Introduction

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Breeding for resistance against soilborne plant pathogens has achieved relatively less dramatic results than breeding against foliar pathogens. This difference may result from the basic differences between the environments of the two types of parasitic systems. The soil environment is extremely complex and the aboveground environment is relatively simple. Foliar parasitic systems are essentially two-component systems (one host versus one parasite) whereas the soilborne systems on a broad scale are usually multicomponent systems (one host versus many parasites). The multicomponents of the soilborne systems consist of the host, true and incidental parasites, and saprophytes. Mostly, soilborne parasites attacking a host do so as members of an interactive disease complex in which the soil organisms collectively modify each other's responses on a given host. The reaction phenotype on the host is, therefore, controlled by phenomena collectively termed "parasitic epistasis."

Parasitic epistasis is a type of parasite-parasite interaction in which one parasite interferes with the disease expression of another parasite. The parasite that modifies the disease expression is called "epistatic," and the parasite whose expression is altered, "hypostatic." The epistatic and hypostatic relationships are relative in a given host-parasite interaction; a particular parasite may behave as epistatic in one disease complex and hypostatic in another.

The importance of parasitic epistasis is not fully realized in plant parasitic systems in general, and particularly as it relates to the soilborne host-parasite interactions. There are many examples of induced resistance or induced susceptibility pertaining to the soilborne plant pathogens. The induction of resistance or susceptibility by one parasite against the other leads to modified (epistatic) phenotypic ratios (1). Such ratios are not mediated by the host genes, but occur due to the parasite-parasite interaction. As the complexity of interaction increases, the phenotypic ratios also become obscure and eventually lead to suggestion of a polygenic type of inheritance. The epistatic ratios or polygenic type of inheritance is often mistaken for the true genetic control of disease resistance and this can mislead the practice of breeding for resistance.

Improving understanding of the mechanism of parasitic epistasis may help in developing effective disease control measures against the soilborne parasites. The emphasis should be given to understanding the nature of epistatic and hypostatic relationships among the important soilborne parasites and possibly in relation to saprophytes. The genetic basis of hyperparasitism needs to be explored in order to manipulate and exploit the activity of parasites against other parasites. The understanding of the nature of parasitic epistasis and the genetic basis of hyperparasitism will lead to a sound basis for strategies of biocontrol and breeding for resistance against soilborne pathogens. The elucidation of the genetic basis for the interactions in a disease complex can provide answers for future breeding for resistance against soilborne pathogens.

The present symposium is more or less a sequel to the previous international symposia on various aspects of pathogenic and saprophytic behavior of soilborne plant pathogens. The genetical aspects of soilborne pathogens were briefly considered by the second international symposium held in conjunction with the First International Congress of Plant Pathology in 1968. The present symposium is an extension of some of the ideas that were previously developed and provides information on theoretical and practical aspects of exploiting the genetic variability of hosts and pathogens in breeding for resistance to soilborne plant pathogens.

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LITERATURE CITED

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