

The interactions between plants and phytopathogenic fungi are complex. The first studies of the processes involved pursued two questions: first what is the physiological and biochemical nature of the interaction; and second how are the interactions genetically determined? The latter approach was particularly important for breeding crops resistant to attack by phytopathogenic fungi. Different experimental systems were employed depending on the view-point and the questions emerging from it. Numerous observations and experiments with many different host-pathogen systems led to the discovery of a great many singular causal connections. Assembling these facts to make a rational and homogeneous picture of the processes going on during plant-pathogen interactions required a series of working hypotheses, some of which still have to be proved or disproved.

During the last twenty years it has become increasingly clear that substantial progress in understanding plant-pathogen interactions can only be achieved if the action of the genes involved can be characterized as precisely as possible, not only formally in terms like “suppression”, “activation” or “recognition”. From this type of analysis, plant pathologists were able to incorporate experimental results into working hypotheses that were derived from genetic analyses and physiological and biochemical investigations. This has led to the formulation of many new questions, which could be tackled by molecular biology and molecular genetics with newly designed experimental systems. The same new tools were also applied to reconsider the older working hypotheses.

Although we are still far from having answers to all of the open questions, our present knowledge and derived hypotheses offer a basis for attempting to integrate systematically many observations and conclusions. This book describes the complex network of plant-pathogen interactions with a general picture in mind. Some especially interesting new findings, and the conclusions and hypotheses emerging from them, will be described in more detail. Well established experimental systems, and their genetic backgrounds, are emphasized in the hope of inspiring the reader to ask further questions and/or to think about novel experimental systems.

To help the reader facts or ideas presented in other chapters are often repeated. Cross references are also given to chapter(s), where a topic is discussed in more detail or in a different context. The glossary and index are a further guide to facts and definitions. Suggestions for further reading are given at the end of

each chapter and are divided into “reviews” and “relevant papers”. Only “relevant papers” are cited in the text and refer to particular experimental work. The reviews cited are of a more general nature and are generally not cited within the text.

The following synopsis was prepared for readers who prefer an overview before becoming immersed in details. It contains commonly used technical terms (boldface printed in this chapter) with short, clear definitions and examples of their use. Unequivocal communication or description of phenomena and their connections requires clearly defined terms. Readers who find this dry and abstract can skip this section and start with chapter 2. In all the chapters new terms are defined and explained where they are first used. Should the reader get lost he can go back to the overview for re-orientation. Definitions of technical terms can also be obtained via the index. Page numbers in italics refer to the glossary.

How do plants and phytopathogenic fungi behave if they meet each other? Ordinarily, plants reject attacking phytopathogenic fungi. Parasitism is an exception to the rule. The result of the interaction between both partners, parasitic colonization of the plant by the pathogen, or failure of parasitism because the pathogen is repelled by the plant’s defenses, depends first of all on the genetic determinants of the plant and the pathogen. Most plants are not normally colonized and parasitized by most pathogens. They are **non-host plants** for these pathogens. Non-host plants were sometimes described as exhibiting **immunity** against pathogen attack. The reasons why plants are non-hosts may differ in detail, but in general the term designates a situation where the pathogen is unable to surmount barriers that prevent colonization of the plant. These various barriers comprise the **basic resistance** or **basic incompatibility** of the plant. The underlying mechanism of basic incompatibility may depend either on the plant, on the pathogenic fungus, or on both. Plant mechanisms may also protect against attack by quite different plant pathogens such as arthropods, nematodes, phytopathogenic bacteria or viruses. Occasionally the term **avoidance** is used when a plant manages to resist a pathogen attack, irrespective of the mechanism involved.

As mentioned above, successful attack of a plant by a pathogenic fungus is a rare exception and not the rule. Successful parasitism by the fungus depends on the production of **pathogenicity factors** acquired during evolution. As result of successful attack by a pathogenic fungus the plant becomes a **host plant** for that pathogen. Between both partners there exists a so called **basic compatibility**. This term embodies all physiological and biochemical requirements for colonizing and parasitizing a particular plant by a particular pathogenic fungus. During the process of colonization the pathogen withdraws nutrients from its host plant and lives and multiplies at its expense. This leads to biochemical changes within the plant that result in more or less conspicuous **disease symptoms** like yellowing of leaves, wilting, necrosis or distortions of plant form which reduce the vitality of the plant and, with time, may finally end in the death of plant parts or even the whole plant. – Basic compatibility is a highly specific phenomenon referring to

only a particular plant species and the appropriate pathogen species or *forma specialis*. On the contrary, basic resistance and basic incompatibility are rather unspecific phenomena observed among plants in contact with almost all pathogens.

Even though plant and pathogen may have evolved basic compatibility, the host plant may deploy defense strategies to limit pathogen attack. Two basically different defense strategies can be distinguished in terms of the physiological and biochemical mechanisms that result in resistance:

- (a) The plant cell(s) injured by the invading pathogen dies very rapidly causing necrosis of immediately adjacent tissue. In this way the pathogen is cut off from its nutritional substrate, is thus prevented from attacking further plant cells, and finally dies by starvation. This is the so called **hypersensitive reaction (HR)** or **hypersensitive cell death**. It is a rapid, and in many cases, macroscopically observable necrotic response of plant cells to pathogen attack.
- (b) The plant cell survives the pathogen attack and is induced to build up new defense barriers by *de novo* synthesis of mRNA and protein. This is called a **non-hypersensitive reaction**. HR can also lead to this response in adjacent tissues.

The interaction of a **host plant** with a basically compatible pathogen is called a **homologous interaction**. The pathogen able to parasitize this plant is an **homologous pathogen**. The interaction of a **non-host plant** with a pathogen is designated a **heterologous interaction**. In this case the pathogen is a **heterologous pathogen**, the plant exhibits basic resistance, and no colonization can ensue. However, when a pathogen interacts with certain non-host plants, a hypersensitive reaction may be triggered which then is termed a **heterologous hypersensitive reaction** or **heterologous HR**.

The homologous interaction of a pathogen with its host plant, and its colonization, is also called a **compatible** interaction of both partners. However, under certain conditions, such as the host plant has a gene for resistance against a particular homologous pathogen, the interaction becomes **incompatible**. This means the pathogen is rejected by its host plant although there is basic compatibility between both partners. This so called **host resistance** comes into action because the infecting pathogen induces defense reactions in the host plant. These block or restrain growth and reproduction of the parasite thereby restricting colonization of the plant. Host resistance presupposes, at least by definition, the existence of basic compatibility between the partners.

In the plant pathology literature, the terms compatible and incompatible are usually applied only to homologous interactions, i.e. to the interactions of a host plant with homologous pathogens. **Homologous compatibility** leads to colonization of the host plant and survival of the pathogen. On the other hand, **homologous incompatibility** means that the pathogen can neither parasitize its host

plant nor survive. An exception to this is the so called **heterologous incompatibility** which designates a **heterologous HR**, i.e. the appearance of an HR reaction between pathogens and certain non-host plants. – In cases where a compatible interaction does not result in the appearance of disease symptoms on the plant, or a significant reduction of crop yield, one speaks of **tolerance** of the plant towards the pathogen.

Phytopathogenic fungi follow two different strategies to ensure their nutrition; necrotrophy and biotrophy. **Necrotrophs** first kill their host cells before they can colonize them. Killing is brought about either by changes in the cell metabolism after infection, or is due to the action of **toxins** or **extracellular enzymes** produced by the infecting pathogen. (Necrotrophy of fungal pathogens is sometimes also called **perthotrophy** thus emphasizing these pathogens first kill their host cells before parasitizing them. In that case the term necrotrophy is confined to saprophytic, non-pathogenic fungi unable to kill plant cells and only feeding on already dead plant materials.) According to the range of activities of toxins synthesized by plant pathogens one distinguishes between **host specific toxins** or **host-selective toxins** on one side and **non-host-specific toxins** or **non-host-selective toxins** on the other.

Biotrophs depend on the metabolism of the infected host cells and surrounding plant tissue. This means that injury leading to host and tissue death is usually delayed until reproduction of the pathogen has been completed. Some biotrophs display during their life cycle first a biotrophic phase and later on, when the plant tissue collapses and dies, a saprophytic phase that exploits the metabolites present in the dead tissue.

As mentioned already, phytopathogenic fungi attack only particular plant species, i.e. they exhibit a limited **host range**. This means a pathogen can overcome the basic resistance of a plant within its host range and establish with it a basic compatibility. The host range of a pathogen is determined by the activities of its **pathogenicity genes** and their **pathogenicity factors**, respectively, “tuned” for this very host plant. In other words, distinct pathogenicity factors determine the specificity of the pathogen for a particular plant. However, pathogens such as *Botrytis* or several *Phytophthora* spp. exhibit little or no specificity for distinct host plants. Some pathogen species can be subdivided into **formae speciales** (**f.sp.**), comparable to the subspecies of a plant. The members of a *forma specialis* are generally confined to a single plant species (**species specificity**), or more rarely to several species.

The different defense mechanisms employed by plants against attacking pathogens can also be distinguished by the level at which they come into action: A pathogen is first faced with basic resistance (non-host resistance, general resistance, basic incompatibility, broad resistance or parasite non-specific resistance) which represents the **first level of pathogen defense** in a plant. When basic resistance is effective, the pathogenic fungus succeeds at best in establishing a transient contact with the plant, but is unable to colonize it. As mentioned above,

this is valid for most pathogens, except those for which the plant serves as a host. However, if this is the case, the pathogen actively breaks the defense barriers of basic resistance by employing its pathogenicity genes. These barriers may have existed before infection or have been induced to develop in the plant by the infecting pathogen. However, changes in the plant's physiological state may interfere with or hinder the expression of basic resistance.

Even when basic compatibility has been established and the plant is a host for the pathogen, other barriers may prevent parasitism and colonization. As mentioned above, a host plant may be resistant to attack by a particular homologous pathogen by inheriting a gene for resistance. In this case, the plant can present a **second level of pathogen defense** which represents **host resistance**. These defense mechanisms are, in contrast to basic resistance, highly specific for the pathogen since only certain races of a pathogen species will be rejected by the resistant cultivar. For that reason, this kind of defense is designated **cultivar-specific resistance** or **parasite specific resistance**. Other homologous pathogen races may not be affected by the particular cultivar-specific resistance and therefore are still able to parasitize that plant. Cultivar specific resistance is almost exclusively observed against attack by biotrophic pathogens. – In summary, the characteristic difference between basic resistance and host resistance of plants consists in the specificity of pathogen defense: The former is unselectively directed against all attacking pathogens, the latter is highly selective against only one particular pathogen species.

There are two different types of host resistance genes: **race-non-specific resistance** (horizontal resistance, uniform resistance or generalized resistance) and **race-specific resistance** (vertical resistance, specific resistance, or differential resistance). Race-non-specific (horizontal) resistance is directed against all members or races of a pathogen species. In contrast, race-specific (vertical) resistance confers resistance only against particular pathogen races. In the latter case a particular mutation to race-specific resistance in the host plant can specifically “recognize”, i.e. discriminate between, different races of the pathogen. Genetic determinants active against one particular pathogen race might even be found in different plant genera. The mechanisms and genetic determination of horizontal and vertical resistance are quite different. Both defense mechanisms are apparent only in the presence of basic compatibility.

Resistance, or the prevention of plant parasitism, can result from two situations: either the plant is not a host for the pathogen, i.e. the pathogen is blocked by basic resistance or basic incompatibility, or the plant is indeed a host but carries genes for parasite- or cultivar-specific resistance, which enable it, in spite of existing basic compatibility, to reject the pathogen on a second level of defense. Both resistance responses are similar and in some cases it may be difficult to determine whether defense is due to basic or cultivar-specific resistance. The specificity of plant resistance to pathogens can be summarized as follows: basic resistance (shown by non-host plants) is directed against nearly all pathogens,

race-non-specific resistance (shown by host plants) is against all races of a pathogen, or of a *forma specialis*, and finally race-specific resistance (also shown by host plants) is only against certain races of one particular pathogen or *forma specialis*.

In other words basic resistance (non-host resistance) and cultivar specific resistance (host resistance) are both terms that describe the inability of a phytopathogenic fungus to parasitize a plant. However, this inability refers to different situations: in cultivar-specific resistance (both horizontal and vertical) we may suppose the wild type host plant acquired by mutation(s) the ability to prevent a particular homologous pathogen from colonizing it. On the other hand, the term basic resistance designates the situation in which a wild type non-host plant can not be colonized by heterologous pathogens because the plant, the pathogen, or both, lack appropriate genes. Since the term “resistance” in colloquial usage refers only to a property of the plant but not of the attacking pathogen it would be more accurate to use the more comprehensive term **basic incompatibility** instead of basic resistance. In the phytopathological literature however, basic resistance is the more widely used term. – Recent research suggests that the molecular basis of release mechanisms for active non-host resistance (or basic incompatibility) and for race-specific or vertical resistance are quite similar or even alike.

Quite different defense mechanisms are observed against necrotrophic phytopathogenic fungi that produce host-specific toxins. Mutation to resistance against a host-specific toxin entails either its detoxification by the host plant or that the plant becomes insensitive to its action.

The colonization of a plant by a pathogenic fungus can be summarized as follows: First the pathogen can parasitize a plant only after it has overcome the plant's basic resistance and second, if it encounters host resistance, the pathogen must negate the corresponding race-specific resistance determinant. Overcoming race-non-specific resistance by pathogens has so far not been observed, whereas negating and overcoming race-specific resistance is by no means rare, since generally only one mutation of the pathogen to **specific virulence** is sufficient.

The term virulence is generally used in a quantitative sense to describe the extent of host plant symptoms. In contrast, specific virulence of a biotrophic pathogen describes a qualitative character, i.e. the ability, acquired as a result of a single step mutation, to overcome the race-specific resistance of the host plant. Specific virulence is either present or absent. Corresponding to the host range determined by pathogenicity genes one can define a “second” type of host range delimited by the race-specific resistance properties of the cultivar. Therefore, pathogens may have two different specificities: their **species specificity**, dependent on their possession of appropriate pathogenicity genes, and their **race-specificity** dependent on their ability to overcome race-specific resistance determinants in the host plant by mutation of the pathogen to specific virulence.

Reviews

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<http://www.springer.com/978-3-540-66727-8>

Plant-Fungal Pathogen Interaction

A Classical and Molecular View

Prell, H.H.; Day, P.

2001, XI, 214 p., Hardcover

ISBN: 978-3-540-66727-8