Colloquium Papers and Authors

Presiding: Carl E. Sams

Management of Postharvest Disease Resistance in Horticultural Crops: Introduction to the Colloquium

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The potential for significant economic losses from decay caused by postharvest pathogens is greater than is often realized. Harvesting and handling, and the attendant costs for labor and postharvest chemical treatments, can increase the cost of fresh fruits and vegetables several-fold from the field to the consumer. Therefore, any produce lost in storage is worth more economically than an equivalent amount lost in the field (National Academy of Sciences, 1978). Considering economics and the problem of world hunger, it is imperative to understand the importance of postharvest disease and to continually strive to reduce postharvest losses from pathogens.

In recent years, many international organizations have identified the necessity of reducing postharvest food losses as an important worldwide goal. While postharvest losses vary from season to season, an average loss of 10% to 30% after harvest is a realistic estimate (Hulse, 1982). Significantly higher losses are not uncommon in developing countries (Booth, 1974; Coursey, 1981). The extent of these losses justifies increased research efforts in postharvest pathology.

Chemical treatments and irradiation have effectively reduced the number of microorganisms on various commodities. However, consumer fears associated with chemical residues and the effects of ionizing radiation on fresh produce are forcing the development of alternative protection methods. Also, many pathogens have developed resistance to several commonly used chemicals, while other decay-causing microorganisms currently cannot be controlled by chemicals. We must, therefore, seek control strategies that will supplement or replace current disease control methods.

Postharvest diseases are the result of two types of infections: 1) those resulting from injuries incurred during harvesting and handling, or 2) latent or quiescent infections initiated during the growing season (Eckert and Ratnayke, 1983). In both wound and latent infections, natural antifungal metabolizes produced by the host play a role in host defense against decay. However, as fruit ripens, the concentrations of natural antifungal inhibitors drop to ineffective levels as ripening fruit tissue senesces. The onset of ripening and senescence in various fruits and vegetables renders them more susceptible to infection by pathogens (Kader, 1985). Therefore, delaying any change that is associated with ripening of the stored product can also delay the onset of susceptibility of fruit to decay and prolong storage life.

Refrigerated storage has been reasonably effective in delaying senescence and reducing losses due to postharvest decay. Combining refrigeration with various types of controlled-atmosphere storage has proven even more effective in reducing postharvest losses (El-Goorani and Sommer, 1981). Controlled-atmosphere storage delays senescence of the commodity and slows the growth and development of many pathogens.

Many postharvest pathogens can degrade the polysaccharides of the cell wall by using pectolytic enzymes. Many of these enzymes are produced inductively (Bateman and Basham, 1976), and the cell walls of immature fruits and vegetables may not yet have the substrates necessary to induce these fungal enzymes. Any factor that can delay the production or reduce the activity of these pectolytic enzymes can therefore reduce decay. The presence of proteinaceous inhibitors of pectolytic enzymes has been documented in some fruits and vegetables (Abu-goukh and Labavitch, 1983; Brown and Adikaram, 1982). Increasing the concentration of these inhibitors, prolonging their presence in effective concentrations, or even genetically engineering their presence in fruits and vegetables that do not contain them may be effective methods of reducing pectolytic enzyme activity and increasing resistance.

Calcium has been more closely related to disease resistance than any other cation associated with the cell wall. Calcium's role in forming cation cross bridges between pectic acids or between pectic acids and other polysaccharides with acid groups may make the cell wall less accessible to enzymes produced by fungal pathogens that cause decay (Tepfer and Taylor, 1981). In bean *(Phaseolus vulgaris* L.) tissue infected with *Rhizoctonia solani* Kuhn, tissue maceration by polygalacturonase was reduced as Ca content increased (Bateman, 1964). Increasing the Ca content of fruits and vegetables may be a way to decrease postharvest decay while reducing pesticide use.

Anew strategy that shows promise in controlling pathogens during storage is the management of microflora on the surface of fruits and vegetables to enhance resistance to postharvest pathogens (Wilson, 1989). The beneficial microflora are considered antagonists to pathogens that cause storage losses and, therefore, act as biological control agents. The mode of action of various biological control agents is being studied, as well as the optimum methods by which these agents can be incorporated into control strategies in the postharvest and storage environments.

The goal of this colloquium, through the papers that follow, is to consider various types of infections and mechanisms involved with postharvest diseases and to discuss recent advances in postharvest disease control strategies. Reducing postharvest losses will not only benefit the U.S. food supply, but also that of developing countries where there are continually increasing demands for food by growing populations.

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Mechanical Injury and Latent Infections Leading to Postharvest Decay

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Economic losses due to postharvest decays are higher than is often recognized. Because of the added costs of harvesting, packaging, transporting, and marketing, the value of fresh fruits and vegetables increases several-fold from the field to the consumer. These losses are accompanied by'" the nonproductive use of resources, such as land, fertilizer," pesticides, water, labor, and energy, during production. Pesticides are needed to produce and protect inexpensive fresh produce on a large scale. However, public concerns over food safety have become a major concern. Preharvest fungicide applications may have a beneficial carryover effect, i.e., in reducing postharvest decays. Postharvest fungicide applications are made routinely on many fresh-market fruits and vegetables. Consequently, fresh produce may contain fungicide residues within established Food and Drug Administration tolerances. In many cases, the cost of reducing postharvest diseases may be greater than the value of the fruits and vegetables at harvest. Farming operations that use integrated pest management, low-input agriculture, organic farming, and others will undoubtedly present us with new challenges in postharvest research. It becomes even more compelling that we as plant pathologists, horticulturists, and plant physiologists develop a cooperative team approach to undertake these challenges.

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INFECTIONS LEADING TO POSTHARVEST DISEASE

Numerous factors influence postharvest quality and shelf life. The preharvest environment and production methods have a large impact on postharvest diseases. Produce grown under less than optimum conditions are more likely to be predisposed to postharvest diseases than those grown under optimum conditions. Proper storage temperature and humidity are probably the most important postharvest disease control measures available. Refrigeration and high humidity retard senescence and the growth of many postharvest pathogens.

Some fungal pathogens are capable of penetrating intact surfaces of fruits and vegetables, although most enter at a wound site. Bacteria gain entry only through wounds or natural openings, such as stomata and lenticels. Fungi are typically involved in postharvest decays of fruit (Dennis, 1983), whereas vegetables may be attacked by either fungi or bacteria (Bartz and Eckert, 1987; Lund, 1983). Infections leading to postharvest decay can be separated into three categories: 1) incipient infections, 2) wound infections, and 3) latent or quiescent infections.

Incipient infections occur at any time before or after harvest. Incipient infections may remain active, causing decay, although they are often arrested as a result of exogenous influences, i.e., fungicide application and refrigeration. By definition, incipient means just beginning to exist or to come to notice. Many incipient infection; on produce may pass through the culling process at any time during harvesting, packaging, or retail display.