce postharvest diseases including SER of mango (Alvindia and Acta*.,* 2015; Coates *et al.,* 1993).

Considering the mode of infection and colonization, a combination of control measures is needed to be employed in the control SER, if fungicides are not used. These practices should commence at the time of flowering and continue through fruit development to the postharvest stage until it reaches the consumer especially in fruits such as mango.

DECLARATION OF CONFLICT OF INTEREST

The authors declare no conflict of interest.

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