2

Apple Scab and its Management

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ABSTRACT: Apple scab caused by the fungus *Venturia inaequalis* (Cke.) Wint. is a destructive disease of apple. The pathogen is a facultative saprophyte that grows subcuticularly on the host. *V. inaequalis* must obtain nutrients through an active means. The fungus grows as a stroma of thick-walled cells between the cuticle and the outer wall of the host epidermis. Initial infections can lead to production of conidia on infected tissues within 9 to 17 days. The asexual spores can cause numerous secondary infections. Several waves of secondary infection can occur during a single growing season. Complete crop loss can result and severe infection can reduce blossom bud formation and crop potential for the following year, which may promote biennial bearing. Scab management is an essential component of orchard management in climates that are conducive to infection. Fungicides that are currently available for control of apple scab can be categorized as either protectant or eradicant in nature.

1. Introduction

Apple scab caused by the fungus *Venturia inaequalis* (Cke) Wint. is one of the most destructive of apple diseases. This chapter will consider literature pertinent to biology of the pathogen and current control strategies. Considerable information is available on the genetics of the pathogen and host and the reader is directed to the following references (Bolar *et al.*, 2000, Hemmat *et al.*, 1998, Le Cam *et al.*, 2002, Xu and Korban 2000, Xu *et al.*, 2001).

The pathogen is a facultative saprophyte that grows subcuticularly on the host (Nusbaum and Keitt, 1938). Because of this growth habit the pathogen must obtain nutrients through an active rather than passive mechanism (Nicholson, 1972, Nicholson *et al.*, 1977). The fungus grows as a stroma of short, thick-walled cells between the cuticle and the outer wall of the host epidermis (Figs. 1, 2, 3).
Germination, appressorium formation and penetration are the same on all apple hosts regardless of their resistance or susceptibility to the pathogen. In susceptible interactions, the stroma becomes thick and can cover an extensive area of host tissue, (Fig. 2). In resistant interactions, growth of the fungus is limited. In hypersensitive interactions, growth may be limited to only a few cells (Fig.4) (Nicholson, 1972).

Maeda (1970) demonstrated that the appressorium of the fungus contains a unique structure that she termed the appressorial infection sac (Fig. 5). Nicholson et al. (1972) demonstrated that during the germination process the conidial germling exhibits a transitory appearance of non-specific esterase enzyme activity (Fig. 6). Subsequently, one of the esterase enzymes was shown to be a cutinase (Köller and Parker, 1989) and this is consistent with the subcuticular growth habit of the fungus. The fact that cutinase is produced by the fungus was indirectly demonstrated first by Maeda (1970) who showed microscopic evidence that the fungus actively degrades the cuticle (Fig. 5). Cell wall degrading enzymes seem not to be particularly significant to the pathogen although literature suggests that V. inaequalis like other fungal pathogens produces cell wall degrading enzymes (Kollar, 1994, 1998).

Recently, Aylor reviewed the means through which the fungus is dispersed as well as some of the current strategies for disease control (Aylor, 1998). MacHardy (1996) also published a thorough review of the disease and its management.

It has recently been shown that apple contains receptor-like genes that are like the Cladosporium fulvum resistance genes in tomato (Vinatzer et al., 2001). Three members of the cluster were sequenced completely. As with the Cf gene family of tomato, the amino acid sequences coded by these genes contained an extracellular leucine-rich repeat domain and a transmembrane domain. It is interesting that Bolar et al. (2000) reported that in transgenic apple endochitinase from Trichoderma harzianum increased the level of resistance to apple scab. In related work, Xu et al. (2001) used a bacterial artificial chromosome (BAC) library of Malus floribunda 821 to investigate the apple scab resistance gene Vf. The resistance gene Vf, from the wild species Malus floribunda 821, was incorporated into a variety of apple cultivars through classical breeding. The aim was to isolate