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Latent postharvest pathogens of pome fruit and their management: from single measures to a systems intervention approach

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Abstract Postharvest diseases of pome fruit are typically caused by a wide diversity of fungal pathogens, and the list of confirmed causal agents is still growing. There is considerable knowledge on the epidemiology of wound pathogens, such as *Botrytis cinerea* and *Penicillium expansum*. In contrast, knowledge on the occurrence of the different postharvest diseases caused after latent (quiescent) infections during long-term storage and their epidemiology is limited. Well-known pathogens causing postharvest losses after latent infections are *Neofabraea* spp. and *Colletotrichum* spp., but in many cases the causal agents that occur in a specific region remain unknown and their control relies on the routine use of fungicide applications. However, due to the growing concern over the use of synthetic fungicides, alternative control measures are highly desired. Over the past years the use of physical treatments, natural compounds, and biocontrol agents have been investigated as alternatives. However, no single method has emerged that can robustly and reliably control postharvest diseases of pome fruit in practice. In this review it is argued to approach latent postharvest diseases as complex problems that require multiple interventions at different stages of the disease process in a systems intervention approach for their control. Such approach requires a deep understanding of the epidemiology of the causal agents in the orchard, fruit defence mechanisms against pathogens, and the molecular biology of host-pathogen interactions in order to develop novel disease control methods in which the deployment of resistant cultivars can be a cornerstone.

Keywords Postharvest diseases · Fruit–fungal interaction · Pathogenicity · Quiescence · Control methods

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General introduction

Apples and pears (pome fruit) are important deciduous fruit species cultivated on a worldwide scale. Mild and humid climatic conditions, such as those prevalent in North Western Europe, favour fungal diseases on pome fruit, such as apple scab (*Venturia inequalis*), brown spot of pear (*Stemphylium vesicarium*), European fruit tree canker (*Neonectria ditissima*), and postharvest fruit rots.

Production and storage of pome fruit in the Netherlands

Apple (*Malus domestica*) and pear (*Pyrus communis*) are important fruit crops that are cultivated in the Netherlands. The main apple cultivar is Elstar grown on 40% of the total apple production area, while the main pear cultivar is Conference grown on 75% of the pear production area (CBS 2016). After harvest, fruit are stored

for up to 11 months under specific controlled atmosphere (CA) conditions, such as ultralow oxygen (ULO) and dynamic controlled atmosphere (DCA), depending on the cultivar and volume to be marketed (Thewes et al. 2015; Van Schaik and Verschoor 2003). However, as fruit are typically stored for extended periods of time, postharvest diseases have become a limiting factor of significant concern.

Postharvest diseases of pome fruit

Postharvest diseases of pome fruit result in significant economic losses during storage worldwide every year. They are typically caused by a variety of fungal pathogens, although also bacterial and oomycete postharvest pathogens can occasionally occur. Despite technological advances in postharvest handling of fresh fruit, postharvest fruit losses range from 5 to 20% with upwards of 50% on susceptible cultivars (Janisiewicz and Korsten 2002; Jones and Aldwinckle 1991; Jurick II et al. 2011). For example, bull's eye rot (*Neofabraea* spp.) is the main disease of stored apples in Poland, causing up to 30–40% of postharvest losses on susceptible apple cultivars (Michalecka et al. 2016). Similarly, bull's eye rot and bitter rot (*Colletotrichum* spp.) have been reported to cause up to 30% decay during storage of organically grown apples in northern Germany (Maxin et al. 2014).

Postharvest diseases of apple and pear are caused by a range of fungal pathogens (Sutton 2014). Wounds caused by insects and birds, as well as by physical damage that is inflicted before or during harvest, are important entrance sites for pathogens such as *Botrytis cinerea*, *Penicillium expansum* and *Monilinia fructigena*, the causal agents of grey mould, blue mould, and brown rot, respectively (Snowdon 1990). These pathogens typically cause rapid decay of fruit in the pre- and postharvest stage. Fungicide applications shortly before harvest and careful handling of fruits during harvest are effective measures to significantly reduce losses by these wound pathogens.

However, another group of pathogens infects developing, unwounded fruits during the growing season. After infection these pathogens remain quiescent, i.e. without causing symptoms during the growing season. In epidemiology, there is a difference between latent and quiescent infections. Briefly, a latent infection is a non-symptomatic infection, whereas a quiescent infection is an incipient visible infection (Jarvis 1994; Verhoeff 1974). This implies the quiescence is the passage from symptomless internal infections (i.e. a latent infection) to visible but non-expanding lesions, due for example to environmental or physiological and biochemical changes (De Silva et al. 2017; Prusky et al. 2013). In this review we use the term latent synonymously with quiescent. Thus, after several months in CA storage symptoms start to appear, when certain physiological or biochemical cues in the host are changed (Coates and Johnson 1997; Lattanzio et al. 2001). Examples of fungal pathogens causing postharvest losses are *Neofabraea alba* (Chen et al. 2016; Soto-Alvear et al. 2013), *Neonectria ditissima* (Weber and Dralle 2013), the *Colletotrichum acutatum* species complex (Spolti et al. 2012), *Cadophora malorum* (Sugar and Spotts 1992), *Phytophthora* spp., *Alternaria* spp., and *Fusarium* spp. (Sever et al. 2012). Moreover, novel latent postharvest pathogens are described continuously, such as *Phacidium lacerum* (Wiseman et al. 2016), *Sphaeropsis pyriputrescens* and *Phacidiopycnis washingtonensis* (Kim and Xiao 2008; Weber 2011; Xiao and Kim 2008). Postharvest pathogens are able to pass or overcome the natural defence systems that operate in fruit (Alkan et al. 2015). They infect through wounds, direct penetration of intact tissue, or colonization of natural openings such as lenticels, stems, and pedicels (Prusky and Lichter 2007). Fruit maturity has been implicated in the susceptibility of apples to particular fruit rot diseases. Brook (1977) observed that apples did not show symptoms of bitter rot caused by *C. gloeosporioides* until fruit were approaching maturity. Similarly, increasing maturity in apples resulted in higher incidences of bull's eye rot caused by *Neofabraea alba* (Edney 1964) and also blue mould caused by *P. expansum* (Vilanova et al. 2014). The increased disease incidence towards the end of a growing season has been hypothesized to be due to changes in the availability of natural openings in response to fruit maturity (Aguilar et al. 2017). For instance, during fruit maturation changes in mineral content but also environmental factors may affect the breakdown of lenticels (Turketti et al. 2012). Alternatively, the increased susceptibility could be due to fruit maturation-related degradation of phenolic compounds that inhibit fungal growth during fruit maturation (Edney 1964). Interestingly, a recent study of Everett et al. (2018) has shown that the incidence of infection of 'Royal Gala' apples by *C. acutatum* was related to temperature rather than to maturity of the fruit. However, in this case, only late in the 'Royal Gala' cultivation season the mean daily

temperatures exceeded 15 °C, so temperatures that are permissive for infection only occurred when fruit were more mature. Also high nitrogen (N) content in fruit has been implicated in the incidence of bull's eye rot, blue mould and brown rot on apple fruit (Lysiak 2013; Sharples 1985), potentially due to weaker cell walls and thus greater sensitivity to fungal pectolytic enzymes (Bateman and Basham 1976).

Because of their complicated biology, which involves an enigmatic switch from a quiescent to a symptomatic stage, latent postharvest pathogens are poorly understood and their control is challenging. In this review we focus on latent postharvest pathogens that are responsible for late postharvest losses of pome fruit and discuss how these pathogens can be controlled.

Specific latent postharvest pathogens

As stated, a growing list of fungi is reported to be associated with latent postharvest fruit rots of pome fruit. In order to develop effective control strategies, it is necessary to assess which are the most important latent postharvest pathogens that occur in a specific region on the crop. Based on the current literature the economically most important latent postharvest pathogens in most apple and pear growing areas are *Colletotrichum* spp. and *Neofabraea* spp.

Colletotrichum spp.

Colletotrichum species are considered as major pathogens associated with pre- and postharvest fruit diseases (Alaniz et al. 2015; Cannon et al. 2012; Dean et al. 2012; Phoulivong et al. 2010). Apple bitter rot caused by *Colletotrichum* spp. is a widespread fruit disease occurring in most countries where apples are cultivated (Shi et al. 1996). *C. acutatum* species complex (SC) infections on apples in Europe are frequently reported with increasing numbers of recent reports from Belgium, England, Italy, France, Norway and Slovenia (Børve and Stensvand 2015; Grammen et al. 2019; Mari et al. 2012; Munda 2014; Munir et al. 2016; Nodet et al. 2016). Studies from Germany and Sweden describe postharvest losses of apple fruits of 10 and 25%, respectively, by *C. acutatum* SC (Børve and Stensvand 2017; Weber and Palm 2010).

In warmer climates, *C. acutatum* SC infections lead to symptoms on apples during the summer growing period while the fruits are still on the trees. However, in cooler growing areas *C. acutatum* is more commonly observed as a latent storage pathogen (Everett et al. 2018). Disease symptoms of bitter rot are characterised by the development of small dark brown spots eventually expanding to light brown sunken lesions. Afterwards conidia are formed in acervuli concentrically in the centre of the lesion (Damm et al. 2012). All apple cultivars are susceptible to bitter rot, and in particular those belonging to the late-harvest group, such as Granny Smith, Pink Lady, and Fuji (Velho et al. 2015). It is considered that apple bitter rot has a higher destructive potential than other apple rots and can result in losses up to 50% at pre- and postharvest stages (Everett et al. 2015; Velho et al. 2015).

Besides *Colletotrichum acutatum* SC, also *C. gloeosporioides* SC has been implicated in bitter rot. In Japan, bitter rot is one of the most severe diseases in apple production in general (Yokosawa et al. 2017). In countries such as in Brazil (Crusius et al. 2002), USA (Shi et al. 1996; Gonzales et al. 2006), and New Zealand (Everett et al. 2015) both *C. gloesporioides* SC and species within the *C. acutatum* complex occur together.

Both the species complexes of *C. acutatum* and *C. gloeosporioides* are considered as hemibiotrophs. It is assumed that they first have a biotrophic infection stage in which they retrieve their nutrients from living plant cells, and this is followed by a necrotrophic stage in which they kill host tissue to obtain their nutrition (Peres et al. 2005). It is known that these fungal species overwinter on infected peach and blueberry buds and twigs, but on apple the source of inoculum is not obvious (Peres et al. 2005). Recently, a few studies of the aetiology and epidemiology on apples have been published (Børve and Stensvand 2013, 2017). Also, a disease cycle for *C. acutatum* SC infecting apples and causing bitter rot in New Zealand was proposed (Everett et al. 2018). In this particular case it is suggested that inoculum is most commonly rain-splashed from inoculum sources, such as decaying petals, twigs and infected fruitlets that have fallen to the ground since spring. Infection is proposed to occur after conidiospore deposition on fruit, leaves and buds if they formed, in the presence of sufficient moisture and temperatures above 15 °C when the spores germinate and form appressoria to establish quiescent infections (Peres et al. 2005). Infections of buds and leaves are symptomless, because symptoms are not observed on leaves on the tree in New Zealand and buds do not seem to be

negatively affected in the following spring. In spring, buds open and the cycle can begin again (Everett et al. 2018).

The penetration and infection is well described for *Colletotrichum* spp. For instance, penetrating hyphae of *Colletotrichum* appressoria develop within the cuticle and uppermost epidermal cell layers of unripe fruit without eliciting visible host reactions, suggesting that fungal effectors that are secreted to support host colonization may interfere host response mechanisms (Giraldo and Valent 2013; Kleemann et al. 2012). The appressoria of *Colletotrichum* spp. are highly polarized cells from which a needle-like penetration hypha emerges in order to puncture the cuticle and epidermal cell wall (Howard and Valent 1996; Latunde-Dada 2001). At this stage, *Colletotrichum* is noted for its ability to maintain itself in an extended quiescent state until fruit ripening (Prusky et al. 2013).

Timely applications of fungicides are presumed to reduce infections of buds during summer, and in that way interrupt the disease cycle and could more effectively control the disease (Everett et al. 2015). This may provide a considerable improvement in reducing the number of applications over the currently recommended practice of calendar spraying throughout the season (Sutton 2014). Over the past few years, resistance of *Colletotrichum* spp. to the quinone-outside inhibitors (QoI) group of fungicides have appeared (Forcelini et al. 2018) and QoI resistant *Colletotrichum* isolates have been recovered from apples (Munir et al. 2016).

Neofabraea spp.

Bull's eye rot of apple and pear is an important postharvest disease, occurring in major fruit-growing areas of North America, Chile, Australia and Europe (Henriquez et al. 2004, 2008; Soto-Alvear et al. 2013; Spotts et al. 2009). The disease commonly occurs in most apple cultivars with an incidence of 10–20%, and may exceed 40% in years that are favourable to pathogen infection (Cameldi et al. 2016; Soto-Alvear et al. 2013). In Europe, 'Golden Delicious' and several late maturing apple cultivars, such as Pink Lady, are particularly susceptible to the disease (Cameldi et al. 2016; Neri et al. 2009). Bull's eye lesions on apple and pear fruits are generally caused by *Neofabraea* species, with *N. vagabunda* (syn. *N. alba*) as the main causal agent. However, also *N. malicorticis*, *N. perennans*, and *N. kienholzii* have been described to cause the disease (Gariépy et al. 2005; Michalecka et al. 2016; Pešicová et al. 2017; Soto-Alvear et al. 2013; Spotts et al. 2009).

Besides symptoms on stored fruit, *Neofabraea* spp. cause cankers on branches or develop saprophytically on pruning stubs and dead tree branches (Henriquez et al. 2006; Verkley 1999). The pathogen spreads by asexual sporulation on fruit mummies and bark cankers (Spotts 1990; Weber 2012). Conidiospores are produced throughout the year, but the highest sporulation levels occur during autumn (Henriquez et al. 2006). Although rain splash is considered the principal way for conidial dispersal, conidia can also be splash-dispersed by over-tree irrigation practices (Grove et al. 1992). Infections typically occur in the orchard throughout the growing season, anytime between petal fall and harvest, when unripe fruits are penetrated through the lenticels. Fruit susceptibility increases gradually during the season (Aguilar et al. 2017; Cameldi et al. 2016; Spotts 1990). After infection, the pathogen arrests its growth and remains quiescent until the fruit reaches a certain stage of ripeness when it can invade fruit tissues. Typically, bull's eye rot symptoms appear only after 3–4 months in cold storage, and numerous lesions may develop on a single fruit (Neri et al. 2009). Fruit lesions are circular, flat to slightly sunken, brown and often with a lighter brown center (Snowdon 1990).

Current management practices to control *Neofabraea* spp. in the orchard include pruning of cankers from infected trees to minimize the build-up of inoculum during the fruit growing season, removal of fallen fruit and dead tree branches from the orchard floor, and reduced use of over-tree irrigation systems that may promote splash dispersal of conidia from sporulating cankers onto developing fruit (Creemers 2014). Furthermore, fungicide application is a common component of bull's eye rot management (Aguilar et al. 2018).

Postharvest pathogens of pome fruit in the Netherlands and their control

Postharvest disease caused by *Colletotrichum* spp. and *Neofabraea* spp. are generally not causing severe problems in the Netherlands, most likely because here the main apple cultivar Elstar and pear cultivar Conference are not susceptible to these pathogens. However, more susceptible apple cultivars, such as Pinova and Topaz,

are frequently affected by *Neofabraea* spp. also in the Netherlands.

Until recently, it was unknown what the main causal agents of postharvest decay of pome fruit in the Netherlands were. In order to determine this, decayed apple and pear fruit were sampled from commercial CA storage facilities. In total, approximately 350 samples, derived from orchards with various apple and pear cultivars and from various production areas in the Netherlands, were analyzed between 2012 and 2018. These surveys revealed the presence of common postharvest pathogens, such as *Botrytis cinerea* and *Neofabraea alba*, but also a number of new and emerging postharvest pathogens, such as *Fusarium avenaceum* on pear and apple, *Neonectria candida* and *Neofabraea kienholzii* on pear, and *Colletotrichum godetiae* and *Truncatella angustata* on apple (Wenneker et al. 2016a, b, c, d, 2017a, b). In most cases these newly described postharvest pathogens were isolated at low incidences only. In contrast, two latent postharvest pathogens more frequently appeared: *Cadophora luteo-olivacea* causing side rot on pears (Wenneker et al. 2016e), and *Fibulorhizoctonia psychrophila* as the causal agent of lenticel spot on apples and pears (Wenneker et al. 2017c). For both diseases incidences range from very low to nearly 100% of stored fruits. Thus, these latter two fungal species are presently considered as the most important postharvest pathogens on pome fruit in the Netherlands.

The use of synthetic fungicides is currently the main means to control side rot and lenticel spot diseases. However, despite the routine use of fungicide applications fruit infections during the orchard phase are a growing problem. This may be due to the use of non-effective chemicals, ineffective spray application technologies or inadequate timing of the applications. Basically, robust knowledge on how to control these diseases with fungicide applications is lacking and current management is largely practiced in an empirical fashion. This requires urgent attention in order to ensure the deposition of sufficient quantities of active ingredients on fruits for disease protection during the entire storage period. However, the growing public concern over the health and environmental risks associated with high levels of fungicide residues on fruits, as well as the development of fungicide resistance in fungal pathogens, has resulted in the urge for developing alternative methods for disease control (Wisniewski et al. 2016a).

Alternatives to chemical fungicides for controlling postharvest diseases

Over the past decades the use of physical treatments, natural compounds, and biocontrol agents have been investigated as alternatives for the use of fungicides for controlling postharvest diseases, including diseases caused by latent infections. More recently, the fruit microbiome is considered as an important factor for controlling latent postharvest diseases (Droby and Wisniewski 2018).

Physical treatments

Physical treatments, like hot water and hot air treatments, radio frequencies and microwaves, hypobaric and hyperbaric pressures and far ultraviolet radiation (UV-C light) are considered as promising control means to reduce or delay the development of postharvest pathogens (Maxin et al. 2012; Usall et al. 2016). In Europe hot water dips are used for organic apples (Maxin et al. 2012). However, there are several disadvantages of hot water dipping that include high investment costs, relatively low throughput, additional labor during harvest time, high running costs and negative CO_2 footprint due to the energy requirement (Maxin et al. 2014). Consequently, hot water dipping is not implemented on larger scales in the fruit industry. As reduction in application time of the heat treatment could increase the interest in commercial use, research efforts have focused on short hot water treatments (rinsing) and expanding machine capacities (Maxin et al. 2012).

Radio frequency and microwave heating may provide effective alternative means to control postharvest diseases. The time required for microwave treatment is more favorable for commercial application, but the design and production cost for an equipment currently still obstructs its widespread application (Usall et al. 2016).

Among the remaining physical means, ultraviolet-C light (UV-C) treatment was considered to be interesting due to the simultaneous combination of direct activity against pathogens through germicidal effects on fungal spores with resistance induction through stimulation of defence mechanisms in several postharvest commodities including stone, pome, citrus fruit and table grapes (Nigro et al. 1998; Stevens et al. 1996; Valero et al. 2007; Wenneker et al. 2013). Although UV-C irradiation does not completely inhibit mycelial growth in vitro, a reduction in growth and sporulation was recorded for

most tested fungal species (Wenneker et al. 2013). However, UV-C has a superficial effect only due to the limited penetrating capacities of the waves. Thus, the potential for controlling latent infections will eventually be limited. Also, control of wound infections is not possible due to shielding effects by pores and irregularities on the fruit surface (Lagunas-Solar et al. 2006). Hormetic UV-C treatment of apples induced resistance to postharvest diseases, although the effect was relatively low (Stevens et al. 1996).

Presently, short hypobaric and hyperbaric pre-storage treatments with low and high ambient air pressure, respectively, are considered as promising alternative treatments for postharvest disease control, although their use remains largely unexploited to date (Usall et al. 2016).

Natural compounds

The application of microbial and plant volatile organic compounds (VOCs) to control postharvest decay have recently been reviewed by Mari et al. (2016). It is shown that plant-produced volatiles, including aldehydes, alcohols, essential oils, isothiocyanates and microbial volatile organic compounds may prevent pathogenic infections in many horticultural commodities (Mari et al. 2011; Sivakumar and Bautista-Baños 2014). However, the introduction of natural compounds into practice is complicated due to the expense of registration and limited market for them as plant protection products. Also, there are concerns about possible residues in fruit, and negative effects on taste and smell of fruits (Mari et al. 2016).

Biological control agents

Biological control agents (BCA's) have been the focus of considerable research efforts (Droby et al. 2016; Janisiewicz and Jurick II 2017), and are used to developed strategies to control postharvest decays of fruits. Especially wound-invading necrotrophic pathogens turn out to be sensitive to biocontrol (Janisiewicz 1988; Janisiewicz 1998; Korsten et al. 1994; Wilson and Wisniewski 1989). The control is facilitated because the antagonists can be applied directly to the targeted area (fruit wounds) by a single application using existing systems such as drenches and line sprayers (Janisiewicz and Korsten 2002). In addition to control of fruit wound infections, biocontrol also has been demonstrated to be effective for stem infection on pears (Janisiewicz 2006).

Several modes of action have been suggested to explain the biocontrol activity of microbial antagonists. The competition for nutrients and a niche between the pathogen and the antagonist is still considered as the major mode of action by which microbial agents control pathogens causing postharvest decay (Droby et al. 1992; Ippolito and Nigro 2000; Jijakli et al. 2001). Other modes of actions comprise the production of antibiotics, direct parasitism, and possibly induced resistance by which the microbial antagonists suppress the activity of postharvest pathogens on fruits (El-Ghaouth et al. 2004; Janisiewicz et al. 2000).

Several microbial antagonists have been identified and artificially introduced on a variety of harvested commodities including citrus, pome, and stone fruits, and vegetables for control of postharvest diseases (Sharma et al. 2009). More specifically for pome fruits, the biocontrol potential of microbial antagonists was reported to control decay caused by *Botrytis cinerea* and *Penicillium expansum* by the bacterial antagonists *Pseudomonas cepacia*, *P. syringae*, and *P. fluorescens* (Janisiewicz et al. 1991; Mikani et al. 2008). Decay of apple was also controlled by antagonistic yeasts such as *Candida sake* (Teixidó et al. 1999; Usall et al. 2001), *C. oleophila* (Wisniewski et al. 1995), and *C. saitona* (El-Ghaouth et al. 1998).

The success of some of these microbial antagonists in laboratory studies and pilot tests resulted in the commercialization of bioproducts containing microbial antagonists for control of postharvest diseases of fruits. For apples such bioproducts comprise Bio-Save (active ingredient (a.i.) *Pseudomonas syringae*), Boni Protect (a.i. *Aureobasidium pullulans*), Candifruit (a.i. *Candida sake*), Nexy (a.i. *C. oleophila*), Pantovital (a.i. *Pantoea agglomerans*), Shemer (a.i. *Metschnikowia fructicola*) and Yield Plus (a.i. *Cryptococcus albidus*) (Janisiewicz and Jurick II 2017; Sharma et al. 2009). Nevertheless, the commercial deployment of postharvest biocontrol agents has met difficulties for widespread success, which has been attributed to various problems, including inconsistent performance, high cost relative to synthetic fungicides, registration hurdles, difficulties in mass production and formulation of the antagonist, and lack of industry acceptance (Droby et al. 2009; Droby et al. 2016). The limitations of biocontrol products may be addressed by enhancing the bio-efficacy of microbial antagonists through: (i) manipulations in the

physical and chemical environment during storage, (ii) use of mixed cultures, (iii) addition of low doses of fungicides in the microbial cultures, (iv) addition of salt additives in the microbial cultures, (v) addition of nutrients and plant products in microbial cultures, (vi) use of the microbial cultures in association with physical treatments, and (vii) use of the microbial cultures with other approaches/additives (Janisiewicz 1996; Janisiewicz and Jurick II 2017; Sharma et al. 2009).

Thus far, research on biocontrol of postharvest diseases has mainly focused on identifying microorganisms that are antagonistic to wound pathogens and the effects of biocontrol agents on latent postharvest pathogens of pome fruit have hardy received attention in these studies (Droby et al. 2009; Sharma et al. 2009). Moreover, biocontrol relying on preventive mechanisms e.g. competition for limiting nutrients or space, which are effective in controlling fruit decays originating from wound infections, are not likely to succeed in this situation. Also, the strategy in most research studies published over the last few years has been to identify a BCA effective for a given pathogen, followed by testing its efficacy against other pathogens, often with limited success (Gava et al. 2018).

Currently, there is no specific biocontrol product available to control fruit decays originating from latent infections. According to Janisiewicz et al. (2011) this is due to the lack of appropriate methods for selecting effective biocontrol agents for controlling latent infections originating from appressoria and testing their effectiveness on fruit. Recently, they developed a novel approach based on a direct interaction of the isolated microorganisms with a pathogen (*M. fructicola*) latent infection structure in vitro and further screening of the selected potential antagonists for biocontrol effectiveness on fruit under laboratory conditions. The next step is to select those antagonists that are best adapted to conditions occurring during storage and handling of the fruit (Janisiewicz et al. 2011).

In addition to the development of novel biocontrol agents to prevent latent infections, a biocontrol agent (as well as a chemical compound) may be applied early in the season or even during the flowering stage. Therefore, application of biocontrol agents in the field during the growing season has been suggested (Ippolito and Nigro 2000; Lima et al. 1997; Lopes et al. 2015). However, knowledge on epidemiology of the causal agents of latent postharvest diseases is limited. Timing of application is complicated as the precise infection periods are often not known, may differ between the various pathogens, and infections may occur during the entire period from flowering until harvest.

The fruit microbiome

Microbial communities living on the surface of fruit have been the source of most of biocontrol agents (Janisiewicz 1987; Janisiewicz and Korsten 2002). They may directly influence pathogen development through antibiosis, parasitism or competition. The microbiota may also have an indirect role by stimulating plant defences. The commonly-used approach to identify novel biocontrol agents involves the identification of a single antagonist that can develop rapidly in wounded fruit tissue, thus preventing pathogens from becoming established. This approach, however, neglects interactions of antagonists with other microbes that occupy the same, or surrounding, niches as part of a microbial network and as a component of a complete biological system with the host (Droby et al. 2016).

Thus far, the overall diversity and composition of microbial communities on harvested produce, how they vary across produce types, and the factors that influence their composition after harvest and during storage, has been poorly studied (Droby and Wisniewski 2018). Recently, massive sequencing of PCR amplicons of specific barcode genes in amplicon metagenomics or metabarcoding approaches have revealed microbial diversities and relative quantities of community members in environmental samples (Abdelfattah et al. 2015; Massart et al. 2015). Such technology can similarly be used to characterize the composition of microbial communities on fruit, and also to identify strains considered as "helper microbial strains" or molecules involved in improving these direct or indirect effects against plant pathogens (Massart et al. 2015).

For example, Abdelfattah et al. (2016) demonstrated that the diversity of the fungal microflora of harvested apples differed significantly between fruit parts. Whereas *Penicillium* was dominant in peel samples, *Alternaria* was dominant in calyx- and stem-end samples. Niem et al. (2007) showed that differing susceptibilities of cv. Red Delicious and cv. Golden Delicious to core rot decay were not determined by the initial colonization of the blossom by the causal agent *Alternaria alternata*, but rather by the capability of the pathogen to colonize the host seed locule of the susceptible fruits. This type of information needs to be considered when designing

biocontrol systems for the management of postharvest diseases. For mango it was recently shown based on microbiome comparisons of stem ends that are resistant and susceptible to stem end rot in red and green fruit, respectively, that fungal and bacterial community change with fruit peel colour, storage duration, and storage temperature (Diskin et al. 2017).

Currently, *Neofabraea* spp. infection levels on apples at the time of harvest and the microbial dynamics on the apple skin during storage are characterized using a metagenomics approach (Bühlmann, pers. comm.). Ultimately, this type of research may lead to the synthetic design of microbial communities that can be used for postharvest disease management. Maintaining the right balance and diversity inside the consortium before and after its application, however, may be difficult. Regulatory difficulties in registering a consortium, composed of multiple microorganisms, as a biocontrol product may also become a problem.

A systems intervention approach

Some of the alternative methods to chemical fungicides for controlling latent postharvest diseases seem to hold promise for future application if the remaining challenges are met. After all, significant gaps still exist between the basic research that led to the discovery of these methods and their implementation under commercially relevant conditions. In order for such method to be applicable in practice, it must perform effectively and reliably, and be profitable to the company that has invested in its development, registration, and marketing. The results of the search for alternatives to chemical fungicides over the past thirty years show that, although several novel approaches have been identified as potential alternatives, no single method has emerged to robustly and reliably control postharvest diseases of pome fruit in practice. Thus, it may be advisable to move the focus from finding a single 'silver bullet' intervention that can be used to effectively control disease to composing and integrated systems approach by selecting the right set of control measures from a wide array of alternatives (Wisniewski et al. 2016a).

Already many recent research efforts are focussing on developing integrated control with biological control as a central pillar in combination with other compatible treatment(s), or combinations of alternative treatments (see reviews by Di Francesco and Mari 2014; Janisiewicz and Conway 2010; Palou et al. 2016; Romanazzi et al. 2016b; Usall et al. 2015). In this respect, this approach is well represented by the "hurdle concept" that was developed for apples (Janisiewicz 2008, 2013), which follows the original idea that was originally developed for food preservation (Leistner 2000). The "hurdle concept" explores the use of mild treatments that collectively maintain fruit quality and lower the incidence of postharvest decay (Palou et al. 2016). In the hurdle concept, each additional treatment reduces the incidence/severity of the decay by a certain percentage, and eventually results in the pathogen not being able to overcome the final hurdle, resulting in control of the fruit decay (Janisiewicz and Jurick II 2017). This view implies that latent postharvest diseases are complex problems that require multiple interventions at different stages of the disease process. Consequently, understanding the epidemiology of latent postharvest pathogens in the orchard, fruit defence mechanisms against pathogens, and the molecular biology of their interactions is required in order to develop novel integrated disease control methods (Droby et al. 2009; Tian et al. 2016).

The inoculum pressure of latent postharvest pathogens

There is considerable knowledge on the epidemiology of typical wound pathogens such as *P. expansum*. However, knowledge on epidemiology of the causal agents of latent postharvest diseases is limited. The control of the complex diversity of postharvest pathogens in orchards is difficult. The precise infection periods are often not known and may differ between the various pathogens, and is often complicated because infections may occur during the entire period from flowering until harvest.

Recently, Köhl et al. (2018) showed that both *N. alba* and *C. luteo-olivacea* were consistently detected in leaf litter of apple and pear and in necrotic tissues of dead weeds and grasses, and in many cases high concentrations of the pathogens were quantified. These are important new findings that may help to better understand how complex population dynamics of these necrotrophic pathogens depend on the availability of various necrotic host and non-host tissues for survival and multiplication. However, further research is still needed to understand: (i) the relationships between the

accumulation of pathogen inoculum on the various substrates over time, (ii) the relative importance of different substrates as inoculum sources for fruit infections, and (iii) infection periods on developing fruits in the orchard. Eventually, this knowledge can be used for the development of sanitation measures (Holb 2006; Llorente et al. 2010), or measures to stimulate beneficial microbiome inhabitants on those substrates that can antagonize pathogen colonization, survival and sporulation (Carisse and Rolland 2004; Llorente et al. 2006, 2010; Rossi and Pattori 2009).

The quiescent stage of postharvest pathogens

The quiescent phase, in this review also called latent phase, is a dynamic equilibrium among host, pathogen, and environment, which does not result in any visible symptoms on the host (Jarvis 1994; Prusky et al. 2013). During this stage, the fungal pathogens reside in the cuticular wax or in the intercellular space until the fruits ripen (Adaskaveg et al. 2000; Prins et al. 2000; Prusky et al. 1981). Apparently, at a particular moment physiological and biochemical responses of the host trigger changes in that equilibrium that activate the pathogen that is kept at a low metabolic level during the quiescent stage to activate pathogenicity mechanisms, resulting in active parasitic development in the host tissues (Prusky 1996). It has been proposed that the termination of the quiescent stage is the result of: (i) induced accessibility of disassembled cell wall substrates during fruit softening and ethylene induction; (ii) a decline in preformed antifungal compounds, such as polyphenols, phytoalexins, and other fungitoxic substances; (iii) a decline in inducible host-defence responses; and (iv) more favourable pH conditions in the host tissue. The pH in the fruit may change either naturally during fruit ripening or through induction by the pathogen that secretes pH modulators such as ammonia and organic acids as one of the first waves in their attack (Prusky et al. 2013; Yakoby et al. 2000). Both increases and decreases of ambient pH, for instance by secretion of ammonia and organic acids, respectively, have been recorded depending on pathogen and host characteristics (Alkan et al. 2013). For example, *Monilinia fruticola* acidifies the ambient pH by the secretion of gluconic acid (De Cal et al. 2013), while *Botrytis cinerea* (Manteau et al. 2003) and *Sclerotinia sclerotiorum* (Cessna et al. 2000) secrete oxalic acid to acidify the pH while enhancing their polygalacturonase gene expression and other cell-wall-degrading enzymes involved in tissue maceration (Misaghi 1982; Prusky and Lichter 2007). The produced endopolygalacturonases and pectin esterases may cause cell wall maceration and death of affected host cells (Paynter and Jen 1975). In contrast, *Colletotrichum* spp. were found to alkalinize the infection court by the secretion of ammonia to stimulate pathogenicity and necrotrophic colonization through the activation of host NADPH oxidases to generate reactive oxygen species, thereby accelerating host cell death (Miyara et al. 2010; Prusky et al. 2001).

Although not much is known for latent postharvest pathogens specifically, recently a pH increase was recorded in apple tissue infected by *N. vagabunda* (Cameldi et al. 2017*)*. However, further research is necessary to clarify the nature and the origin of the alkalizing compounds and to understand the effects of the pH modulation on *N. vagabunda* pathogenicity.

Plant and fruit defence mechanisms against postharvest pathogens

Plants have an innate immune system that comprises a wide variety of constitutive and inducible defence mechanisms to protect themselves against pests and pathogens (Cook et al. 2015; De Wit 2007; Tian et al. 2016). Constitutive or preformed defences include physical barriers such as cell walls and epidermal cuticles, but also chemicals such are antimicrobial phytoanticipins and some pathogenesis-related (PR) proteins (Van Loon et al. 2006). In addition to these preformed barriers, plant cells have the ability to detect invading pathogens and respond with inducible defences (Cook et al. 2015; De Wit 2007). These can be triggered when plant cells recognize microbe-associated molecular patterns (MAMPs), including structural proteins, lipopolysaccharides, and cell wall components commonly found in microbes, through a set of cell surface receptors, also referred to as pattern recognition receptors (Nürnberger et al. 2004) or invasion pattern receptors (Cook et al. 2015). In a study focused on tomato fruit defence responses against *C. gloeosporioides*, it was shown that the expression of several fruit genes was induced even before appressorial penetration. Such genes included PAMP receptors and genes related to fatty acid biosynthesis,

elongation, and the synthesis of cutin and waxes (Alkan et al. 2015).

Upon recognition, various defence responses are induced such as cell wall alterations, deposition of callose and the accumulation of PR proteins that include chitinases, glucanases and proteases that all negatively affect microbial colonization (Van Loon et al. 2006). Typically, also an oxidative burst occurs that involves the release of highly reactive oxygen molecules that damage the cells of invading organisms, cross-links host cell-wall components and acts as a signalling molecule to further enhance host immunity (Pitzschke et al. 2006).

Compatible pathogens can overcome the activation of host immunity by the secretion of effectors that perturb such responses (Kleemann et al. 2012; Weiberg et al. 2013; Zhang et al. 2014). Thus, pathogen effectors are crucial molecules for disease establishment (Rovenich et al. 2014). However, in turn plants have evolved receptors to recognize effectors or effector-mediated perturbations of host targets (Chisholm et al. 2006; Jones and Dangl 2006). These receptors may reside on the cell surface, but also inside the cytoplasm to detect (the activity of) cytoplasmically-delivered pathogen effectors (Cook et al. 2015). Often, the recognition of effectors has been associated with the occurrence of a hypersensitive response (HR); a localized programmed cell death response that may limit pathogen access to water and nutrients, and thus block further growth of the pathogen (De Wit 2007). However, necrotrophic pathogens may actually benefit from such cell death response, and have evolved in some cases to deliberately activate this host immune response to their benefit (Cook et al. 2015; Lorang et al. 2012).

In tissues that are distal from the infection site, plants are protected by so-called systemic acquired resistance (SAR) (Grant and Lamb 2006). SAR is effective against a broad range of pathogens and is dependent on various plant hormones, including salicylic acid (SA), jasmonic acid (JA), and ethylene (ET) (Grant and Lamb 2006). More recently, however, also various other hormones, such as auxin, abscisic acid (ABA), cytokinins (CKs), and brassinosteroids have been implicated in the activation of defence responses (Robert-Seilaniantz et al. 2011). SAR requires the signal molecule SA and is associated with accumulation of pathogenesis-related (PR) proteins (Durrant and Dong 2004; Terry and Joyce 2004).

Induced resistance against postharvest pathogens of fruits

A large amount of data has been generated related to priming of host plant defences and induce resistance during postharvest of fruits and vegetables (Janisiewicz and Jurick II 2017; Romanazzi et al. 2016a). The elicitation of host defences may be achieved by: (i) biocontrol agents, (ii) physical means (such as ultraviolet-C (UV-C) light, heat, hypobaric and hyperbaric treatments), (iii) natural and synthetic chemicals (such as phytohormones and chemical elicitors, salicylic acid, benzothiadiazole), (iv) biological elicitors (such as harpin and chitosan), (v) disinfecting agents (such as ozone, electrolyzed water, ethanol), and (vi) microbial and plant volatile organic compounds (VOCs) (Romanazzi et al. 2016a). However, there are a number of weaker points linked to the application of strategies based on induced resistance, such as possible inconsistent results or difficulties in their implementation in packinghouse practices. Moreover, to correctly induce resistance in fruits, it is necessary to know and understand the host–microbe interactions, and the effects on postharvest physiology and handling of the different fruits (Da Rocha and Hammerschmidt 2005; Tian et al. 2016).

Breeding for resistant cultivars to postharvest diseases

In general, fruit breeding objectives include high fruit quality, good agronomic performance and sometimes a durable disease resistance. In the latter case, in apple breeding mainly focussing on apple scab (*Venturia inaequalis*), fire blight (*Erwinia amylovora*), and powdery mildew (*Podosphaera leucotricha*) (Baumgartner et al. 2015). However, the success of newly developed disease resistant apple varieties is largely dependent on their fruit quality (Baumgartner et al. 2015). It should be emphasized that classic pome fruit breeding is a long-term and labor-intensive approach. The first fruits can usually be expected at the earliest in the fourth year after crossing. However, usually the first fruit quality selection step is carried out at the fifth to the seventh year after crossing. Currently, cultivated apples have often no resistance to fungi causing fruit decay as breeders seldom evaluate for resistance to postharvest diseases

(Ahmadi-Afzadi et al. 2013; Janisiewicz et al. 2008; Volk et al. 2015).

Fruit cultivars may show a large variation in susceptibility to (latent) postharvest diseases (Tian et al. 2016), as was demonstrated among apple cultivars for bull's eye rot caused by *Neofabraea* spp. (Blazek et al. 2007; Hortova et al. 2014; Soto-Alvear et al. 2013) and for *Colletotrichum* spp. (Biggs and Miller 2001; Grammen et al. 2019). Unravelling resistance mechanisms in fruits can be very helpful to make progress in breeding programs.

A number of new methods that allow a more precise selection of tree and fruit characters in breeding programs were developed in recent years (Laurens et al. 2018). For instance, marker assisted selection (MAS) facilitates the selection of novel cultivars in a shortened period for evaluation. Patocchi et al. (2005) developed a strategy called genome scanning approach (GSA) for traits that are primarily controlled by single major genes. This method can be used to identify linked molecular markers without generating a complete genetic map. For traits controlled by multiple genes, the quantitative trait loci (QTL) mapping is generally applied (Tian et al. 2016; Wisniewski et al. 2016b). In peach breeding programs, Pacheco et al. (2014) and Martínez-García et al. (2013) have identified QTLs for brown rot response traits, while preliminary results from apple breeding programs have identified QTLs for blue mould resistance in *Malus sieversii* (Norelli et al. 2014) and a mapping population of 'Royal Gala' × *M. sieversii* PI613981 (Wisniewski et al. 2016b).

In order to understand mechanisms involved in apple resistance to postharvest pathogens an approach involving temporal and spatial regulation of the transcriptome, proteome and metabolome combined with pathological analysis must be undertaken (Abdelfattah et al. 2015, 2016; Prusky et al. 2013). In this respect, sequencing of the genome of *Colletotrichum* species and transcriptome analysis of fungal–fruit interactions has revealed genes and key enzymes that are involved in the biosynthesis of fungal secondary metabolites that are important for pathogenicity and fruit defence responses (Alkan et al. 2015; Moraga et al. 2018). Nevertheless, typically, annotation processes and gene functional analyses are tedious and complicated. However, significant progress has been made in the determination of transcriptomic and proteomic factors that may lead to resistance in cultivated apples (Buron-Moles et al. 2015a, b; Vilanova et al. 2014), and several studies have provided data on genetically determined levels of resistance to *P. expansum* in apple cultivars (Ahmadi-Afzadi 2015; Tahir et al. 2015), and wild apples (Janisiewicz et al. 2016; Norelli et al. 2014).

Recently, a number of fruit crop genomes has been sequenced, including those of grapevine (Jaillon et al. 2007), apple (Velasco et al. 2010), banana (D'Hont et al. 2012), citrus (Xu et al. 2013), peach (Verde et al. 2013), and pear (Chagné et al. 2014). Also, the genomes of several postharvest pathogens have been sequenced, including those of *B. cinerea* (Amselem et al. 2011), several species of *Alternaria* (Dang et al. 2015), *Colletotrichum* (Gan et al. 2013), *P. expansum* and *P. italicum* (Ballester et al. 2015; Li et al. 2015). The genetic information that has been disclosed by these projects will provide insights in the virulence factors of these important postharvest pathogens, which can again be used in breeding and selection programs. In addition, genome-editing technologies involving CRISPR/Cas9 (Hsu et al. 2014) may be used to manipulate molecular regulators and edit promoters of apple fruit defence genes to enhance decay resistance in apple cultivars (Janisiewicz and Jurick II 2017). However, first of all, this requires a deeper knowledge of the fruit-pathogen-environment interactions at the physiological, biochemical and molecular level. Considering that combining plant genomics with classical breeding is a challenge for molecular biologists as well as for traditional breeders, an increased understanding of the basis of effective resistance mechanisms against the causal agents of postharvest pathogens is required. Eventually, such resistance mechanisms can be introduced into breeding programs to obtain postharvest disease resistant cultivars.

Concluding remarks and future perspectives

Losses due to the postharvest decay of pome fruits still represent a major concern from an economic point of view. However, it should be realized that fruit decay is a natural process to release seeds from mature fruit in order to start a new generation of the plant genotype. Currently, chemical fungicides represent the main tool for controlling the major postharvest pathogens as well as the deployment of optimal storage conditions. Interestingly, the synthetic cyclic olefin 1-methylcyclopropene (1-MCP) that blocks ethylene receptors and that is used to extend fruit firmness during

storage and marketing (Köpcke 2015) has also been shown to delay of onset of storage rots (McArtney et al. 2011) such as bull's eye rot on pears (Spotts et al. 2007) and on apples (Maxin and Weber 2011; Cameldi et al. 2016). Due to the growing concern over the use of synthetic fungicides, alternative measures to control postharvest diseases are sought. However, most of the alternative treatments developed so far have limitations that impede their effectiveness as single treatments. Combining different treatments within an integrated latent postharvest disease management strategy needs further development. Such integrated control methods should focus on reduction of the inoculum pressure of latent postharvest pathogens, interference of the typical latent stage of late postharvest pathogens and maximum exploitation of the plant's own immune system.

Electronic supplementary material

Ethical statements This article does not contain any studies with human participation or animals performed by any of the authors.

Conflicts of interest The authors declare that no known conflicts of interests exist.

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