GENETICS IN RELATION TO DISEASE RESISTANCE

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I. Introduction

Disease has been an ever-present ravage of crop plants throughout agricultural history. Man has attempted to cope with the disease problem by defending his plant, by attacking the parasite or when all else fails by taking evasive action. Undoubtedly the most attractive form of defence is provided by disease resistance, for as long as it remains effective it provides protection at no cost to the farmer or the community. In some instances resistance remains stable for long periods, for example, flag smut resistance first incorporated in Australian wheat varieties by Farrer and resistance to loose smut in NP 790 wheat still provide stable protection. Unfortunately, however, disease resistance often loses its effectiveness after a relatively short period. The average commercial lifetime of rust-resistant wheat varieties in Mexico has been only four years and in Columbia five years. This loss of effectiveness is not due to a loss of resistance genes from the host plant but to the acquisition of new virulence genes by the pathogen. These new virulence genes overcome the action of the genes in the host which previously conferred resistance.

While we see this happening in a very dramatic fashion in some varieties, wild species appear to have more stable forms of resistance. Perhaps there is a lesson to be learned from a study of the wild species that will help to provide more stable protection for crop plants.

II. Resistance among insects to pesticides

In Nicaragua, chemical control of cotton pests in the beginning, gave excellent results in increasing the cotton yield to become the highest in the world. This initial success led to a large number of contracts with pesticide firms to carry out chemical control schedule in a routine fashion. This indiscriminate chemical control resulted in the serious upsetting of the balance in nature and a great increase in the resistance of pests to pesticides used. The combined effect of these two phenomena was that within a couple of years, they had to give up cotton cultivation. Another spectacular case of resistance has been reported from Japan. In one rice area of Japan the malathion, which had been used successfully to control the rice green leafhopper, Nephotettix cincticeps began to lose its effectiveness in 1955. The confirmation of resistance developed in this pest led the research for alternative chemicals which took so much time that malathion was replaced by Carbaryl in 1965.

There are two cases of resistance in agricultural pests included in F.A.O. list for India. These are: (1) the Singhara Beetle (Galerucella birmanica) and (2) the tobacco cutworm Spodoptera (Prodenia) litura. A species of cattle tick (Boophilus microplus) is also there in the F.A.O. list.

III. Evolution of resistance

Natural selection has resulted in the evolution of two genetically different forms of protection against disease. Specific genes in the host provide high-level resistance against particular strains of the pathogen. This I will call resistance. A lower level of protection also occurs which I will call tolerance. Tolerance is not strain specific, and is generally controlled by the interaction of several different genes. My use of the terms resistance and tolerance broadly conform to the vertical and horizontal resistance of Van der Plank (1963) or the specific and generalised resistance of Hooker (1967).

Resistance results from the parallel evolution of the host and the pathogen. Alternate selection pressures of the host, for resistance, and on the pathogen, for virulence, result in the build-up of a complementary genetic system with a gene-for-gene relationship between the genes for resistance in the host and those for virulence in the pathogen. Susceptibility results only when all resistant genes in the host are matched by the complementary virulence genes in the pathogen.

As genes for resistance are accumulated a complex system is built up, protection the host against the full range of strains of the pathogen to which it has been exposed throughout its revolutionary history. To overcome this resistance, the pathogen also has to accumulate the complementary virulence genes. This accumulation of virulence genes in the pathogen tends to reduce its competitive ability. When hosts are available which lack accumulated resistance genes the pathogen tends to lose all necessary virulence genes.

In the host, however, the accumulation of resistance genes does not appear to have the same deleterious effect, and as the host has less genetic and population flexibility the complex genetic system for resistance tends to be maintained.

Examples of the gene-for-gene relationship between host and pathogen are to be found in the flax-rust system where it was first described by Flor (1956); with potato late-blight resistance in Solanum (Toxopeus, 1956); with stem rust of wheat (Watson, 1958); with leaf mould of tomatoes (Fincham & Day, 1965); and in numerous other less well documented cases.

Examples of the reduction of competitive ability or fitness of the pathogen with the accumulation of virulence genes have been cited by Van der Plank (1963); Black (1952, 1960); Watson (1958) and Day (1966).

Compared with the large amount of breeding for disease resistance in crop plants and the extensive use of wild species as sources of resistance, relatively little effort has been devoted to studying the genetic of disease resistance in naturally occurring species. The genus studied most extensively is Solanum, particularly S. demissum which has provided a number of genes for resistance to late blight (Niederhauser et al, 1954). Nicotiana species have also provided sources of resistance to a number of diseases (Clayton, 1954; Burk & Heggestad, 1966) although in most cases little is known of the

gene-for-gene relationship existing between the host and pathogen. Wark (1963) has extracted a single major gene conferring resistance to a specific race of tobacco blue mould from three different Nicotiana species and he has also demonstrated the existence of polygenes, conditioning disease reaction in at least one species. The major gene does not provide resistance against other races of the pathogen (Wark et al, 1960; Hill, 1963). The majority of the genes conditioning resistance to rust in wheat, flax and oats have been isolated in cultivars and although they probably originated from wild species this has not been extensively documented.

Relatively little is known about the distribution of virulence genes within the genotype of the pathogen, but in the host the resistance genes tend to be widely distributed throughout the genotype. This provides ample scope for the accumulation of genetic systems for resistance. Although there are some reported cases of multiple allelic systems for resistance which would reduce this flexibility (Mayo, 1956; Person, 1959), several such systems when subjected to close study have been shown to be closely linked genes and not multiple alleles (Person, 1959).

From an evolutionary point of view the gene-to gene system of disease resistance has one major disadvantage. It leaves the host completely unprotected from the time when a virulence mutation occurs in the pathogen until resistance is restored by the occurrence of a new resistance gene in the host. Hence it is not surprising that an alternative system of protection should have evolved to operate during the alternating periods of susceptibility.

IV. The Evolution of Tolerance

The genetic nature of the systems conditioning tolerance are not as well understood as the gene-for-gene system conditioning resistance. Stable tolerance systems have been reported from several species (Van der Plank, 1963). They are not restricted to specific stains of the pathogen and most are quantitatively inherited, indicating the interaction of numerous genes. Selection for tolerance would begin to operate as soon as single gene resistance failed and would continue until high-level protection was restored by a new mutation in the host. Selection would favour the incorporation of tolerance genes which restrict the reproductive rate of the pathogen because such genes would reduce the rate of disease spread and hence the severity of epidemics. The reduced reproduction of the pathogen would also assist in stabilizing the protection provided by resistance genes by reducing the frequency of occurrence of new virulent mutants.

It is not known whether the genes for tolerance are independent of the resistance genes or whether the resistance genes assist in providing tolerance.

Thus, the host is protected by two genetic systems, major genes providing resistance against specific strains of the pathogen and polygenes providing tolerance against a broad spectrum of strains. The pathogen on the other hand, tends to retain only those genes for virulence which are currently functional.

V. What happens under Agricultural Systems?

When a susceptible variety is grown under conditions favourable for disease development, or when resistance breaks down, the plant breeder is called upon to provide resistant varieties. When this happens for the first time the breeder has no alternative but to test a range of genotypes against the pathogen that is present. Because of the high reproductive capacity of the pathogen and the deleterious effects of carrying many virulence genes this usually results in the tests for resistance being conducted with a small range of the potential variability in the pathogen.

When resistance is located it has to be transferred to genotypes that are agriculturally acceptable. As this is a lengthy process, involving from six to eight plant generations in the simplest cases, and many more when resistance has to be transferred from a non-agricultural species, the pathogen has time to exhibit its potential for variation. Additional virulence genes are acquired by mutation, sexual recombination, or somatic recombination. The resistant genotypes being developed in the breeding programme provide the selection sieve for the multiplication of the new strains of the pathogen. Hence it is quite common for the plant breeder to be aware of the existence of a strain of the pathogen capable of breaking down the resistance of his advanced selections before they can be released for commercial use.

The probability of chance inclusion of additional resistant genes is low because of the distribution of major loci for resistance throughout the genetic complement of the host.

Hence resistant genotypes tend to have only one or a few genes for resistance and a relatively simple genetic change in the pathogen will render them susceptible. The plant breeder then has to start again.

The efficiency of a disease-resistant breeding programme is generally measured by the speed with which the breeder can locate and incorporate resistance into acceptable agricultural genotypes. These factors tend to favour the inclusion of simple genetic systems which are most easily broken down. Naturally these problems apply to a lesser degree when there is extensive knowledge of the variation in the pathogen and the resistant sources in the host, e.g., wheat rust, nevertheless the problem of incorporating something analagous to the complex system protecting naturally resistant species into an agricultural genotype and maintaining an 'efficient' breeding programme is a very real one.

What plant-breeding techniques can be employed to counter this situation?

Ideally, one wants to incorporate both tolerance and resistance. To do this the breeder must have available a full range of variation in the pathogen, which is impossible, and have techniques for manipulating a number of resistant genes, which in most cases is impracticable.

Techniques for manipulating genes are available but they require accurate measurement of phenotypic expression. This in turn requires not only close cooperation between breeder and pathologist, but in many cases better knowledge of the effects of environmental conditions on disease development than is available. Accurate control of environmental conditions is also essential.

There are several alternatives to this hypothetical ideal which are being used, and in numerous cases very efficiently used, to keep the ravages of plant diseases at bay. Despite the variability of the rust fungus and the occurrence of several new races, no substantial losses due to wheat-stem rust have occurred since 1947 in areas of Australia where breeding has been carried out (Watson & Luig, 1966). This success has been achieved by skilful manipulation of the genetic diversity available in the host (Luig & Watson, 1965) rather than by the development of stabilised genetic protection. It is an unfortunate fact that most disease-resistance breeders are breeders for resistance or breeders for tolerance; rarely are both approaches combined.

VI. Breeding for Resistance

a) New resistant genes: The continued search for new resistance genes and the successive replacement of varieties, combined with a close study of the variation in the pathogen, can often provide an effective answer to the disease as a field problem. This method is most likely to be successful if the pathogen population changes slowly and there is control of the host genotypes grown. It is a necessary first approach if knowledge is lacking and provides sources of resistance for other approaches.

b) Combined resistance: The incorporation of all or several genes for resistance into a common genotype is theoretically desirable but practically difficult to achieve. Breeding populations are necessarily very large when three, four or more genes are segregating. When genes from non-agricultural species are being incorporated, closely linked deleterious genes are a problem in maintaining yield and quality. Accurate control of the strains of pathogen is essential for this method.

c) Multi-line resistance: Multi-lines or composite varieties which consist of mechanical mixtures of phenotypically similar lines, each of which carries a different gene for resistance, were proposed by Borlaug (1958). The system is based on the hypothesis that the sparse distribution of susceptible genotypes to any particular strain of the pathogen will delay disease development. This will not only reduce damage but by keeping the population of the pathogen at a low level will reduce the probability of occurrence of new virulence genes. This method is theoretically very promising but so far it has only been tested on a very limited scale (Borlaug, 1965; Derera & Watson, 1965).

d) Novel sources of resistance: Techniques for the induction of mutations offer the prospect of locating and using novel specific genes where these are not available or readily useable from other sources. While most of the genes for disease resistance that have been induced by mutation techniques in crop plants are similar or identical to genes already available in world gene pools, novel variants have occurred (Favret, 1965).

Translocations following chromosome breakage and the development of genetic techniques permitting chromosome substitution and addition offer the prospect of utilizing genetic material from allied species where this cannot be incorporated by crossing. Such genes or blocks of genes may provide greater stability than the more readily available sources of resistance. However, such transfers are likely to include genes deleterious to yield or quality.

VII. Breeding for Tolerance

Theoretically, breeding for tolerance is highly desirable, particularly when it is combined with breeding for resistance. However, there are many practical difficulties. The breeder must be particularly careful in defining his selection criteria. Yielding ability of the plant is probably a better measure of tolerance than disease symptoms, but attention must be given to the rate of reproduction of the pathogen. As the breeder will be dealing with a quantitatively inherited character he must be able to handle large populations and to distinguish small phenotypic differences. There must be control of the strain composition of the pathogen and cooperation with pathologist is essential if efficient selection techniques are to be developed.

About a decade ago, Prof. D. Lewis propounded the dictum "Breed plants against insects and develop chemicals for controlling diseases". This was based on the concept that while physiologic specialization is common in fungii, it is rare or absent in insects. The work done in potato on breeding for resistance to the golden nematode does not, however, substantiate this view. A summary of the situation in potato taken from the paper of C.A. Huijsman (Euphytica 13: 223-228, 1964) is given here.

VIII. Breeding for resistance to the Golden Nematodes in Potato

The first indication of genetic variability in the potato-root eelworm Heterodera rostochiensis WOLL. was found by Van Der Laan and Huijswan (1957) after testing resistant offspring of CPC 1673 (Solanum tuberosum subsp. andigena) against cysts from Peru.

In 1955, DUNNETT in Scotland discovered that certain populations of the parasite could multiply strongly in the roots of resistant potato varieties derived from CPC 1673 (DUNNETT, 1957). These aggressive populations are indicated by the letters AB; the non-aggressive ones, which are not capable of multiplication in the above mentioned material are indicated by the letter A(6).

THE AB-POPULATIONS OF THE POTATO-ROOT EELWORM

Testing for AB-populations is carried out as follows: A number of 12 cm pots is filled with a soil mixture from the field to be tested. In half of them a tuber of a susceptible variety is planted and in the other half a tuber of a resistant variety bred from clone CPC 1673. After 6-8 weeks the roots at the surface of the pot balls are examined. If cysts are found on the roots of all plants then the field concerned is infested with an AB-population; if cysts occur only on the roots of the susceptible variety then an A-population is present. If no cysts are found then the infestation of the pot soil must have been too slight and the test has failed.

The larvae of an AB-population have a genetic constitution which enables them to multiply strongly in the roots of plants incorporating the resistance of the clone CPC 1673. Nothing is known as yet about the gene or genes determining this behaviour but whether or not a population can be classed as an AB-population must depend ultimately on their frequency in the gene-pool of that population. Only populations with a high frequency will be recognised as AB-populations by the foregoing method. Once case has been described in which the frequency was so low that resistant varieties were grown for nine successive years before an attach occurred which suggested that such genes might after all have been present in the original population (HUIJSMAN, 1963).

If the aggressiveness results only from a combination of genes one can imagine populations in which those genes are present, but in which not a single larvae has the combination required for inclusion in the AB races. It is possible that the required combination of genes is brought about only after growing varieties with the resistance of CPC 1673 for a number of years. From then onwards aggressiveness towards the material with CPC 1673 resistance is detectable. Summing up, the data obtained by the Plant Protection Service must be interpreted as follows:

a) 24% of the infested fields in the Netherlands have populations with a high frequency of genes for aggressiveness towards CPC-1673 material.

b) 76% of the infested fields have populations with a very low frequency of these undesirable genes. Some of these fields may even be infested with populations in which this frequency is zero.

IX. The prospects of the resistant potato varieties

a). The resistant varieties as offspring of CPC 1673: When resistant varieties bred from CPC 1673 are grown on plots infested with populations which are aggressive towards this material - in the Netherlands about 24% of the total infested area - the infestation of the soil will increase. Dependent on the initial degree of infestation and its uniformity over the plot several crops of resistant varieties can be grown before the population-increase is measurable. After that it is useless to plant more of this material.

In areas infested with non-aggressive populations a great decline in the degree of infestation will result from the growing of resistant varieties. This will make it possible to obtain good yield even of susceptible varieties, while the danger of spreading the disease is decreased. However, it can be expected that part of this area will be infested with populations which in their gene pool possess genes for aggressiveness towards these varieties, be it in low unobservable frequencies. In the course of time the eelworm populations developed there will multiply strongly in the roots of this andigena material so that these plots too will be outwitted by this kind of control. In the Netherlands, where potato growing is legally restricted to once in three years in any arable plot, resistant varieties will be useful in these areas for an estimated period of 15-30 years and longer.

b). Resistant varieties bred from S. vernei and S. kurtzianum: The resistance in these species is such that varieties bred from them can be regarded in much the same way as material derived from CPC 1673. However, the area in which they can at first be grown safely is much larger and certainly covers more than 90% of the total infested areas. Eventually, however, it is again to be expected that the selective effect of the resistant varieties on the larval populations will result in part of the infested area becoming unsuitable for the continued growing of these varieties.

From the foregoing it is apparent that a number of major genes occurring in various wild and primitive potatoes are used in breeding for resistance to potato root eelworm. However, the protective effect of these genes can be nullified because the parasite can adapt itself to the environment in the roots of these resistant forms, thanks to its genetic variability.

This means that the breeders for resistance to potato sickness have to face the same problem as for instance the breeders for Phytophthora resistance who are using the R-genes of S. demissum. Breeders for Heterodera-resistance have the advantage that the new races of the potato root eelworm only spread slowly. They are at a disadvantage in so far as the potato-root eelworm reproduces sexually, so that its genetic variability is used to best advantage.

A question which arises is how many different resistance genes are likely to be found within the total of all the potato species. The large number of resistant wild forms which has become known in recent years need not be a reason for optimism. It is very likely that the genes governing resistance in the various species may include several indistinguishable ones. Therefore, not all of the known, resistant wild species may be useful for broadening the basis of breeding for resistance.

It is possible that some other form of resistance may be found to have a different effect on the parasite than the resistance of subsp. andigena, S. kurtzianum, S. vernei and S. famatinae. The economic significance of such a new kind of resistance would be very great. It is, therefore, desirable to keep searching for new resistant forms of the potato and to subject new accessions to intensive investigations into the nature of the resistance.

Another possibility of controlling potato sickness lies in the development of tolerant varieties. This must be considered feasible (OOSTENBRINK, 1950). Such tolerant varieties would show only a small reduction in yield when grown in infested soil. The disadvantage of this sort of material is that through its cultivation a high degree of infestation is maintained, which could lead to greater spread of the parasite. For countries where potato sickness is already widespread and where no legal restrictions supplement the control of potato root eelworm by resistant varieties, the breeding for tolerance seems to offer better prospects than using major genes for resistance.

Selected Reading

Hooker, A.L. 1967. The genetics and expression of resistance in plants to rusts of the genus Puccinia. Ann. Rev. Phytopathology 5: 163-182.

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