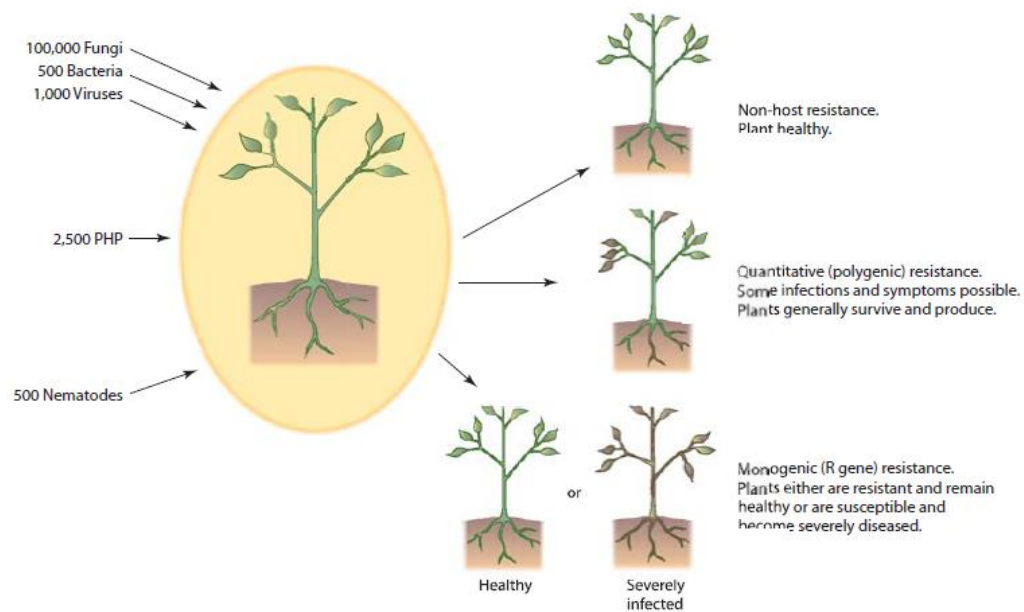


## WHATEVER THE PLANT DEFENSE OR RESISTANCE, IT IS CONTROLLED BY ITS GENES

One concept that must be made clear at the outset is that whatever the kind of defense or resistance a host plant employs against a pathogen or against an abiotic agent, it is ultimately controlled, directly or indirectly, by the genetic material (genes) of the host plant and of the pathogen (Fig).



**FIGURE:** Types of reaction of plants to attacks by various pathogens in relation to the kind of resistance of the plant.

### Non-host Resistance

A plant may find it easy to defend itself, i.e., to stay resistant (immune) when it is brought in contact with a pathogenic biotic agent to which the plant is not a host. This is known as non-host resistance and is the **most common form of resistance** (or defense from attack) in nature. For example, apple trees are not affected by pathogens of tomato, of wheat, or of citrus trees because the genetic makeup of apple is in some way (s) different from that of any other kinds of host plants, which, of course, are attacked by their own pathogens. However, apple can be attacked by its own pathogens, which, in turn, do not attack tomato, wheat, citrus, or anything else. Similarly, the

fungus that causes powdery mildew on wheat (*Blumeria graminis* f.sp. *tritici*) does not infect barley and vice versa, the fungus that causes powdery mildew on barley (*B. graminis* f.sp. *hordei*) does not infect wheat, and so on. All such unsuccessful plant/pathogen interactions are thought to represent non-host resistance. It has been shown recently however, that in at least some related pairings, e.g., the wheat, powdery mildew fungus inoculated on barley, the fungus produces haustoria and the host reacts by producing **hydrogen peroxide (H<sub>2</sub>O<sub>2</sub>)**, **cell wall appositions under the appressoria**, and a **hypersensitive response in which epidermal cells die rapidly in response to fungal attack**.

### **Partial, Polygenic, Quantitative, or Horizontal Resistance**

Each plant, of course, is attacked by its own pathogens, but there is often a big difference in how effectively the plant can defend itself (how resistant the plant is) against each pathogen. **Even when conditions for infection and disease development are favourable, a plant, upon infection with a particular pathogen, may develop no disease, only mild disease, or severe disease, depending on the specific genetic makeup of the plant and of the pathogen that attacks it.** Many genes are involved in keeping a plant protected from attack by pathogens. Many of these genes provide for the general upkeep and well-being functions of plants, but **plants also have many genes whose main functions seem to be the protection of plants from pathogens.** Some of the latter plant **genes code for chemical substances that are toxic to pathogens or neutralize the toxins of the pathogens**, and these substances may be present in plants regardless of whether the plant is under attack or not. **Plants also have genes that produce and regulate the formation of structures** that can slow down or stop the advance of a pathogen into the host and cause disease. These structures can also be **present in a plant throughout its life** or they may be **produced in response to attack by one of several pathogens or following injury by an abiotic agent**. Pre-existing defense structures or toxic chemical substances, and many of those formed in response to attack by a pathogen or abiotic agent, are important in the defense of most plants against most pathogens.

**When a pathogen attacks** a host plant, the genes of the pathogen are activated, produce, and release all their weapons of attack (enzymes, toxins, etc.) against the plants that they try to infect. With the help of different combinations of **pre-existing or induced toxic chemical substances or defense structures**, most plants manage to defend

themselves partially or nearly completely. Such plants show sufficient resistance that allows them to survive the pathogen attacks and to produce a satisfactory yield. This type of defense or resistance is known as polygenic, general, or quantitative resistance because it depends on many genes for the presence or formation of the various defense structures and for pre-existing or induced production of many substances toxic to the pathogen. This type of resistance is present at different levels against different pathogens in absolutely all plants and is also known as partial, quantitative, horizontal, multigenic, field, durable, or minor gene resistance.

Most plants depend on general resistance against their pathogens, especially non-obligate parasites, e.g., the semibiotrophic or necrotrophic oomycetes *Pythium* and *Phytophthora*, the fungi *Botrytis*, *Fusarium*, *Sclerotinia*, and *Rhizoctonia*, and most bacteria, nematodes, and so on. In at least some polygenic plant–pathogen combinations, such as the early blight of tomato caused by the necrotrophic fungus *Alternaria solani*, the more resistant the varieties are, the higher the constitutive concentration and the more rapid the accumulation in them of **pathogen-induced pathogenesis related (PR)** proteins, than in susceptible varieties. These PR proteins include some of the specific antifungal isozymes of chitinase and  $\beta$ -1,3-glucanase. Also, total enzyme preparations from resistant varieties were able to release **elicitors of the hypersensitive response (HR) (see later) from purified fungal cell walls**, whereas enzymes from susceptible varieties could not. Furthermore, partially purified chitinases from tomato leaves could release HR elicitors from germinating *A. solani* spores but not from mature intact cell walls. This suggests that, perhaps, **constitutively produced hydrolytic enzymes may act as a mechanism of elicitor release in tomato resistance to the early blight disease**. Quantitative resistance has also been shown to increase in **transgenic plants** carrying introduced R genes and matching avirulence genes, even though the latter do not express the hypersensitive cell death.

### **Race-Specific, Monogenic, R Gene, or Vertical Resistance**

In many plant–pathogen combinations, especially those involving biotrophic oomycetes (downy mildews), fungi (powdery mildews, rusts), and many other fungi, e.g., *Cochliobolus*, *Magnaporthe*, *Cladosporium*, many bacteria, nematodes, and viruses, defense (resistance) of a host plant against many of its pathogens is through the presence of matching pairs of juxtaposed genes for disease in the host plant and the pathogen. The host plant carries one or few resistance genes (R) per pathogen capable

of attacking it, while each pathogen carries matching genes for avirulence (A) for each of the R genes of the host plant. As explained in some detail later, the avirulence gene of the pathogen serves to trigger the host R gene into action. This then sets in motion a series of defense reactions that neutralize and eliminate the specific pathogen that carries the corresponding (matching) gene for avirulence (A), while the attacked and a few surrounding cells die. This type of defense or resistance is known as race-specific, hypersensitive response (HR), major gene, R gene, or vertical resistance. However, some R genes, e.g., Xa21 of rice, do not induce a visible HR.