INTRODUCTION

Traditionally, measures to prevent viral diseases have included breeding strategies to introduce natural forms of plant resistance from wild relatives into economically important crops. Thus, many crop plants have been selectively bred to incorporate specific resistance genes that target a wide variety of pathogens (Fraser, 1990). One shortcoming of this practice is the inability to transfer resistance across species barriers. Recent advances in biotechnology are overcoming this limitation as modern molecular techniques now provide the means to transfer resistance from one plant species to another (Whitham et al., 1996). However, as new resistance genes are introduced into plants, pathogens may evolve to overcome resistance. Fraser and Gerwitz (1987) examined over 50 virus-host interactions where resistance genes have been identified. They showed that fewer than 10% of these genes remained effective against long-term exposure to multiple virus strains. Understanding the molecular interactions between pathogens and the plant genes controlling resistance will allow for the development of new and better approaches to providing more effective long-term protection.

One of the most effective ways in which plants resist pathogen infection is through induction of the hypersensitive response (HR). The HR is an active defense mechanism that plants employ to prevent the spread of viral, bacterial, fungal, and nematode pathogens. An important feature of the HR is that it is a generalized response. Despite the different characteristics of the various types of pathogens, the same set of biochemical responses ensue: production of pathogenesis-related proteins, hydrolytic enzymes, callose and lignin precursors, oxidative bursts of H₂O₂, activation of systemic acquired resistance, etc. (Baker et al., 1997). These responses act in concert to restrict pathogen infection and prevent systemic disease. The end result of HR induction is localized cell death and necrosis at site of pathogen infection (Fig. 1).

In contrast to the generalized defense responses, induction of this phenomenon occurs in a highly specific manner. The ability of a plant to respond to the presence of a particular pathogen and initiate defense mechanisms implies that there is a specific recognition event between plant and the invading pathogen. Observations by Flor (1971) that resistance segregated with single dominant loci in plants and single
dominant loci in pathogens provided the framework for the current model termed the gene-for-gene hypothesis. This hypothesis suggests that plants carry specific resistance (\(R\)) genes, the products of which directly or indirectly interact with the products of pathogen encoded avirulence (\(avr\)) genes leading to induction of the HR (Keen, 1990).

**The Hypersensitive Response**

![Diagram of the hypersensitive response](image)

**Pathogen**
- Avirulence Gene

**Plant**
- Resistance Gene

**Gene-for-Gene Interaction**

- Hydrolases (chitinases and glucanases)
- Protease inhibitors
- PR proteins
- Ethylene biosynthesis enzymes
- Phytoalexins
- Aromatic metabolism synthesis
- AVF and IVR
- Enzymes for the production of:
  - lignin, suberins, phenolic polymers, polysaccharides, callose

**Resistance (HR)**

**Figure 1** Diagram outlining the steps leading to induction of the hypersensitive response and some of the biochemical responses involved. Leaf photograph shows an eggplant leaf that has been mechanically inoculated with TMV. Dark spots are local lesions resulting from HR induction.