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Journal of Postharvest Technology
2018, **06**(1): 001-011
http://www.jpht.info

REVIEWARTICLE

Decay mechanism of postharvest pathogens and their management using non-chemical and biological approaches

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Received: 30.09.2017 Accepted: 13.11.2017

ABSTRACT

Ripening is a physiological process that involves numerous biochemical changes including change in sugar composition and change in pH of the fruit and vegetable tissues. These changes attract a variety of pathogens causing decay, thereby leading to losses up to 25-50% in harvested crops. The infection by the postharvest pathogens may remain quiescent during fruit or vegetable growth due to unfavourable growth conditions for pathogen's development to pathogenesis. Owing ripening and senescence the pathogen transform to necrotrophs, activation of the pathogen is primarily due to change in pH and cause typical decay symptoms. Some pathogens have developed a mechanism for altering the pH of host environment (tissue), thereby increasing virulence. The postharvest pathogens cause acidification by producing organic acids and alkalization by ammonification of host tissues. Management of postharvest tissue-decaying pathogens is usually performed by use of fungicides but due to raised environmental concerns and residual effects of fungicides on health alternative management strategies are required. This approach should be eco-friendly; the use of plant extract and biological agents are in limited use. Thus, it should be emphasized that management of postharvest food commodity (particularly those of direct consumption like fruit) pathogens can be achieved by altering the physiological environment and use of alternative management strategies to reduce the residual effect of inorganic fungicides and other chemicals. This review deals the both of the aspects and encourages the wide use of biorational and biological approaches for management of postharvest pathogens.

Key words: biocontrol, decay mechanism, pathogen, plant extract, postharvest

Citation: Kumar, R., Ghatak, A., Balodi, R. and Bhagat, A.P. 2018. Decay mechanism of postharvest pathogens and their management using non-chemical and biological approaches. *Journal of Postharvest Technology*, **6**(1):001-011.

INTRODUCTION

In agriculture, harvesting is performed after the several stages of plant growth and application of various practices of crop production. The harvesting further includes cooling, cleaning, sorting and packing, which is considered postharvest handling. The postharvest deterioration/ spoiling is rapidly initiated when a crop is uprooted from the ground or separated from its parent plant. Therefore, to sustain the quality of the product it is very important to keep the product cool and limit moisture loss from the harvested crop or its commodity. Slowing down the undesirable chemical changes and avoiding physical damage would delayed the spoilage and reduce the possibility of pathogenic infection (Bachmann and Earles, 2000).

The postharvest fungal pathogens infect the host tissue through three major routes and cause detrimental effect to the material: (i) - through wounds; (ii) - through natural openings, and (iii) - by direct penetrating host cuticle. The pathogenic

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ISSN: 2348-4330

process may start immediately as the spores of the pathogen land on the wounded tissue or the pathogen may remain inactive for a long time until the maturity of the harvested product is extended. The penetration process may remain unnoticed by the host, or it may result into a defence signalling that results in the induction of defence mechanisms that will limit the growth of fungal development (Prusky and Lichter, 2007). The period from host infection to the activation of fungal development and symptom expression is known as the quiescent stage (Prusky, 1996). During ripening and postharvest storage the mechanisms that protects the tissue from fungal attack becomes insufficient. This transition from a resistant to susceptible state parallels physiological changes that occur during ripening, activating pathogen for disease initiation. For management of postharvest diseases modulation of host environment for reducing fungal colonization can be an alternative method along with chemical management and biological control. When fungicides are applied for postharvest disease management, human populations are more exposed to residues of the chemicals causing health hazard.

Fungicidal management are harmful for the postharvest commodities meant for direct consumption (Ghatak et al., 2015). Considering the raising concerns for ecological and anthropological wellbeing use of chemicals is discouraged in agriculture. Thus, there is an urgent need for alternatives to fungicides for the control of plant diseases. This review is therefore deal with the management options that have no toxic or residual effect on human. Our attempt is to expose the researchers to non-chemical and biological management alternatives for postharvest disease management by inhibiting the pathogenic growth. This review may draw the attention of policy makers for discouraging the toxic chemical use and to promote the other alternatives with no harmful effect on mankind for the management of postharvest commodities with increased shelf-life.

MECHANISM OF PATHOGENESIS DURING POSTHARVEST DECAY

Quiescent stage

Postharvest fungal pathogens have two main modes of nutrition during the colonization on plant hosts: (i) - biotrophy, in which the nutrients are obtained from the living host cells, and (ii) - necrotrophy, in which nutrients are obtained from dead host cells killed by the fungus (Perfect et al., 1999). Opportunistic postharvest pathogens may also reside in the matured fruit in an inactive (quiescent) mode, awaiting fruit for wounding, ripening or senescence. The length of each period may vary among pathogens, hosts, and host developmental stages.

Quiescent and necrotrophic stage

Several factors at cellular levels are responsible for transition to necrotrophic from the quiescent stage and these factors are a function of nutrients and pH of the harvest. Theories regarding the mechanism for secretion of chemicals for infection are (Kim et al., 2008):

a) Oxalate weaken the host plants for invasion,
b) Acids (oxalic acid and gluconic acid) loosen the plant cell wall by chelation, and
c) Activation of Reactive Oxygen Species (ROS) generation, which leads to necrotrophic mode of development.

Disease free plants have an electrochemical proton gradient across the plasma membrane. Schaller and Oecking (1999) reported that ephemeral alkalization is responsible for plant defence response, thereby contributing to activation for development of necrotrophic mode.

The role for ammonium secretion in the induction of host ROS accumulation through the NADPH oxidase mechanism in a pH-dependent manner was explained by Alkan et al. (2009). ROS activation by ammonium can act as a factor for promoting cell death in tomato fruit. Ammonium can directly affect the fungus pathogenicity by activation of the expression of genes encoding pathogenicity factors, such as expression of pelB gene, which encodes pectate lyase (PL) secretion in *Colletotrichum*

gloeosporioides. Finally ammonium can also indirectly affect the pathogen-host interaction by elevating pH of the infection site to create an optimal environment for production of pathogenicity factors as PL and endoglucanase (Yakoby et al., 2001; Eshel et al., 2002). This suggests that ammonification by *Colletotrichum* at the infection site may be responsible for call death and activation of fungal pathogenicity factors. Change in pH may be responsible for transition from quiescent to necrotrophic stage in fungal pathogen in this case. Examples of pathogens which activate necrotrophic development in senescing and ripening fruits are *Alternaria*, *Botrytis*, *Colletotrichum*, *Monilinia* etc.

Colletotrichum spores adhere and germinate on the plant surface, produce germ tubes, develop appresorium and penetrate by infection peg through cuticle followed by subcuticular colonization and proliferation inter- or intra-cellularly in the host (Perfect et al., 1999); this phase is followed by necrotrophic phase of fungal growth (Mendgen and Hahn, 2001). In contrast, *Botrytis* and *Monilinia* can penetrate through wounds and also breach the fruit cuticle by using an infection peg from an appresorium that then remains quiescent for long periods of time (Pezet et al., 2003).

Inflection of pH by postharvest fungal pathogens

Alkalizing fungi and acidifying fungi (e.g. *Penicillium* sp., *Botrytis cinerea*) has the ability to modify pH in the surrounding environment (the host tissue). Modification of enzyme expression in response to the ambient pH in the host by the fungus shows the importance of the specific regulatory system, which can lead to the activation of infections (Prusky and Yakoby, 2003). Postharvest pathogens secrete organic acids and cause tissue acidification for their attack e.g. *Penicillium digitatum*, *Botrytis cinerea* etc. In case of apple and citrus, pH change ranging from 3.95-4.31 in healthy tissue and 3.64-3.88 in the decaying tissue caused by *P. expansum* results in rapid decaying of apple in several cultivars. Similarly the pH decline has been observed in citrus after the infection of *P. digitatum* and *P. italicum* (Prusky et al., 2004). In the necrotrophic fungus *Sclerotinia sclerotiorum*, during plant infection, a pH gradient was established in relation to oxalic acid secretion and the pH of the host reached 4.0 (Rollins and Dickman, 2001). The production of oxalic acid during plant infection has been implicated as a primary determinant of pathogenicity in this and other phytopathogenic fungi. The size of lesions induced by different strains of *Botrytis cinerea* on grapevine and bean leaves correlated with the amount of oxalic acid (Germeier et al., 1994) and it is suggested that oxalic acid act as a co-factor in pathogenesis instead primary disease inciting agents (Manteau et al., 2003).

Mechanism of pH modification by pathogens

Mechanisms for tissue acidification have been proposed on the basis of several studies. Two mechanisms have been used by *Penicillium* spp. for ambient acidification: (i) - the production of organic acids, mainly citric acid and gluconic acid (Prusky et al., 2003), and (ii) - $NH4^+$ utilization associated with H^+ efflux. In decayed fruit by *P. expansum* and *P. digitatum*, both produces citric acid and gluconic acid in the decayed tissue and reduced the host pH by 0.5-1.0. Ammonium depletion from the growth medium or from the fruit tissue is directly related to acidification.

Several studies on fungal pathogen demonstrate that the ambient pH conditions in host induce the expression of a particular subset of fungal genes, selected from large gene families that encode cell wall degrading enzymes (Prusky and Yakoby, 2003). In *P. expansum*, the secretion of gluconic acid and, to a lesser extent, citric acid enhanced the expression of pectolytic enzymes and the establishment of conditions for necrotrophic development (Hadas et al., 2007). Analysis of transcripts encoding the endo-polygalacturonase gene, pepGL, from *P. expansum* under acidic culture conditions (pH 3.5-5.0), suggests that the acidification process is a pathogenicity enhancing factor for *Penicillium* spp. This was supported by the findings that cultivars with lower pH as well as citric acid treatments that reduced tissue pH, increased *P. expansum* development. Conversely, local alkalinization with $NaHCO_3$ reduced decay development (Prusky et al., 2003). The observations demonstrate that ambient pH is a regulatory cue for processes linked to pathogenicity, and that specific genes are expressed as a result of the modified host pH created by the pathogen.

Plant pathogens also make the environment alkali for enhancing pathogenicity. In ripening and senescing fruits, the pH levels change; for example, the pH of avocado increases from 5.2-6.0 during ripening (Yakoby et al., 2000). Alkalinization of the host environment during colonization by postharvest pathogens such as *Colletotrichum* and *Alternaria* is associated with fungal secretion of ammonia (Eshel et al., 2002). In avocado, the fruit's pericarp showed a pH of 5.2 that increased up to 7.5-8.0, upon infection caused by *C. gloeosporioides* (Yakoby et al., 2000). In tomato fruits, the initial pH ranged between 4.1 and 4.5 and decay development at the infection site increased the pH to 8.0; moreover, the ammonia accumulated to 3.6 mM compared to 0.2 mM in the healthy tissue (Alkan et al., 2008; Prusky et al., 2001). In wide-host-range pathogens such as *A. alternata*, a threefold to tenfold increase in ammonia concentration and a pH elevation of 0.2-2.4 units were detected in several hosts like tomato, pepper, melon, cherry and persimmon (Eshel et al., 2002). Alkalization by pathogens is achieved by active secretion of ammonia, which is produced as a result of protease activity and deamination of amino acids (Prusky and Yakoby, 2003). The initial acidic pH has been shown to be conducive to the enhanced ammonium secretion and host-tissue alkalization that facilitate the virulence of pathogen (Alkan et al., 2008). Ammonium accumulation is a critical factor contributing to necrotrophic development in ripening fruits (Prusky and Yakoby, 2003). Expression of the endoglucanase gene AaK1 by A. alternata is maximal at pH levels above 6.0 that are characteristic of decayed tissue. The AaK1 gene does not express at the lower pH values in which the pathogen is quiescent (Eshel et al., 2002). In the pathogen *C. gloeosporioides*, the gene pelB is expressed when the pH was above 5.7 and was positively correlated with ambient alkalinization (Yakoby et al., 2000; 2001). The transcription factor pac1, which is involved in pH regulation, follows a pattern similar to that of pelB, which suggests that they are co-regulated or that pac1regulates the expression of pelB (Drori et al., 2003).

PLANT-DERIVED FUNGICIDES

Plant derived fungicides are the secondary metabolites produced by the plants which are unsuitable for the growth or development of plant pathogens. Secondary metabolism pathways utilize a significant part of the energy from the plant to produce these secondary metabolites (Ansar et al., 2017). A number of effective insecticides have been derived from plants, but at present no fungicides of plant origin are available commercially. Earlier studies demonstrate that aqueous and organic solvent extracts of garlic contained very potent fungicidal and bactericidal activity against a variety of plant pathogens (Ark and Thompson, 1959) specifically for brown rot of peaches. Also, Wilson et al. (1987) reported that the volatile compounds naturally produced by ripening peaches were highly fungicidal. A number of essential oils are produced by plants that have fungicidal activity and may have potential for the control of postharvest diseases of fruit (Maruzzella et al., 1960). Approximately, < 2% of the described higher plant species has been screened for pesticide activity (Grainge and Ahmed, 1988). Most of this screening has been for insecticides: < 1% of the plants have been observed for antimicrobial activity. This leaves a large untapped source for future fungicides.

Although, there is an importance of secondary metabolites in plant protection, but not much effort has gone into utilizing these compounds in plant disease control. This has been mainly because a number of very current and active synthetic fungicides that are thought to be safe for man and the environment. The co-evolution of man with plants may have weakened the impact of toxic plant metabolites through the process of natural selection. Moreover, it would seem that the natural plant metabolites are more biodegradable than synthetic ones. Basson (1987) while studying poisoning of wildlife in southern Africa found that 'wherever co-evolution with toxic plants existed, antelopes usually showed some superior detoxifying mechanisms or innate resistance'.

This review listed a number of plant extract and essential oil with their effect on plant pathogen inhibition (Table 1). The use of natural compounds to control postharvest diseases should be more appealed. Commodities may be fumigated with the volatile or it may be incorporated into sprays or packaging. Prasad and Stadelbacker (1973) controlled *Botrytis cinerea* and *Rhizopus stolonifer* rots of strawberry and raspberry with acetaldehyde vapour. Acetaldehyde has also been used as a fumigant to

control the green peach aphid on head lettuce but, phytotoxicity from the fumigant has been also reported (Stewart et al., 1980).

Table 1. Postharvest pathogen management in various commodities using non-chemical and biological approaches

Approach	Targeting pathogen	Commodity	Impact	Reference
Plant extract				
Anadenanthera colubrine	*Alternaria alternata*	Murcott tangor fruits	Suppression of lesions development	Costa Carvalho et al. (2011)
Cassia	*A. alternata*	Cherry tomatoes	Reduction of decaying	Feng and Zheng (2007)
Cassia	*Botrytis cinerea*	Strawberries	Reduction of decaying	El-Mogy and Alsanius (2012)
Origanum vulgare	*B. cinerea* and *P. expansum*	Pears	Reduction in lesion diameter	Matos and Barreiro (2004)
O. vulgare	*Colletotrichum coccodes*	Tomato	Viability suppression of spore	Tzortzakis (2010)
Cicuta virosa	*A. flavus*, *A. oryzae*, *A. niger* and *A. alternata*	Cherry tomatoes	Reduced infected fruits for all the species	Tian et al. (2011)
Brassica oleracea	*A. alternata*	Green bell pepper	Great fungicidal effect	Troncoso et al. (2005)
Allium and *Capsicum*	*Botrytis cinerea*	NI	Antifungal activity	Wilson et al. (1997)
Essential oil				
Lippia scaberrima oil	*Penicillium digitatum*	Citrus	Excellent disease control and overall fruit quality was maintained	du Plooy et al. (2009)
Lippia scaberrima oil	*Botryosphaeria parva* and *Colletotrichum gloeosporioides*	Mango	Reduced fungal infection by both pathogens	Regnier et al. (2008)
Cymbopogon martini	*Botrytis cinerea*	NI	High levels of antifungal activity	Wilson et al. (1997)
Thymus zygis	*Botrytis cinerea*	NI	High levels of antifungal activity	Wilson et al. (1997)

Cinnamomum zeylanicum	*Botrytis cinerea*	NI	High levels of antifungal activity	Wilson et al. (1997)
Eugenia caryophyllata	*Botrytis cinerea*	NI	High levels of antifungal activity	Wilson et al. (1997)
Biological agent				
Candida guilliermondii and *Pichia membranefaciens*	*Rhizopus stolonifer*	NI	Limited pathogen growth	Tian et al. (2002)
Bacillus amyloliquefaciens	*Penicillium crustosum*, *Alternaria citri*, *Colletotrichum gloeosporioides*, *Phomopsis persea*	NI	Spore germination inhibited	Arrebola et al. (2009)

NI: Not indicated

MANIPULATION OF PLANT RESISTANCE RESPONSES

Resistance against the plant pathogens is the rule and susceptibility is the exception. Resistance in harvested fruits has been identified in few host-pathogen systems. Resistance responses have been demonstrated in harvested citrus (Baudoin and Eckert, 1985), apple (Sommer and Fartlage, 1988) and potato (Austin et al., 1988). Division of labour by the pathogen within the plant tissues for the infection is an important mechanism. Baudoin and Eckert (1985) reported that the lignin-like substances were deposited in the outer peel after wounding. The level of resistance to *Geotrichum candidum* is same as the amount of lignin-like deposition. Skene (1981) showed that apples wounded soon after harvest healed quicker and to a greater extent than apples wounded later in storage. This study suggested that wound healing is a cell-mediated reaction that resulted in lignin-like deposits. In potato, wound resistance was highly correlated with lipid and lignin deposits at the wound site (Marvis, Forbes-Smith and Seriven, 1989). As in apples and oranges, the longer the period between wounding and infection, the lower the disease incidence (Workman et al., 1982).

Plants have been shown to produce pectinase and proteinase inhibitors (Cervone et al., 1989). Albersheim and Anderson (1971) showed that proteins from plant cell walls inhibited poly-galacturonases (PG) secreted by plant pathogens. The level of the PG-inhibitor activity was correlated negatively with the rate of fungal rot development in apple fruits. Labavitch et al. (1983) showed that the PG-inhibitor activity in pear fruit decreased as the fruit matured, demonstrating a correlation between developmentally related changes, PG-inhibitor activity, and tissue susceptibility to pathogens. Identification and manipulation of pectinase and proteinase inhibitors in harvested commodities may provide an effective new way to control postharvest diseases.

Various treatments have been shown to induce resistance responses in fruit. Stevens et al. (1990) and Lu et al. (1987) found that low doses of UV-irradiation induced resistance in sweet potato and onion to postharvest rots. Swinburne (1978) demonstrated that proteases from *Nectria galligena* were able to elicit benzoic acid in apple fruit that was antibiotic to the

pathogen. The multiplicity of chemical and physical elicitors that can induce resistance in harvested commodities need to be explored and used for biological control. If resistance in harvested fruits is to be manipulated, a more fundamental understanding of the nature of the resistance is needed. In this way a few attempts have been made to select and breed plants for resistance to postharvest diseases. Breeders, in selecting the fruits for desirable horticultural characteristics may have inadvertently selected for greater susceptibility to postharvest diseases.

BIOLOGICAL CONTROL

Biological control of plant diseases is defined as 'the decrease of inoculum or the disease-producing activity of a pathogen accomplished through one or more organisms, including the host plant, but excluding man' (Baker, 1987). A number of potential biological control avenues are open for controlling postharvest diseases, which includes use of antagonistic microorganisms, plant derived fungicides and manipulation of resistance response. For example, control of the crown gall bacterium, *Agrobacterium tumefaciens*, through the use of an antagonistic bacterium *A. radiobacter* (Kerr, 1980) and the use of *Trichoderma* for the control of a number of soil-borne pathogens (Lewis and Papavizas, 1984) are some of the notable examples of the use of antagonistic microorganisms to control plant pathogens.

Antagonistic microorganisms

Postharvest environment appears to present a better ambiance for biological control than that of field conditions. In postharvest storage conditions temperature and humidity can be controlled and microbial community is different than the natural microbial community. These conditions favour introduction of suitable biocontrol agents for management of post harvest diseases (Pusey and Wilson, 1984). The antagonists obstruct the pathogen directly by the secretion of antibiotics, compete with the pathogen for nutrients and space and are better adapted to adverse environmental conditions. Biocontrol agents may be able to use nutrients at low concentrations and survive and develop at the infection site under temperature, pH, or osmotic conditions which are unfavourable for the growth of the pathogen. Such an antagonist will inhibit, but not kill the pathogen.

Yeast antagonist US-7 has the antagonistic activity against the citrus pathogen, *Penicillium digitatum*, which can be reduced markedly by the addition of nutrients to the interaction site between the yeast and the pathogen (Droby et al., 1989). Effective competition for nutrients with the pathogen was demonstrated by Droby et al. (1989). Likewise, *Enterobacter cloacae* also inhibit the germination of *Rhizopus stolonifer* by nutrient competition (Wisniewski et al., 1989).

Various screening tests have been performed for naturally occurring microbial antagonists of postharvest diseases of citrus and deciduous fruits by Wilson and Chalutz (1989). A large proportion of the epiphytic microbial population isolated from the surface of these fruits showed inhibition against fungal pathogens in culture. *Bacillus subtilis* exhibits potent antibiotic activity in culture against several important pathogens of fruits (Pusey, 1989). The uniqueness of the active substance named "iturin" which is involved in the inhibition has been elucidated (Gueldner et al., 1988). *Pseudomonas cepacia* also controls a number of fruit rot pathogens by producing an antibiotic named "pyrrolonitrin" (Janisiewicz and Roitman, 1988).

Many of the post harvest diseases have been successfully controlled with the help of biocontrol agents which includes, pome fruits (Roitman, 1988), citrus fruits (Singh and Deverall, 1984), and various other fruits and vegetables. This review is enlisted a few examples where the growth and progress of the postharvest pathogens have been inhibited using biological agents of microbial origin (Table 1). Apart from these works, patents have been issued or are waiting on a number of these antagonistic microorganisms. Attempts have been made to commercialize some of these antagonists. *Bacillus subtilis* has been patented for the control of brown rot of stone fruit (Pusey and Wilson, 1988), *Pseudomonas cepacia* and *Acremonium* for the control of *Botrytis* and *Penicillium* rots of pome fruit (Janisiewicz and Roitman, 1988), and yeasts (*Pichia guilliermondi*, strain US-7 and

Hanseniaspora uvarum (strain 138) for the control rots of citrus, stone fruit, pome fruit and tomato (Wilson, 1990).

CONCLUSION

It has become clear that the activation of quiescent infections is not a simple process and a weak host resistance leads to the activation of pathogenic attack. The complexity of the pathogenic process can be recognized to the significant physiological and biochemical changes which occur in the host during ripening and senescence, and lead to declined host response and increase in disease susceptibility. The activation of quiescent infections consists of processes which include host defences directly or indirectly by detoxification of antifungal agents. The physiological changes that leads to fruit ripening and host senescence, including pH, sugar content, cell-wall components and oxidation of wounded tissue, trigger fungal host modulation. The acidification of the tissue by organic acids (oxalic and gluconic) or its alkalization by ammonia and the possible modulation of ROS, contribute to rapid narcotisation of the tissue. Increase in the decay can result from activation of gene expression and release of cell-wall-degrading enzymes. Additionally, the clarification of the role of putative signals (pH, nitrogen and sugar) in postharvest pathogenesis during fruit ripening is clearly needed. Nevertheless, the current state of knowledge of fungal modulation of host pH has already opened new avenues to the control of postharvest pathogens thus studies of virulence as a result of pH-conditioning by the pathogen support the developing of new strategies of postharvest fungal pathogen control involve local pH changes (Prusky et al., 2006).

The toxic effect has been reported from various chemicals used for management of postharvest diseases. The alternative is to encouraging application of plant extract having diseases inhibition property for the postharvest decaying. Some biocontrol agents like antagonistic microorganisms have also appeared to play a critical role in saving of postharvest damages. Although antagonistic microorganisms promise as 'living fungicides' for the control of postharvest plant diseases but the path from the laboratory to commercial production of these agents has several economic and biological obstacles. Patentability of an antagonist as a biocontrol agent is important and mandatory. Companies are not ready for the risk of investment needed to develop a product, without assurance of a market advantage. The market must be large enough to justify the research and development necessary for the commercialization of antagonistic microorganisms. The specificity of an antagonist may prohibit its commercialization on economic grounds, even though it is highly effective. However, with the continued withdrawal of synthetic fungicides from the market, places for safer alternatives are being created. The works on management of postharvest pathogens by plant origin metabolites and biological agents presented here in this review unveil the hidden arena of postharvest research on such approaches. This review would, thus, provide a scope to a specific group of researchers and attract the policy makers to think over this area of approach without letting harm (residual effect) to the postharvest commodity. Such approaches may be applicable for long-term management system for example food corporation agencies and other storage chains in a country.

A huge loss has been reported for several commodities in storage due to several diseases that are caused by postharvest pathogens. The only option adopted is the use of synthetic fungicides for control of such diseases. The withdrawal of synthetic fungicides from the market will cause short-term economic hardships to growers. Long term effects, however, may be favourable in that a greater effort will be put into finding safer alternatives to synthetic fungicides. A variety of biological control strategies are available as alternatives to the use of synthetic fungicides for the control of postharvest diseases. Development and implementation of this new method of disease control will require greatly accelerated research. Research among state and industrial laboratories is needed to develop products and procedures that are effective, safe and profitable.

Acknowledgement

The authors are grateful for the support extended by the BAU, Sabour and ICAR-IGFRI, Jhansi. This article bears a BAU Communication Number 318/2017.

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