

Molecular Biology of Plant Disease

S. SURYANARAYANAN

*Centre of Advanced Study in Botany,
University Botany Laboratory, Madras-5*

The principles of molecular biology are just beginning to be applied to the study of plant diseases. It is well recognised that genes and gene products of both the parasite and the host participate in host/parasite interactions. It, therefore, implies that gene activity of the parasite may be controlled by host genes and *vice versa*. Although the gene for gene concept is firmly entrenched in plant pathology, the underlying molecular mechanisms have not been elucidated. However, available evidence indicates that genetic activity of host and parasite may be controlled at the transcriptional, translational or enzyme level. Recent work suggests that the transcription pattern of host DNA may be changed during pathogenesis and inhibition of transcription of host DNA in infected cells may be mediated by histones.

The infection process by fungi is frequently accompanied by morphogenetic changes and the formation of infection structures would appear to depend on the synthesis of messenger RNA. Cell wall degrading enzymes seem to be under the genetic control of both the pathogen and the host. Resistance genes of the host are assigned functions in determining the type of glycosidic linkages in cell walls. Virulence may be reflected in the absence of a specific gene product. Mono- and disaccharides released from host cell walls may act as highly specific effectors for the induction and repression of polysaccharide-degrading enzyme synthesis by pathogens. Newer evidence also suggests that glycoproteins of plant cell walls may be analogous to animal antibodies. These proteins inhibit pathogen-

other enzymes, and distinguish between polygalacturonases secreted by different species of pathogenic fungi.

Synthesis of proteins and increased enzyme levels are ubiquitous phenomena in diseased tissues of plants. It is suspected that a *de novo* synthesis is involved owing to the triggering of silent host genes into activity by gene products of the pathogen. In incompatible host-parasite relationships this may lead to the synthesis of abnormal metabolites (phytoalexins) implied in disease resistance. It has been proposed that genes controlling the phytoalexin responses may be derepressed by compounds having the potential to change the conformation of double stranded DNA. Interaction between specific products of avirulent genes of the pathogen and specific receptors, controlled by resistance genes, on host cell membrane has also been invoked in explaining the gene for gene relationship in plant diseases.