Title Mechanisms modulating fungal attack in postharvest pathogen interactions.

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Abstract

As biotrophs, insidious fungal infections of postharvest pathogens remain quiescent during fruit growth while at a particular phase during ripening and senescence the pathogens transform to necrotrophs causing typical decay symptoms. Exposure of unripe hosts to pathogens (hemi-biotrophs or necrotrophs), initiates defensive signal-transduction cascades that limit fungal growth and development. Exposure to the same pathogens during ripening and storage activates a substantially different signaling cascade which facilitates fungal colonization. This presentation will focus on modulation of postharvest host-pathogen interactions by pH and the consequences of these changes. Host pH can be raised or lowered in response to host signals, including alkalization by ammonification of the host tissue as observed in *Colletotrichum* and *Alternaria,* or acidification by secretion of organic acids as observed in *Penicillium* and *Botrytis*. These changes sensitize the host and activate transcription and secretion of fungal hydrolases that promote maceration of the host tissue. This sensitization is further enhanced at various stages by accumulation of fungal ROS that can further weaken host tissue and amplifies fungal development. Several particular examples of coordinated responses which follow this scheme are described, followed by discussion of the means to exploit these mechanisms for establishment of new approaches for postharvest disease control.