

GENES AND DISEASE

When different plants, such as tomato, apple, or wheat, become diseased as a result of infection by a pathogen, the pathogen is generally different for each kind of host plant. Moreover, the pathogen is often specific for that particular host plant. Thus, the fungus *Fusarium oxysporum* f. sp. *lycopersici*, which causes tomato wilt, attacks only tomato and has absolutely no effect on apple, wheat, or any other plant. Similarly, the fungus *Venturia inaequalis*, which causes apple scab, affects only apple, whereas the fungus *Puccinia graminis* f. sp. *tritici*, which causes stem rust of wheat, attacks only wheat. What makes possible the development of disease in a host is the presence in the pathogen of one or more genes for pathogenicity, for specificity, and for virulence against the particular host.

The gene(s) for virulence in a pathogen is usually specific for one or a few related kinds of plants that are hosts to the pathogen. Also, the genes and gene combinations that make a plant susceptible, i.e., a host to a particular pathogen, are present only in that one kind of plant and possibly a few related kinds of plants. All plants also have pre-formed and induced defenses that provide resistance against most pathogens. **The specificity of microbial virulence genes that condition growth and disease on particular plants explains why a pathogen that is virulent on one kind of plant is not able to attack other kinds of plants and why a plant that is susceptible to one pathogen is not susceptible to all other pathogens of other host plants.** This is known as non-host resistance (Figures).

Of course, a few pathogens are able to attack many kinds, sometimes hundreds, of host plants. Such pathogens tend to be **necrotrophs** and can attack so many hosts apparently because they either have many diverse genes for virulence or, more likely, because their genes of virulence somehow have much less plant specificity than those of the commonly more specialized pathogens. Each species of

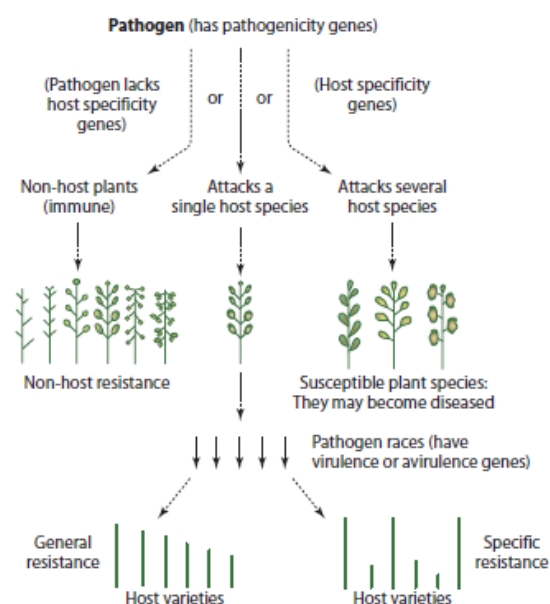


Figure: General Interaction of Pathogen with its host plant and Non-Host Plant

Plant Resistance to Disease

plant, however, seems to be susceptible to a fairly small number of different pathogens, usually less than a hundred for most plants. Despite the many pathogens that can infect them, sometimes a few and many times countless numbers of individuals of a single plant species, such as corn, wheat, or soybean, survive in huge land expanses year after year. These plants survive either free of disease or with only minor symptoms, even though most of the other plants in the field have been killed (Fig) and their pathogens

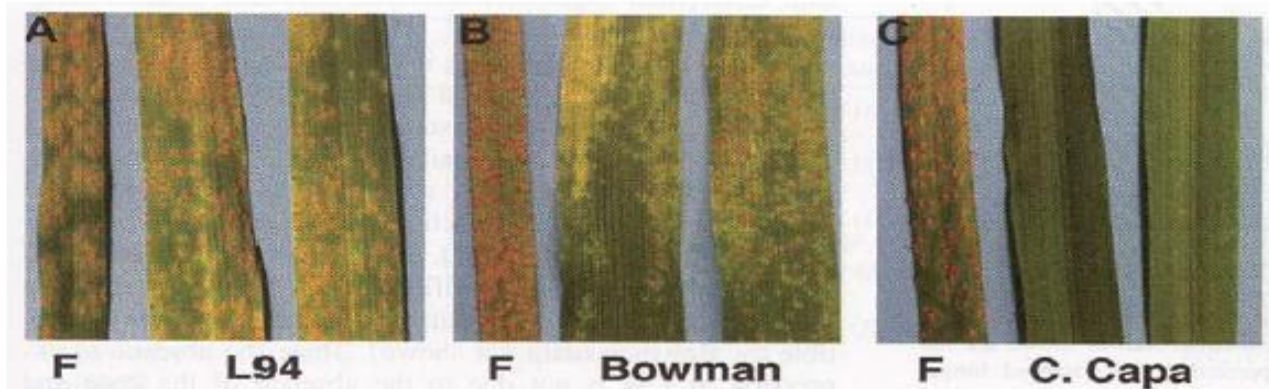


Figure: Infection type of two seedling leaves from each of three barley cultivars after 10 days of artificial inoculation with inappropriate wheat leaf rust *Puccinia triticina*. Wheat cultivar F was used as susceptible control. Only the cultivar *C. capa* behaved as non-host. Infection on other was bridged by pathogen presumably but apparently not limited to wheat

are often widespread among the surviving plants. **Why all the plants are not attacked by their pathogens? And why are those that are attacked not usually killed by the pathogens?** The answer is complex, but basically it happens because plants, through evolution or through systematic breeding, **have acquired**, in addition to the genes that make them susceptible to a pathogen, **one or usually numerous genes for resistance** that protect the plants from infection or from severe disease. When a new gene for resistance to a pathogen appears or is introduced into a plant, the plant becomes resistant to all or most of the previously existing individuals of the pathogen. Such pathogens contain one and usually more than one gene for virulence, but if they do not contain the additional new gene for virulence that is required to overcome the effect of the new resistance gene in the plant, they cannot infect the plant and the plant remains resistant. Thus, **even one new gene for resistance to a pathogen can protect plants that have the gene from becoming infected by all or most pre-existing races of the pathogen — at least for several months and possibly for several years.**

It has been the experience of researchers with numerous host–pathogen combinations, however, that, after a **new gene for resistance** to a pathogen is introduced into a crop variety and that variety is planted in the fields, a **new population (race) of the**

pathogen appears that contains a **new gene for virulence** that enables the pathogen to attack the crop plants containing the new gene for resistance. **How did this new population of pathogens acquire the new gene for virulence?** In most cases the new gene had already been present earlier at low levels, or by mutation, but only in a few pathogen individuals.

New genes can arise randomly and suddenly *de novo* through mutations, or by rearrangement of the genetic material of the pathogens through the ever-ongoing events of **genetic variability in organisms**. Such pathogen individuals may have been but a tiny proportion of the total pathogen population and were undetected before plants with the new resistance gene were planted widely. After such plants were introduced, however, the new resistance gene excluded all other pathogen individuals except the few containing the new gene for virulence, which could attack these plants. Exclusion of the pathogens that lacked the new gene allowed the few that carried the gene to multiply and take over.

GENETICS OF VIRULENCE IN PATHOGENS AND OF RESISTANCE IN HOST PLANTS

Infectious plant diseases are the result of the interaction of at least two organisms, the host plant and the pathogen. The **properties** of each of these two organisms are governed by **their genetic material, the DNA**, which is organized in numerous segments making up the genes. It has been known for more than a century that the **host reaction, i.e., the degree of susceptibility or resistance to various pathogens, is an inherited characteristic**. This knowledge has been used quite effectively in breeding and distributing varieties resistant to pathogens causing particular diseases. The ability of pathogens to inherit their infection type, however, i.e., the degree of pathogen virulence or a-virulence, has been studied intensively only since the 1940s. It is now clear that pathogens consist of a multitude of races, each different from others in its ability to attack certain varieties of a plant species but no other varieties. Thus, **when a variety is inoculated with two appropriately chosen races of a pathogen, the variety is susceptible to one race but resistant to the other. Conversely, when the same race of a pathogen is inoculated on two appropriately chosen varieties of a host plant, one variety is susceptible while the other is resistant to the same pathogen (Table)**. This clearly indicates that, in the first case, one race possesses a genetic characteristic that enables it to attack the plant, while the other race does not, and, in the second case, that the one variety possesses a genetic characteristic that

Plant Resistance to Disease

enables it to defend itself against the pathogen so that it remains resistant, while the other variety does not. When several varieties are inoculated separately with one of several races of the pathogen, it is again noted that one pathogen race can infect a certain group of varieties, another race can infect another group of varieties, including some that can and some that cannot be infected by the previous race, and so on (Table).

Table: Possible Reaction of Two and Four varieties of plants to two and four races of pathogens

Plant	Pathogen Race		Plant	Pathogen Race			
Variety	1	2	Variety	1	2	3	4
A	-	+	A	+	-	-	-
B	+	-	B	-	+	+	+
			C	-	+	-	+
			D	+	-	+	-

Plus sign indicates susceptible (Compatible reaction, Infection): Minus sign indicate resistant, incompatible, non-infectious

Studies of the inheritance of resistance versus susceptibility in plants prove that **single genes control resistance and their absence allows susceptibility**. Studies of the **inheritance of a-virulence versus virulence in pathogens prove that single genes control a-virulence and their absence allows virulence**. Studies of their interactions prove that R genes in the plant are specific for avr genes in the pathogen. Thus, varieties possessing certain genes for resistance react differently against the various pathogen races and their genes for a-virulence. **The progeny of these varieties react to the same pathogens in exactly the same manner as the parent plants**, indicating that the property of resistance or susceptibility against a pathogen is genetically controlled (inherited). Similarly, **the progeny of each pathogen causes on each variety the same effect that was caused by the parent pathogens**, indicating that the property of virulence or a-virulence of the pathogen on a particular variety is also genetically controlled (inherited). It thus appears that, under favourable environmental conditions, the outcome — infection (susceptibility) or non-infection (resistance) — in each host–pathogen combination is predetermined by the genetic material of the host and of the pathogen. The number of genes determining resistance or susceptibility varies from plant to plant, as the number of genes determining virulence or a-virulence varies from

pathogen to pathogen. In most host–pathogen combinations the number of genes involved and what they control are not yet known. In some diseases, however, particularly those caused by oomycetes, such as potato late blight, fungi, such as apple scab, powdery mildews, tomato leaf mold, and cereal smuts and rusts, and also in several viral and bacterial diseases of plants, considerable information regarding the genetics of host–pathogen interactions is available.