

Current Status of Post Harvest Soft Rot in Vegetables: A Review

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Abstract: Soft rot is one of the destructive diseases of vegetables and occurs worldwide wherever fleshy storage tissues of vegetables and ornamentals are found. It causes a greater total loss of produce than any other bacterial disease. The disease can be found on crops in the field, in transit, in storage and during marketing; resulting in great economic losses. It is primarily caused by *Erwinia carotovora* sub-sp. *carotovora* and sometimes by *Erwinia carotovora* sub-sp. *atroseptica*. The soft rot disease has a very wide host range infecting vegetable species belonging to all families. Name of the disease aroused from the characteristic soft decay of fleshy tissue which terminates into watery or slimy mass. The bacteria enters the host tissue through injuries. The decay is aggravated when high humidity is coupled with high temperature which results in fast rate of multiplication of the pathogen. For this reason much of the loss due to this disease occurs during middle of the summer. Increased amounts of pectolytic enzymes released by the pathogen results in maceration of tissue and are of great significance to the pathogenesis of the disease. Control of the disease which includes sanitation of packing house, lowering of storage temperature and humidity, host resistance and other physical and chemical measures are discussed in detail.

Key words: Soft rot, vegetables, *Erwinia carotovora*, pectolytic enzymes

INTRODUCTION

Soft rot is one of the destructive diseases of vegetables. It occurs worldwide wherever fleshy storage tissues of vegetables and ornamentals are found. The disease can be found on crops in the field, in transit and in storage or during marketing resulting in great economic losses. Soft rot causes greater total loss of produce than any other bacterial disease. Post harvest bacterial soft rot losses have been estimated to vary between 15-30% of the harvested crop (Agrios, 2006). Vegetables coming from the field may be already infected although they may not yet show visible symptoms at harvest which later on may cause severe damage because of high air temperature, humidity and poor transport management. *Erwinia carotovora* subsp. *carotovora* and *Pseudomonas fluorescens* causes the most common and most destructive soft rots *Erwinia carotovora* sub sp. *atroseptica* is also found associated with soft rot.

The soft rot bacterium enters plant tissues primarily through wounds, often created by insect feeding or

bruising at harvest. Insects and water are effective in spreading the bacterium. Once in the plant tissue, the bacterium produces increasing amounts of pectolytic enzymes that break down the pectic substances of the middle lamella causing break down and maceration of the tissues (Gupta and Thind, 2006). Soft rot (*Erwinia carotovora* subsp. *carotovora* (Jones) (Bergey *et al.*, 1939) is of great importance both in the field as well as in transit and storage, causing heavy economic losses to various vegetables. In this study, an attempt has been made to review the relevant available literature concerning soft rot disease in vegetables.

CAUSAL ORGANISM AND ITS MORPHOLOGICAL CULTURAL AND BIOCHEMICAL CHARACTERISTICS

Soft rot of vegetables is caused by various species of, *Bacillus*, *Pseudomonas* and *Erwinia* (Agrios, 2006). *Erwinia carotovora* subsp. *carotovora* is however considered to be one of the major soft rot causing

bacterium (CCHAU, 2002; Larka, 2004). *Erwinia carotovora* subsp. *carotovora* and *Pseudomonas fluorescens* cause the most common and most destructive soft rots. *Erwinia carotovora* sub-sp *atroseptica* may be thought of as a cool temperate variety of *Erwinia carotovora* sub-sp. *carotovora* (Agrios, 2006). *Erwinia carotovora* are straight rods, 0.5-1×1-3 microns mainly single; motile with peritrichous flagella. Gram negative, facultatively anaerobic; catalase positive, oxidase negative, Urease not produced by the bacterium. Acid is produced from D (+) glucose, D (+) galactose, D(-) fructose and sucrose. Starch is not hydrolyzed but the bacterium readily hydrolyses gelatin and pectin. Optimum temperature for *Erwinia carotovora* ranges from 27-30°C maximum varies from 32-40°C (Bradbury, 1986).

They are non pigmented and grows well in most laboratory media, produces H₂S from cystine. Reduces nitrate, some strains also produce gas but others are anaerobic produce acid actively from various carbohydrates (Bradbury, 1986). *Erwinia carotovora* sub-sp *atroseptica* has similar properties except its maximum temperature for growth is 35°C and shows acidic reaction in 7 days with alpha-methylglucoside and utilizes maltose unlike *Erwinia carotovora* sub-sp. *carotovora* (Bradbury, 1986). Bacterium generally produces white colonies on nutrient agar and cloudy growth in nutrient broth (Gupta and Thind, 2006).

OCCURRENCE AND SEVERITY OF THE DISEASE

Bacterial soft rot occurs world wide and causes serious diseases of vegetable crops in the field, in transit and especially in the storage. It causes a greater total loss of produce than any other bacterial disease (Agrios, 2006). Bacterial soft rot of vegetables has been studied since 1891 when Halsted described it on celery (Walker, 1998). A few years later it was recorded as injuring cabbage and other crucifers (Russel, 1898). It was in 1901 the disease was described in detail on carrots (Jones, 1901; Harrison, 1904). Since then the organism under various names, has been observed on following additional hosts; artichoke, asparagus, cauliflower, egg plant, pepper, potato, radish, rhubarb, tomato and turnip (Hurding *et al.*, 1909; Johnson, 1910). Recently Farrar *et al.* (2009) reported that lenticel rot caused by *E. carotovora* in potato has probably been in California since potato production became established in Kern County in 1912, but did not reach damaging levels until the late 1990s. soft rot in vegetables have been reported from all over the world, Rajeh and Khlaif (2000) reported soft rot disease caused by *E. carotovora* from different areas of Jordan including Jordan valleu and upland.

Togoshi *et al.* (1988) gave the first report of bacterial soft rot in vegetables grown on burnt fields in Japan. In Norway this disease has been reported to cause serious problem in Chinese cabbage (Balvoll, 1995). In India *E. carotovora* subsp. *carotovora* is considered to be one of the major soft rot causing bacterium in the tropics (CCHAU, 2002; Larka, 2004). In northern India where cabbage is grown for seed production the disease cause rotting of cabbage heads which later on fails to through flowering shoots (Gupta and Thind, 2006). Patel *et al.* (1970) reported during analysis of second major Black rot epidemics in cauliflower during 1968-69 that stump rot in cauliflower due to *E. carotovora* and other associated pathogens generally follow a severe infection of black rot. This had not been described previously from India and occurred in nearly all fields surveyed. Occurrence of soft rot on maturing cabbage heads were also reported from Udaipur (Rajasthan) by Chakravarti and Hegde (1972).

HOST RANGE

Soft rot disease caused by *Erwinia carotovora* has a very wide host range infecting large number of vegetable species belonging to different families. Walker (1998) has reviewed bacterial soft rot in crucifers and reports that if not all but many crucifers are susceptible to bacterial soft rot which include cabbage, cauliflower, brussel sprouts, kohlrabi, turnip, radish, rape, horse radish and rutabaga. The host range includes genera from all the plant families of vegetables, fruits and ornamentals, nearly about sixty four plant species are susceptible to the disease (Anonymous, 1990). Togoshi *et al.* (1988) isolated 52 isolates of bacteria from 15 different vegetable crops showing soft rot symptoms from Tsuruoka district of Japan, out of which 36 isolated were of *E. carotovora* subsp. *carotovora* and 16 were *E. carotovora* subsp. *atroseptica*. Phokim *et al.* (2006) conducted a DNA polymerase chain reaction assay using Y1 and Y2 primers specific to amplify 434 bp pectate lyase gene (Pel) of *E. carotovora* from ten different varieties of vegetables viz., cabbage, cauliflower, Chinese cabbage, pakchoi, spinach, carrot, Chinese radish, cucumber, tomato and asparagus. The result showed that primers Y1 and Y2 detected all isolates and were classified as soft rot *Erwinias*.

Rajeh and Khlaif (2000) during a survey in different areas in Jordan collected eighty seven isolates of bacteria from soft rot symptoms from different vegetable crops including, cabbage, cauliflower, chard, common mellow, *Gladiolus* sp., lettuce, onion, pepper, potato, spinach, sweet melon and tomato. Upon identification it is observed that cause of soft rot in all the vegetables is

E. carotovora. Bradbury (1986) has reviewed the host range of *E. carotovora* subsp. *carotovora* and reported that *B. chinensis* (Chinese cabbage) *B.o* var *acephala* (kale), *Cucumis sativus*, *Delphinium ajacis* *Glycine* max, *Helianthus tuberosus*, *Lupinus* sp., *Phaseolus vulgaris*, *Nicotiane tabacum*, as its hosts while *Solumum tuberosum* as main host of *E. carotovora* subsp. *atroseptica* but natural infection has also been reported for this sub-specie on cauliflower, cabbage, iris and tomato *E. carotovora* have been reportedly causing severe post harvest soft rot and stem end decay that result in losses to bell pepper in Florida (Sherman and Allen, 1983).

SEVERITY

There are no accurate measurements of fleshy fruits and vegetables to post harvest bacterial soft rots. They are estimated, however to vary between 15-30% of harvested crop (Agrios, 2006). The economic importance of losses caused by soft rot bacteria can be very great, depending upon the value of the crop and the severity of the attack. Although accurate estimates of the losses are not available, they may cause up to \$50 to 100×10⁶ annually on a world wide basis. The extent of these losses varies from country to country and is influenced by climate, conditions of growth and storage (Perombelon and Kelman, 1980). Walker (1998) reported that no actual figure of losses have been collected for all the hosts yet it probably is safe to assume that few other phytopathogenic organisms cause a greater total loss. He also describes the disease extremely destructive in some instances causing injury to cabbage seed plants and often follows black rot of cabbage. Severe post harvest losses had been reportedly incurred by bacterial soft rot or stem end decay in bell pepper during transit and marketing (Sherman and Allen, 1983). Ceponis and Butterfield (1974) reported sever market losses in metropolitan New York in cucumber and bell pepper which were produced in Florida. Bacterial soft rot has been said to be the most important single cause of wastage in stored potatoes (Harris, 1979). In Japan the disease has been reportedly been causing severe damage to vegetables including cabbage (Tatsuyama, 1961; Umekawa, 1991). Soft rot is also known to cause severe damage to cabbage during distribution under high temperature and poor transportation system in countries like Indonesia, Malaysia and Singapore (Higashio and Yamada, 2004). Farrar *et al.* (2009) observed that the potato in Keren County, California is affected by *E. carotovora* subsp. *carotovora* an estimated of 30% of the harvested produce is effected. They further reported that excessive post

harvest losses caused by *E. carotovora* subsp. *carotovora* have resulted in significant losses to San Joaquin Valley. Produce buyers for large national grocery outlets carefully scrutinize California potatoes for sunken and rotted tissue. Unsightly rotted or discolored lenticel tissues render the potatoes unmarketable and occasionally shipments of California potatoes are dumped. A survey of the post-harvest fruit rot diseases of tomato was conducted in five states of Nigeria. During severe infections, the diseases could cause 25% loss at harvest and 34% loss of the remaining product in transit, storage and market stalls; thus giving an overall loss of about 50% of the product. Two types of rots, soft and dry were recognised. The soft rot was found to account for about 85% of total losses (Fajola, 1978).

SYMPTOMS

Disease has much the same appearance on each host. The effected tissue becomes soft and slimy without much discoloration, but often accomplished by an offensive smell. Soft rot symptoms begin as small water soaked lesion, which enlarge rapidly. The effected area becomes soft and mushy while its surface become discolored and some what depressed tissue within effected region become slimy (Walker 1998). However crucifer plants and onions when infected by soft rot almost always give off a repulsive odor (Agrios, 2006). Walker (2004) in his description reports that the name of the disease aroused from characteristic soft decay of fleshy tissue. He further reports that fleshy tissue of storage organs have cells in a semi-dormant condition and content of carbohydrate is high, when soft rot effects, the tissue softens, becomes watery or slimy in consistency and as the rot progresses the water exudates from the effected portion.

PREDISPOSING FACTORS

Harvest bruises, freezing injury and insect wounds are predisposing factors. Abundant moisture at the surface of tissue where wounds are present is essential. After infection fairly high RH is essential for progress of disease (Walker, 2004). The bacteria that cause *Erwinia* rot are common on the surface of potato tubers, in soil and in surface irrigation water (Romberg *et al.*, 2002). Farrar *et al.* (2009) reported that during the lifting and harvest of potatoes, tubers can be smeared with soft-rot bacteria from decayed seed pieces. At the packing shed, potatoes are first dumped into a wash tank to clean them. Surface bacteria can be pushed into lenticels by hydrostatic (exerted by water) pressure in the wash tanks (Bartz and Kelman, 1985). Hydrostatic pressure increases

with increasing depth of the tank. Once inside the lenticel tissue, the bacteria multiply and cause lenticel rot. Several species of insects are known to carry the causal organism the relation of the insect with bacterial soft rot disease has been worked out and first reported by Leach (1926). Johnson (1930) described the relation of cabbage maggot and other insects to spread as well as development of soft rot in cruciferae. Similar observations were recorded by Bonde (1939) while working on blackleg of potato

Walker (1998) has summarized that when a high humidity is combined with a temperature of 80°F the pathogen is capable to cause greatest injury. The optimum temperature for its growth is 85°F the maximum slightly over 100°F. For this reason much of the loss due to this disease occurs during middle of the summer. Bhat *et al.* (2010) also reported higher growth of pathogen at 30°C and storage temperature ranging from 30 to 35°C as the most favourable for development of soft rot on cabbage. Temperature in range of 30-37°C were found optimum for soft rot development in different vegetable crop species (Farrar *et al.*, 2000). Raju *et al.* (2008) also reported enhancement of rotting ability of the pathogen *E. carotovora* subsp. *carotovora* at increased temperature of 20 to 30°C. They also reported that increased RH levels enhanced rotting. The highest rotting was recorded when radish discs were incubated at 35°C and 100%. Walker (2004) reported that when decaying roots effected by soft rot are placed in dry atmosphere the rot tissue dehydrated rapidly and further advancement of disease is checked out. Kikumoto (1974) during field observations using 3 varieties of Chinese cabbage reported that disease was prevalent from early summer to autumn and developed rapidly at high temperature the population of *Erwinia* reached to high levels during mid June and remained for six months then disappear from root zone or plant parts by following January. Clayton (1929) had earlier reported that stump rot caused by *E. carotovora* and other associated pathogens is unable to attack plants without the presence of black rot. Similar observations were recorded by Patel *et al.* (1970) during analysis of black rot epidemic in 1968-69 in Solan, in which they observed that stump rot presumably caused by *E. carotovora* generally follow severe infection of black rot. Frost injury has also been implicated as predisposing factor for the causation of soft rot. Kapoor (1987) reported that in valley areas of Kashmir and Himachal Pradesh where cabbage is grown for seed production the rotting starts just after frost, chilling after melting of snow this is because frost injury provides avenues for entry of bacterium. Galati *et al.* (2005) reported that disease becomes apparent under conditions of high soil temperature and moisture. In Western

Australia, bacterial soft rot is seasonal it is most severe under warm, wet conditions (summer and autumn) and it is usually not a problem under cooler weather conditions. Excess nitrogen fertilization and irrigation have both been associated with increased susceptibility to bacterial soft rot, as excess fertility or water promotes more succulent tissues, making the fruit more susceptible to physical damage during harvest and handling (Anonymous, 2006).

Production of enzymes: Enzymes that hydrolyze the middle lamella of host tissue in the development of soft rot were studied in great detail by Jones (1901) and referred the enzymes as pectinase. He reported that action of this enzyme produced by bacteria is to dissolve middle lamella, which serves to cement the adjacent cell walls together and thus to loosen the cells from one another. The by product of the bacterial growth also set ex-osmosis of liquid containing sugar and salts from within the cells to inter cellular spaces, where it un-doughtily serve as a source of food for further bacterial growth. The continuation of this process accounts for the watery condition and for the loss of consistency of the decayed tissue. Jones (1901) showed clearly that the pathogenic action was actually brought by the diffusion of enzymes in advance of bacteria and the later were really saprophytic upon the hydrolyzed lamella and ex-osmoses material. When soft rot bacteria enter wounds they feed and multiply at first on the liquid released by the broken cells on the wound surface then they produce increased amounts of pectolytic enzymes that break down pectic substance of middle lamella and bring about maceration of tissue. Bacteria continue to move and multiply in intercellular spaces; as a result cells plasmolyse, collapse and die in the intercellular spaces, while their enzymes advance ahead of them and prepare the tissue for invasion. The epidermis of most tissue is not attacked by the bacteria; however cracks are usually present and the slimy mass extrude through them (Agrios, 2006).

The capacity to produce pectic enzymes is wide spread in bacteria. According to most of the workers pectic enzymes which are currently known are, pectinesterases (PE or PME) and polygalacturonases (PG), pectinesterases or pectin methylesterases catalyse the hydrolysis of methyl ester groups of pectinic acid to methyl alcohol. It is of interest that rate of cleavage of α 1, 4 bond of pectic substances is enhanced by removal of methoxy group and this is illustrated by the fact that endopolygalacturonase prefers de-methylated pectin (Bateman and Miller, 1966; Mehrotra and Aggarwal, 2003).

Polygalacturonases or pectic glycosidase are chain splitting enzymes which break the links between adjacent galacturonic acid units in pectic substances this occurs by hydrolytic mechanism (hydrolases) or an eliminative mechanism (lyases) (Mehrotra and Aggarwal, 2003). There had been some confusion about chain splitting enzymes of pertinacious material; Wood (1960) followed the classification followed by Denim and Phaff (1957) who distinguished between the polygalacturonases (PG) which prefer Pectic acid of the de-esterified parts of pectinic acid as substrates and Pectin Methyl Galacturonases (PMG) which prefer the esterified chain parts. They further distinguished these polygalacturonases according to whether they cause cleavage in glycosidic linkage at random by an endo-type of polygalacturonases or polymethyl galacturonases, or by an exo-type of action in which the end linkages are preferred.

Albersheim *et al.* (1969) made a significant advance by discovering pectin transesterase (PTE) which split the pectin chain by a transesterinative mechanism. This enzyme breaks the glycosidic linkage of pectin at C4, accompanied by simultaneous elimination of H⁺ and C5. Protopectinases are thought to attack protopectin which is a native substance in cell wall, although the pectinases are widely used for macerating activity (Mehrotra and Aggarwal, 2003). Denim and Phaff (1957) devised a new system of classification where the macerating principal was shown to be associated with chain splitting enzymes polygalacturonases (PG and PME) Maceration of plant tissue has been associated with hydrolases and lyases which degrade the alpha 1-4 linkages in the galacturonic acid polymer of pectin substance in a random manner (Mehrotra and Aggarwal, 2003).

The work done by McDendon (1974), on purified pectin enzymes suggests that macerating enzymes are not of only one type. Brown (1965), Bateman and Miller (1966) emphasized that the non specific term macerating enzyme should be retained but all macerating enzymes which have been purified and identified till date have proven to be pectic enzymes (Mehrotra and Aggarwal, 2003).

There has been considerable variation among the isolates of *Erwinia* with respect to their pectinolytic activities, Gregg (1952) suggested that the quantitative pathogenic superiority in potato tuber of *E. carotovora* was compared with two other strains associated with production of greater quantity of pectolytic enzymes. Phokim *et al.* (2006) while assessing disease severity of *Erwinia* sp. by determining the activity of tissue macerating enzymes; pectate lyase, polygalacturonases, cellulose and protease with spectrometer and measuring the area of soft rot symptom on Chinese cabbage after artificial inoculation found out that *Erwinia* isolate from

carrot was the most severe because it produced higher macerating enzymes and macerated plant tissue with highest soft rot area in comparison to other soft rot *Erwinias* isolated from other 10 varieties of vegetables. Similar observations were recorded by Zaki *et al.* (1978) Smith and Bartz (1990) isolated thirty seven strains of soft rot *Erwinias* and they found significant differences among the strains for aggressiveness in tuber and fruits, but the relative aggressiveness on one host were not always associated with the pathogenicity in the other host.

CONTROL

A number of measures like through cleaning of storage house with formaldehyde (1 pint in 100 gallons of water) or copper sulphate (1 pound in 5 gallons of water), sorting out of any bruises or diseased vegetables and only perfect ones should be stored. It is further suggested to have sufficient ventilation to keep the humidity of air reasonably low, at the same time temperature should be as nearly 32°F, as possible. The trouble may be eliminated partly by long rotations with crops that are immune, as corn, cereals and grasses. In certain cases it may be practical to destroy the insects that may aid in disseminating the bacterium, or injure the host. Reporters also suggests that provisions should be made to control bruising at harvest, healing of wounds and drying of surface (Walker, 1998).

If diseased material had been stored previously in store house, such stores should be swept clean and thoroughly sprayed from ceiling to floor with a disinfectant solution like sodium hypo chloride (1000 to 1900 ppm), sodium o-phenylphenate (0.1 to 0.3%), calcium hypochlorite (700 to 5000 ppm), peracetic acid (3000 ppm) and disinfecting of tools by dipping in 70% alcohol, or 5% Formaldehyde solution (Anonymous, 1990).

Tanaka and Kikumoto (1976) reported no elongated lesions development when detached Chinese cabbage leaves were exposed to 30% R.H. for less than two hours at 24- 27°C before inoculation, they reported host cells were dead in lesions in both dried and non dried leaves Growth and movement of *E. carotovora* subsp. *carotovora* was inhibited in the dried leaves compared with that in non dried ones. In the dried leaves they reported no out flow of host cell contents or flow of water from living to dead cells and failure of development of lesions due to localization of bacteria in incubated area. Higashio and Yamada (2004) during a study recognized drying as one of the simple factors to control soft rot. They further reported that drying cut end contributes greatly to control and keeping cabbage indoors for more

then one day after the harvest was a more easy and simple way to prevent occurrences of soft rot in cabbage.

The appearance of soft rot symptoms in storage or in transit can be delayed by holding plants and produce at a temperature of 4° to 6°. Low temperature do not prevent bacterial infections, but slow down bacterial soft rot (Anonymous, 1990)

Few varieties have resistance to soft rot and no variety is immune (Agrios, 2006). However number of workers have reported more or less degree of susceptibility. Walker (1998) reported that among forty varieties of turnip that were tested for comparative susceptibility nearly all showed some rotting, the Jersey navel being the only one listed immune.

Kikumoto (1984) reported the number of bacterial cells recovered from inoculated sites of lesions being constantly higher in susceptible cultivars of Chinese cabbage.

Hot water treatment is recognized as a simple and easy technique to control the disease and insect damage and is already been put to practical use in many foreign countries (Gonzalez-Aguilar, 1999; Miura, 2001). However in a study conducted by Higashio and Yamada (2004) found no suppressive effect by dipping cabbage heads in hot water having temperature of 50°C. They found that in certain cases rot was promoted and concluded that lethal temperatures for soft rot bacterium was more than used in their study. Similar conclusions were made by Tasuyama (1961) during their studies on soft rot disease on Chinese cabbage.

As per Farrar *et al.* (2009) There are no effective chemical controls for any of the soft-rot *Erwinias*. As per Galati *et al.* (2005) chlorination is only recommended if carrots break down, because the disease occurs only under certain conditions. He suggested the use of sodium or calcium hypochlorite as the source of chlorine. Chlorination should take place after dirt and debris is removed from the carrot root. Chlorine quickly loses effectiveness when soil, leaves or diseased roots are present in the water, so it is important to check chlorine concentrations and pH often. Washing water should be changed as often as practical. However reportedly there are limitations to chlorination. Chlorination acts as a preventative measure only. If there is already disease, decay or injury in the field, chlorine will help limit spread of soft rot in storage. The disinfectant activity of a chlorine solution is determined by its pH, lower the pH greater the amount of hypochlorous acid available for disinfection. However vegetables and fruits are damaged by low pH chlorine solutions (Anonymous, 1990). In Japan hypochlorous acid is the only chemical used to disinfect fresh vegetables and is being used to control

microorganisms in shredded vegetables (Ishigaro, 2002). In the study conducted by Higashio and Yamada (2004), reported only a certain degree of soft rot suppression after hypochlorous acid application during post harvest management of disease. They further reported that effect on hypochlorous acid treatment did not occur at high humidity conditions. They stored the cabbage under various degrees of dryness after harvest and found that by keeping heads indoors for more than one day after the harvest had good effect in control of soft rot, which they attributed to healing of injuries of cabbage head which they receive at the time of harvest because it is thought that soft rot bacterium does not enter a plant without a wound.

Mills *et al.* (2006) tested several salt compounds as inhibitors of *Erwinia carotovora* subsp. *atroseptica* and *Erwinia carotovora* subsp. *carotovora* causing bacterial soft rot of potato. *In-vitro* studies with sodium metabisulphate, propyl paraben, alum, potassium sorbate, calcium propionate and copper sulphate pentahydrate showed that they were completely inhibitory at lowest concentration (0.002M). They further reported that as preventive disease control measures, tubers treated only with alum, aluminum acetate, calcium propionate, sodium bicarbonate sodium hypochlorite or copper sulphate pentahydrate resulted in less soft rot than untreated control, while as for curative disease control measures tubers treated with only alum, aluminum acetate, sodium hypochlorite, or copper sulphate pentahydrate resulted in significantly less soft rot than untreated control.

During a study conducted by Sherman and Allen (1983) on post harvest soft rot in bell pepper stored in jumble packed waxed corrugated cartons, reveals that inoculation with *Erwinia* greatly increased decay. This fact underscores the need for strict sanitation measures during pepper harvesting and handling procedures. Chlorination of vegetable packing house water gave conflicting results as washing with water alone before packing was as effective as washing with (150 ppm concentration). They further reported vacuum cooling of bell pepper was beneficial in reducing bacterial soft rot during storage and transit.

In Indonesia cement in which main constituent being calcium hydroxide application at the cut end was introduced to control soft rot (Tasuyama, 1961). The efficacy of cement was also confirmed by Higashio and Yamada (2004) who evaluated various calcium chemicals like calcium hydroxide, calcium carbonate, calcium sulphate dehydrate, calcium phosphate tribasic, calcium dihydrogen phosphate monohydrate and calcium hydrogen phosphate along with cement. Calcium proved effective, however some chemicals showed a tendency to

promote soft rot despite being calcium compounds. Because of which they considered that the effect of cement application was caused by the interception of bacteria at wounds rather than any physical function of calcium.

Harris (1979) while testing five chemicals against soft rot to batches of wounded potato tubers by dipping in a 1.5g Lit⁻¹ solution for 1 min reported, 5-nitro-8-hydroxyquinone as most effective in reducing rotting. 8-hydroxyquinoline, SD740823AX and chlorine dioxide also gave some control, while El badyne had no significant effect. However during in vivo tests El badyne and chlorine dioxide were less effective.

Not much work has been so far conducted to control bacterial soft rot by antibiotics. However certain reports pertaining to control of soft rot disease are those of, Kapoor (1999) who has recommended streptomycin in combination with copper oxychloride for control of this disease. He also reported that cruciferous like cauliflower seed crop and curd rot have been reportedly under check by the application of chloramphenicol + captofol (1:2). Mazzucchi and Svampa (1972) have reported that in green house test using inoculated courgette seedlings, antibiotic preparations based on streptomycin and / or tetracycline gave the best control of *E. carotovora* while copper compounds and a few experimental compounds gave a partial control. Good calcium fertility management in the field also reduces postharvest *Erwinia* losses. Calcium is integral to maintaining cell-wall rigidity and it counters the activity of soft-rot *Erwinia* enzymes, which degrade the cell walls (Farrar *et al.*, 2009). They further reported antimicrobial agents such as peroxyacetic acid and hydrogen peroxide, applied as a final rinse in the packing process, are effective in reducing the tuber surface populations of soft-rot organisms, resulting in less postharvest loss to *Erwinia* rot.

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