Neth. J. Pl. Path. 82 (1976) 133–145

Genetics of host-parasite relationships and uniform and differential resistance

A. H. EENINK

Institute for Horticultural Plant Breeding (IVT), Wageningen

Accepted 23 January 1976

Abstract

The value of resistance depends on its level and stability. The stability is determined by the genetics of host-parasite relationships and not by the genetics of resistance. Quantity as well as quality of resistance and pathogenicity genes may be important. Monogenic and polygenic resistances can be stable or unstable. Research on the backgrounds of stable forms of resistance, e.g. concerning the R-gene and the possibly corresponding gene complex, would be of importance for detection or development of such resistances. The terms uniform and differential resistance should be used with discrimination.

Introduction

The value of resistance in cultivated crops depends on its stability and level. Until the mid-1960's plant breeders as well as plant pathologists were mainly concerned with the absolute differential (vertical) resistance. Thereafter, interest in the partial uniform (horizontal) resistance mounted steadily (Van der Plank, 1963, 1968; Robinson, 1969) because differential resistance was rather frequently accompanied by a rapid breakthrough by 'new' races or biotypes of the parasite. Uniform resistance generally was and is expected to be more stable because of its often polygenic inheritance, while the mono- or oligogenic inheritance of differential resistance is held responsible for the instability of this type of resistance. The generalization, frequently made both by phytopathologists and plant breeders (Anonymous, 1975; Person, 1966; Van der Plank, 1963, 1968, 1975; Zadoks, 1972a, 1972b), that monogenic resistance is unstable and polygenic resistance is stable has led to an underestimation of the contribution by mono/oligogenic resistance to crop protection.

The present paper reviews the literature on resistance and aims at stimulating the discussion on uniform and differential resistance, to arrive at a more differentiated opinion regarding these two forms of resistance, especially with respect to their stability.

Co-evolution of host and parasite

Between host and parasite there is an intimate relationship (Hogenboom, 1975; Person and Ebba, 1975), regulated by matching gene systems. These may first have been simple but became more or less complicated through co-evolution. A change in resistance of the host, for example, may lead to an adaptive change in pathogenicity

of the parasite and to closer interrelationship. Conversely, changes in the parasite may induce changes in the host.

This mutual influence is likely to be frequent in regions where populations of both host and parasite occur (Braverman and Leppik, 1972). Such interdependent changes result from natural selection in populations that are heterogeneous for genotypes of resistance or pathogenicity. The mutual influence particularly relates to the gene potential for resistance and pathogenicity. For these characters only one or a few loci may be available in host and parasite, so that multiple allelism will occur. The frequently complicated interactions between host and parasite suggest, however, that large(r) numbers of loci are involved in resistance and pathogenicity.

Genetics of host-parasite relationships and stability of resistance

Several resistance mechanisms are operative in plants. Some act pre-infectionally (Martin, 1964; Walker and Stahmann, 1955) others post-infectionally (Ingham, 1973; Kaars Sijpesteijn, 1969). The genetics of such resistances has been investigated in many crops (Ausemus, 1943; Dickson, 1956; Hansen, 1934; Hare, 1965; Holmes, 1954; Hooker and Saxena, 1971; Nelson, 1973; Williams, 1975) and so has the genetics of the pathogenicity (Hamid and Aragaki, 1975; Person and Ebba, 1975; Yoder and Gracen, 1975).

The genetics of the relationship between host and parasite or interorganism genetics (Loegering, 1971) has been analysed more or less extensively in over 20 host-parasite combinations (Day, 1974; Person and Sidhu, 1971). Most of these concern the simplest form of interorganism genetics, namely the gene-for-gene relationship, where a new resistance gene may be readily overcome by a new pathogenicity gene. This may explain part of the pessimism which prevails regarding the stability of new monogenic resistance that may have to be incorporated yet in cultivated types.

Expectations regarding stability of resistance are often only based on the number of resistance genes in the host and not on the often numerous pathogenicity genes that might be present in the parasite to overcome resistance.

The stability of resistance, besides being determined by such factors as growing conditions and genetical heterogeneity of the host population, largely depends on the genetic potential of the parasite (the number of loci available for parasitism or pathogenicity) and on the rapidity with which the parasite can adapt itself to a new situation, a new resistance. The stability of resistance increases if more or more-complicated genes (Prakken, 1974; Saxena and Hooker, 1974) are required in the parasite to overcome the resistance.

Table 1 shows my hypothesis on a possible relationship between the quantity of resistance and pathogenicity genes and the stability of resistance and also the effect of the quality of these genes on the stability. For it is conceivable that a qualitatively superior monogenic/oligogenic resistance can only be overcome by a qualitatively superior pathogenicity gene which seldom or never occurs in the pathogen. This may be due for instance to a negative influence of this gene on the fitness of the pathogen. By qualitatively superior or complicated genes, genes are meant with, e.g., qualitatively or quantitatively deviating loci (e.g. resulting from a particular sequence of nucleotides or absolute linkage between several genes).

Table 1. The relation between genetics of resistance and pathogenicity and the stability of resistance.

Genetics of resistance	Genetics of pathogenicity	Expected stability of resistance	
		(a) simple pathogenicity gene(s) can overcome resistance	only (a) complex pathogenicity gene(s) can overcome resistance
monogenic/oligogenic	monogenic/oligogenic	unstable	stable
monogenic/oligogenic	polygenic	stable	stable
polygenic	monogenic/oligogenic	unstable	stable
polygenic	polygenic	stable	stable

Tabel 1. De relatie tussen de genetica van resistentie en pathogeniteit en de stabiliteit van resistentie.

The table suggests that both monogenic and polygenic resistances can be stable or unstable and that (un)stability is determined by the genetics of the relationship.

Monogenic resistance. A number of gene-for-gene relationships encountered in many plant-fungus relationships (Day, 1974; Person and Sidhu, 1971) and also, e.g. in the plant-insect relation of wheat and Hessian fly (Hatchett and Gallun, 1967), demonstrate how monogenic unstable resistance has been overcome by monogenically inherited pathogenicity.

Table 2, however, shows several host-parasite relationships with resistance controlled by one or a few genes and appearing to be very stable, perhaps because in the

Table 2. Host-parasite relationships with stable, simply inherited resistance.

Host-parasite relationship	Genetics of resistance	Duration of resistance	Author(s)
Bean-*Colletotrichum lindemuthianum*	1 gene	± 15 years	Mastenbroek, 1960
Cabbage-*Fusarium oxysporum*	1 gene	> 60 years	Jones and Gilman, 1915
Cucumber-*Cladosporium cucumérinum*	1 gene	> 12 years	Andeweg, 1953
Cucumber-*Corynespora melonis*	1 gene	> 15 years	Van Es, 1958
Cucumber-cucumber mosaic virus	3 genes	> 17 years	Tjallingii, 1952
Lettuce-lettuce mosaic virus	1 gene	> 10 years	Ryder, 1970
Maize-*Helminthosporium carbonum*	1 gene	> 30 years	Ullstrup and Brunson, 1947
Pea-*Fusarium oxysporum*	1 gene	> 15 years	Yen and Cruickshank, 1957
Pea-pea early-browning virus	1 gene	> 10 years	Hubbeling and Kooistra, 1963
Oats-*Helminthosporium victoriae*	1 gene	> 25 years	Murphy and Meehan, 1946
Potato-potato virus X, Y and A	1 gene	> 15 years	Ross, 1958
Spinach-*Peronospora spinaciae*	2 genes	± 25 years	Eenink, 1976
Spinach-Cucumber mosaic virus	1 gene	> 20 years	Pound and Cheo, 1952
Wheat-*Puccinia graminis*	1 gene	± 20 years	Caldwell, 1968
Wheat-*Ustilago tritici*	2 genes	> 40 years	Caldwell, 1968

Tabel 2. Waard-parasiet relaties met een stabiele, eenvoudig overervende resistentie.

parasite highly complicated pathogenicity genes may be required to cause a congruous reaction (Hogenboom, 1975). Congruity and incongruity are new terms for compatibility and incompatibility, respectively, as far as interspecific (inter alia host-parasite) relationships are concerned.

Part of the monogenic/oligogenic resistances in Table 2 may be stable because the resistance can only be overcome through polygenic pathogenicity. An example of such a plant-parasite relationship may be barley where cleistogamous flowering results in resistance to *Ustilago nuda*. Cleistogamy is often very simply inherited and occurs in various plant families (Dillé and Knowles, 1975). To overcome this character, the genotype for pathogenicity would have to change drastically; an entirely new penetration mechanism would have to be acquired.

Similar host-parasite relationships in which resistance is not very specific are rather numerous (see also Horber, 1972). Some examples are given in Table 3. The genetics of the characters causing this resistance is known for a number of these relationships, their inheritance is controlled by only one gene or a few genes.

Polygenic resistance. A polygenic resistance which can be broken by the parasite more or less rapidly or simply with the aid of only one or a few pathogenicity genes is conceivable if all resistance genes (minor genes) jointly influence one character or process in a quantitative sense. This character can be either of a morphological or a biochemical nature. For example, it was found in Mexico that the level of uniform resistance in potato against *Phytophthora infestans* decreased after a certain period, possibly resulting from a more or less gradual adaptation of the parasite (Riley, 1973). Perhaps this uniform resistance is similar to the type described above. Thus, the frequent supposition that polygenic resistance is, by definition, practically stable may not always be correct.

If the many genes causing resistance, influence different processes or characters, and consequently, have a qualitatively and possibly also a quantitatively different expression, then polygenic resistance is stable.

Table 3. Host-parasite relationships in which nonspecific resistance occurs.

Host-parasite relationship	Character responsible for resistance	Author(s)
Apple-*Venturia inaequalis*	thick cuticle	Maeda, 1970
Cotton-insects/bollrot fungi	frego bracts and absence of nectaries	Cauquil and Ranney, 1967 Jones et al., 1968/69
Cucumber-*Tetranychus urticae*	cucurbitacins	Da Costa and Jones, 1971
Pea-*Ascochyta pisi*	thick cuticle	Hagedorn, 1973 Lyall and Wallen, 1958
Potato-aphids/*Trialeurodes vaporariorum*	glandular hairs	Gibson, 1971 Hogenboom et al., 1974
Wheat-*Oulema melanopus*	leaf surface pubescence	Webster et al., 1975

Tabel 3. Waard-parasiet relaties waarbij niet-specifieke resistentie aanwezig is.

Changes in host and parasite in congruous and incongruous relationships in connection with the genotype of resistance

In host and parasite, both in congruous and incongruous relationships, more or less marked morphological, physiological and/or biochemical changes may occur as a consequence of the interaction between the two partners. The degree and nature of these changes are associated with the type of resistance: pre-infectional (mechanical barriers or biochemical barriers such as fungitoxins) or post-infectional (e.g. mechanical barriers, enzyme inhibitors, detoxification of phytotoxins, hypersensitivity reaction to toxins from fungi). In a number of cases different, perhaps complementary resistance systems may be active in the host.

In the parasite differences occur in a qualitative and/or quantitative sense, e.g. in the production of enzymes (Anderson and Albersheim, 1972; Hall and Wood, 1973; Pitt and Galpin, 1973; Stahmann and Demorest, 1973), specific or non-specific toxins (Bhullar et al., 1975; Comstock and Martinson, 1975; Heitefuss, 1970; Pringle and Scheffer, 1964; Strobel, 1973, 1975), in growth regulators (Lang, 1970; Skoog and Armstrong, 1970; Zalewski and Sequeira, 1974) and/or in proteins (Valent and Albersheim, 1974). Great changes often occur at the host-parasite interfaces (Slusher et al., 1974).

The host may respond after infection with an increased permeability of cell walls or membranes (Damann et al., 1974; Van Dijkman, 1972), possibly as a consequence of an increased ethylene production (Hislop, 1973; Lund, 1973), and with an increased respiration (Bushnell, 1970; Comstock and Martinson, 1975; Fuchs, 1971). Callose formation sometimes occurs (Shimomura and Dijkstra, 1975). Qualitative or quantitative changes may occur in enzyme activity (Farkas et al., 1963; Pitt and Galpin, 1973) and an alteration in synthesis of nucleic acids and proteins can take place (Heitefuss, 1966, 1970). Sometimes proteinase inhibitors are formed (Peng and Black, 1974), and changes in the formation of growth regulators are found (Pilet, 1960). In some cases changes in the production of phytoalexins may be essential for the incitement of a congruous or incongruous reaction (Bailey, 1973; Cruickshank and Perrin, 1967; Van den Heuvel and Glazenier, 1975; Ingham, 1972).

So, in the various host-parasite relationships apparantly complicated interactions frequently occur. The number and nature of the genes involved are practically unknown. To explain e.g. the stability of the types of resistance mentioned in Table 2, investigations would have to be made into the relationships between genes for resistance and pathogenicity and the morphological, physiological and/or biochemical changes in host and parasite which are responsible for these stable types of resistance.

A model of gene activity in connection with certain congruous or incongruous host-parasite interactions was developed by Britton and Davidson (1969). In this model groups of genes (sensor genes, integrator genes, receptor genes and producer genes) control such interactions through a range of processes. Resistance genes are said to correspond with sensor genes and consequently are responsible for putting or not putting in operation a series of genes which in turn, can initiate various processes. A model of gene regulation slightly comparable to the model mentioned above was developed earlier by Jacob and Monod (1961) for bacteria. A resistance gene may then be comparable to an operator gene of this model. Thus, with so-called monogenetical resistance only the top of the genetical iceberg may be visible. The quality/

complexity of the resistance gene (sensor gene or operator gene) could in such a case be decisive for congruity or incongruity of a relationship and for stability of the resistance.

The quality of a resistance gene could, for example, be determined by a very close linkage between this gene and other genes, e.g. of the above-mentioned series (integrator genes – producer genes). Owing to such linkage certain interactions could proceed unfavourably for the pathogen, so that the setting in action of a mechanism or of a series of processes resulting in susceptibility does not take place, or on the contrary, that a mechanism leading to resistance is set going.

Such a qualitatively high-grade R-gene can give both stable pre-infectional and stable post-infectional resistance. For instance, it could be imagined that such a gene is responsible for the production of a fungitoxin that cannot be broken down by 'old' or 'new' races. A stable post-infectional resistance is conceivable if the R-gene is responsible for e.g. a very wide spectrum of action, (a) against phytotoxins (breakdown of phytotoxins or no binding with cell membranes as a consequence of deviating binding proteins (Strobel, 1975)) or (b) of inhibitors produced. In the case of a hypersensitivity resistance, the presence of the stable R-gene (sensor-gene) may be responsible for extreme sensitivity; for example to Al-Ax avirulence genes and maybe also al-ax virulence genes. Such qualitatively superior resistance genes might be present in primary or secondary gene centres where certain equilibria between host and parasite are likely to exist (Braverman and Leppik, 1972; Browning, 1974).

For the detection or development of stable resistance it would be very important to have more information on the relation between stable resistance genes and processes in the host and possibly in the parasite, caused directly or indirectly by these genes, as a result of the interaction between the two partners. Biochemical tests (see e.g. Van Dijkman, 1972 and Strobel, 1975) could then demonstrate whether a resistance will be stable or not. Such biochemical investigations would have to be carried out together with genetical research. Biochemical changes in both host and parasite would have to be examined in host-parasite relationships as mentioned in Table 4.

Qualitatively or quantitatively different biochemical processes resulting from interaction in congruous or incongruous relationships depending on the presence of a stable or an unstable resistance, might yield information on the backgrounds of the (un)stability of resistance. If differences in processes could be revealed, they may be due to the R-gene (the switch) as well as to any corresponding gene complex (integrator genes-producer genes). In my opinion, the action of modifier genes, encountered in various host-parasite relationships involving monogenic resistance (Innes, 1964; Rouselle, 1974), might correspond with that of genes from the gene complex.

Table 4. Host-parasite relationships were (biochemical) processes resulting from interactions should be investigated.

Host(s)	Parasite(s)	Relationships to be investigated
one genotype for resistance	different races	congruent and incongruent
different genotypes for resistance	one race	congruent and incongruent

Tabel 4. Waard-parasiet relaties waarbij biochemische processen, die optreden als gevolg van interacties, zouden moeten worden onderzocht.

Investigation of this (possible) gene complex is essential because it may directly or indirectly play an important part in e.g. level and stability of the resistance. Also the influence on resistance of the cytoplasm (Comstock and Martinson, 1974, 1975; Kruger et al., 1974; Watrud et al., 1975), its organelles, or possibly the interaction of cytoplasm and chromosomes should be investigated.

This type of research could be carried out by transferring R-genes to different genetical or cytoplasmical environments and subsequently determining the effect of this transfer on the level of resistance or on changes in host-parasite interactions. In this connection, monogenic partial resistances may also be interesting and so may be changes in host-parasite interactions resulting from induced gene or point mutations in the gene complex. Useful information could also be derived from intercrossing genetically different host plants with the same R-gene. The crossing of a large number of genetically different plants without resistance to known races or biotypes of the parasite might give an idea of the genes that are active in the gene complex. For instance, when a number of host plants is susceptible owing to lack of essential genes in the gene complex despite the possibility of the R-gene being present, crossing could yield plants that are resistant because in such plants the lacking genes are supplied by the parents.

Research of the type described above has been very limited so far. In some cases use was made of nearly isogenic lines to facilitate the study of the action of various resistance genes (Gnanamanickam and Patil, 1974; Green et al., 1974; Hammerschlag and Mace, 1975). These investigations showed that qualitative and/or quantitative differences in processes occurred as a result of certain interactions between host and parasite, as with respect to the production of toxins depending on the genotype for resistance.

Number and expression of genes for uniform and differential resistance

Genes for uniform or differential resistance. Differential resistance is controlled by one or a few genes, whereas uniform resistance is generally supposed to be inherited in polygenic fashion. From the foregoing it appears (see also Table 2) that with a great many host-parasite relationships stable (uniform) resistance to a parasite, that may consist of various races or biotypes, is determined by only one or a few genes (see also Luke et al., 1975). Therefore it is not correct to link the concepts of uniform resistance, polygenic resistance and stable resistance.

In the case of the type of resistance just mentioned, stable uniform resistance is not governed by 'minor genes' but by 'major genes'. The concept of minor and major gene with respect to the uniform resistance is very debatable for that matter. For instance, genes which give resistance in wheat to *Puccinia graminis* var. *tritici* and are localised on the homeologous chromosomes 7A, 7B and 7D, seem to act against certain races as major genes and against others as minor genes (Law and Johnson, 1967; Watson and Luig, 1965). According to Nelson et al. (1970), with maize the same genes govern both uniform and differential resistance to *Trichometasphaeria turcica*. The uniform partial resistance in tomato to *Cladosporium fulvum* may be caused by a major gene (Boukema and Garretsen, 1975). Such 'ghost genes' were also found in other host-parasite relationships (Riley, 1973).

Level of uniform and differential resistance. Also with respect to the level of uniform and differential resistance no essential differences can be shown. Uniform resistance which is governed by minor genes is partial, whereas uniform resistance which depends on major genes is mostly complete, although partial resistance also occurs (Grümmer et al., 1969).

Differential resistance is often complete although in many host-parasite relationships the resistance level can be influenced. Sometimes the level of monogenic resistance appears to be influenced by gene dosage effects (Dunn and Namm, 1970) or by modifier genes (Innes, 1964; Rouselle, 1974). Other internal conditions of the plant, including its physiological state, as in connection with age can also effect the resistance level (Bartos et al., 1969; Caldwell et al., 1957). In a considerable number of host-parasite relationships extraneous factors such as soil, temperature, air humidity or light appear to raise or lower the level of the monogenic resistance (Heitefuss, 1965; Hubbeling, 1966; Walker and Williams, 1973).

Epidemic development with uniform and differential resistance. From an epidemiologic viewpoint (e.g. infection ratio, latency period, lesion growth and infectious period; see also Zadoks, 1972) it is impossible to draw any definite borderline between uniform and differential resistance. Epidemiology only considers the consequences of the expression of genes which are active in congruous and incongruous host-parasite relationships. Because there are no essential differences with respect to the genotypes of uniform and differential resistance it is not likely that essential phenotypical differences from host-parasite interaction will occur.

In host plant populations with uniform partial resistance, an epidemic develops more slowly than in a completely susceptible host plant population because a process or a series of processes develops unfavourably for the parasite. This type of resistance, however, shows only a gradual difference from completely or almost completely differential resistance in which one or more processes of the above series are completely arrested. With a number of differential resistances, penetration and a certain development of the parasite in the host do occur. This development can continue up to the formation of secondary hyphae and may even lead to sporulation on a limited scale as was found in various host-parasite relationships (Conway et al., 1974; Ellingboe, 1972; Maeda, 1970; Riggle, 1974; Turner and Hart, 1975; Watkins and Statler, 1974).

Consequently, neither in genotypes nor in phenotypes can essential differences be shown to exist between uniform and differential resistance. I believe that at least in a number of cases, partial polygenic resistance may be caused by a completely or almost completely autonomous gene complex in which the R-gene rarely, if ever, acts as a 'switch'. If the R-gene(s) belonging to such a complex should function, then perhaps complete resistance occurs.

Conclusions

The stability of resistance depends on the genetics of the host-parasite relationships and not on the genetics of the resistance. Therefore it is not correct to suppose that polygenic resistance, by definition, is stable and monogenic resistance unstable.

Phytopathologists and plant breeders in close cooperation should investigate the causes of stability of certain simply inherited resistances. Knowledge of this matter would greatly help to detect or develop stable resistances.

With respect to both the genetics and the stability and level of resistance there are no essential differences between uniform and differential resistance. Those two terms then. should be used with discrimination.

Acknowledgments

I am indebted to Dr N. G. Hogenboom for the valuable discussions during the preparation of this paper. Other colleagues of the IVT are also thanked for critically reading the manuscript.

Samenvatting

De genetica van waard-parasiet relaties en uniforme en differentiële resistentie

Tussen waard en parasiet bestaat een intieme relatie. Deze berust op corresponderende gensystemen, welke in veel gevallen gen-om-gen relaties bleken te zijn.

De stabiliteit van resistentie wordt bepaald door de genetica van de waard-parasiet relatie en niet alleen door de genetica van de resistentie (Tabel 1). Deze stabiliteit is ondermeer afhankelijk van het genetisch vermogen van de parasiet om zich aan te passen aan een nieuwe resistentie. De stabiliteit neemt toe naarmate meer en/of meergecompliceerde genen in de parasiet aanwezig moeten zijn om de resistentie te doorbreken. Niet slechts polygene, maar ook monogene/oligogene resistenties kunnen stabiel zijn (Tabel 2 en 3).

Meer informatie is gewenst over de achtergrond van stabiele resistenties. Deze informatie zou kunnen worden verkregen door samenwerking van biochemici-fytopathologen en plantenveredelaars. Veranderingen die optreden als gevolg van bepaalde interacties in waard-parasiet relaties, waarbij wordt gewerkt met verschillende resistenties en fysio's, zouden moeten worden onderzocht (Tabel 4). Deze informatie zou misschien kunnen worden gebruikt voor de opsporing of ontwikkeling van stabiele resistenties.

Zowel ten aanzien van genotype en niveau van resistentie als epidemiologisch, bestaan er geen principiële verschillen tussen uniforme en differentiële resistentie.

References

Anderson, A. J. & Albersheim, P., 1972. Host-pathogen interactions. V. Physiol. Pl. Path. 2: 339–346.

Andeweg, J. M., 1953. Vruchtvuurresistentie bij platglaskomkommers. Zaadbelangen 7: 252–253.

Anonymous, 1975. The proposed FAO international programme on horizontal resistance to crop pests and diseases. AGPP: MISC/19.

Ausemus, E. R., 1943. Breeding for disease resistance in wheat, oats, barley and flax. Bot. Rev. 9: 207–260.

Bailey, J. A., 1973. Phaseollin accumulation in *Phaseolus vulgaris* following infection by fungi, bacteria and a virus. pages 337–350. In: R. J. W. Byrde & C. V. Cutting (Eds), Fungal pathogenicity and the plants response. Acad. Press, London, 499 pp.

Bartos, P., Dyck, P. L. & Samborski, D. J., 1969. Adult plant leaf rust resistance in Thatcher and Marquis wheat: a genetic analyses of the host-parasite situation. Can. J. Bot. 47: 267–269.
Bhullar, B. S., Daly, J. M. & Rhefeld, D. W., 1975. Inhibition of dark CO_2 fixation and photosynthesis in leaf discs of corn susceptible to the host-specific toxin produced by *Helminthosporium maydis* race T. Pl. Physiol. 56: 1–7.
Boukema, I. W. & Garretsen, F., 1975. Uniform resistance to *Cladosporium fulvum* Cooke in tomato (*Lycopersicon esculentum* Mill.) 2. Euphytica 24: 105–116.
Braverman, S. W. & Leppik, E. E., 1972. Origins of cultivated plants and the search for disease resistance. N.Y. Food Life Sci. 5: 15–18.
Britten, R. J. & Davidson, E. H., 1969. Gene regulation for higher cells: a theory. Science N.Y. 165: 349–357.
Browning, J. A., 1974. Relevance of knowledge about natural ecosystems to development of pest management programs for agro-ecosystems. Proc. Am. phytopath. Soc. 1: 191–199.
Bushnell, W. R., 1970. Patterns in the growth, oxygen uptake and nitrogen content of single colonies of wheat stem rust on wheat leaves. Phytopathology 60: 92–99.
Caldwell, R. M., 1968. Breeding for general and/or specific plant disease resistance. 3rd Int. Wheat Genet. Symp. 263–275.
Caldwell, R. M., Schafer, J. F., Compton, L. E. & Patterson, F. L., 1957. A mature plant type of wheat leaf rust resistance of composite origin. Phytopathology 47: 691–692.
Cauquil, J. & Ranney, C. D., 1967. Internal infection of green cotton bolls and the possibility of genetic selection. Tech. Bull. agric. Exp. Stn Miss. 53: 24.
Comstock, J. C. & Martinson, C. A., 1974. Respiratory rates of Texas male-sterile and normal cytoplasm corn leaves infected with *Helminthosporium maydis* race O and race T. Proc. Am. phytopath. Soc. 1: 124.
Comstock, J. C. & Martinson, C. A., 1975. Involvement of *Helminthosporium maydis* race T toxin during colonization of maize leaves. Phytopathology 65: 616–619.
Conway, W. S., Mac Hardy, W. E. & Kornahrens, H., 1974. Colonization and distribution of *Fusarium oxysporum* f. sp. *lycopersici* race 2 in near-isogenic lines of tomato susceptible or resistant to race 1. Proc. Am. phytopath. Soc. 1: 101.
Cruickshank, I. A. M. & Perrin, D. R., 1967. Studies on phytoalexins. X. Phytopath. Z. 60: 335–342.
Dacosta, C. P. & Jones, C. M., 1971. Cucumber beetle resistance and mite susceptibility controlled by the bitter gene in *Cucumis sativus* L. Science N.Y. 172: 1145–1146.
Damann, K. E., Gardner, J. M. & Scheffer, R. P., 1974. An assay for *Helminthosporium victoriae* toxin based on induced leakage of electrolytes from oat tissue. Phytopathology 64: 652–654.
Day, P. R., 1974. Genetics of host-parasite interaction. W. H. Freeman Co. San Francisco. 238 pp.
Dickson, J. G., 1956. Diseases of field crops. MacGraw-Hill, New York, 517 pp.
Dunn, G. M. & Namm, T., 1970. Gene dosage effects on monogenic resistance to northern corn leaf blight. Crop Sci. 10: 352–354.
Dijkman, A. van, 1972. Natural resistance of tomato plants to *Cladosporium fulvum*, a biochemical study. Organisch Chemisch Instituut TNO, Utrecht, Holland. 94 pp.
Dillé, J. E. & Knowles, P. F., 1975. Histology and inheritance of the closed flower in *Carthamus tinctorius* (Compositae). Am. J. Bot. 62: 209–215.
Eenink, A. H., 1976. Linkage in *Spinacia oleracea* L. of two racespecific genes for resistance to downy mildew, *Peronospora farinosa* f. sp. *spinaciae* Byford. Euphytica 25: in press.
Ellingboe, A. H., 1972. Genetics and physiology of primary infection by *Erysiphe graminis*. Phytopathology 62: 401–406.
Es, J. van, 1958. Bladvuurresistentie bij komkommers. Zaadbelangen 12: 116–117.
Farkas, G. L., Dézsi, L., Horvath, M., Kisbán, K. & Udvardy, J., 1963/64. Common pattern of enzymatic changes in detached leaves and tissues attacked by parasites. Phytopath. Z. 49: 343–354.
Fuchs, W. H., 1971. Physiological and biochemical aspects of resistance to diseases. pages 5–16. In: Mutation breeding for disease resistance. IAEA, Vienna, 249 p.
Gibson, R. W., 1971. Glandular hairs providing resistance to aphids in certain wild potato species. Ann. appl. Biol. 68: 113–119.
Gnanamanickam, S. & Patil, S. S., 1974. Bacterial growth, toxin production and levels of ornithine carbamoyltransferase (oct) in resistant and susceptible varieties of bean inoculated with *Pseudomonas phaseolicola*. Proc. Am. phytopath. Soc. 1: 66.

Green, N. E., Hadwiger, L. A. & Graham, S. O., 1974. Phenylalanine ammonia lyase, tyrosine ammonia lyase and lignin in nearisolines of wheat inoculated with *Erysiphe graminis* f. sp. *tritici*. Proc. Am. phytopath. Soc. 1: 65.

Grümmer, G., Günther, E. & Eggert, D., 1969. Die Prüfung von Tomatensorten und ihren Hybriden auf Blatt- und Fruchtbefall mit *Phytophthora infestans*. Theor. appl. Genet. 39: 232–238.

Hagedorn, D. J., 1973. Peas. pages 326–343. In: R. R. Nelson (Ed.), Breeding plants for disease resistance. Pennsylvania St. Univ. Press, London. 499 pp.

Hall, J. A. & Wood, R. K. S., 1973. The killing of plant cells by pectolytic enzymes. pages 19–31. In: R. J. W. Byrde & C. V. Cutting (Eds), Fungal pathogenicity and the plants response, Acad. Press. London. 499 pp.

Hamid, A. H. & Aragaki, M., 1975. Inheritance of pathogenicity in *Setosphaeria turcica*. Phytopathology 65: 280–283.

Hammerschlag, F. & Mace, M. E., 1975. Antifungal activity of extracts from *Fusarium* wilt-susceptible and -resistant tomato plants. Phytopathology 65: 93–94.

Hansen, H. P., 1934. Inheritance of resistance to plant diseases caused by fungi, bacteria and vira. Yb. R. vet. agric. Coll., Copenhagen, pp. 1–74.

Hare, W. W., 1965. The inheritance of resistance of plants to nematodes. Phytopathology 55: 1162–1167.

Hatchett, J. H. & Gallun, R. L., 1967. Genetics of the ability of the Hessian fly, *Mayetiola destructor*, to survive on wheats having different genes for resistance. Ann. ent. Soc. Am. 63: 1400.

Heitefuss, R., 1965. Untersuchungen zur Physiologie des temperaturgesteuerten Verträglichkeitsgrades von Weizen und *Puccinia graminis tritici* I. Phytopath. Z. 54:379–400.

Heitefuss, R., 1966. Nucleic acid metabolism in obligate parasitism. A. Rev. Phytopath. 4: 221–244.

Heitefuss, R., 1970. Wirt-Parasit-Interaktionen. Ber. dt. bot. Ges. 83: 203–219.

Heuvel, J. van den & Glazenier, J. A., 1975. Comparative abilities of fungi pathogenic and non pathogenic to bean (*Phaseolus vulgaris*) to metabolize phaseolin. Neth. J. Pl. Path. 81: 125–137.

Hislop, E. C., Hoad, G. V. & Archer, S. A., 1973. The involvement of ethylene in plant diseases. pages 87–113. In: R. J. W. Byrde & C. V. Cutting (Eds). Fungal pathogenicity and the plants response. Acad. Press, London. 499 p.

Hogenboom, N. G., 1975. Incompatibility and incongruity: two different mechanisms for the non-functioning of intimate partner relationships. Proc. R. Soc. London. Sect. B. 188: 361–375.

Hogenboom, N. G., Ponti, O. M. B. de & Pet, G., 1974. Veredeling van tomaat (*Lycopersicon esculentum* Mill.) op resistentie tegen witte vlieg (*Trialeurodes vaporariorum* Westwood). Zaadbelangen 28: 160–163.

Holmes, F. O., 1954. Inheritance of resistance to viral diseases in plants. Adv. Virus Res. 2: 1–30.

Hooker, A. L. & Saxena, K. M. S., 1971. Genetics of disease resistance in plants. A. Rev. Genet. 5: 407–424.

Horber, E., 1972. Plant resistance to insects. Agric. Sci. Rev. 10: 1–10.

Hubbeling, N., 1966. Resistance to American vascular disease and 'near wilt' on pea. Neth. J. Pl. Path. 72: 204–211.

Hubbeling, N. & Kooistra, E., 1963. Resistance to early browning in peas. Euphytica 12: 258–260.

Ingham, J. L., 1972. Phytoalexins and other natural products as factors in plant disease resistance. Bot. Rev. 38: 343–424.

Ingham, J. L., 1973. Disease resistance in higher plants. Phytopath. Z. 78: 314–335.

Innes, N. L., 1964. Resistance conferred by new gene combinations to bacterial blight of cotton. Euphytica 13: 33–43.

Jacob, F. & Monod, J., 1961. Genetic regulatory mechanisms in the sysnthesis of proteins. J. molec. Biol. 3: 318–356.

Jones, L. R. & Gilman, J. C., 1915. The control of cabbage yellows through disease resistance. Res. Bull. agric. Exp. Stn Univ. Wis. 38, pp. 70.

Jones, J. E., Andries, J. A., Sloane, L. W. & Phillips, S. A., 1968–'69. Frego bract reduces cotton boll. rot. Bull. La. agric. Exp. Stn 12: 8–11.

Kaars Sijpesteijn, A., 1969. Aspects of natural disease resistance. Meded. Rijksfak. Landbouwwet. Gent. 34: 379–391.

Kruger, W. A., Josephson, L. M. & Hilty, J. W., 1974. Response of mitochondria from corn cytoplasms to the pathotoxin of *Helminthosporium maydis* race T. Phytopathology 64: 735–737.

Lang, A., 1970. Gibberellins: Structure and metabolism. Ann. Rev. Pl. Physiol. 21: 537–570.

Law, C. N. & Johnson, R., 1967. A genetic study of leaf rust resistance in wheat. Can. J. Genet. Cytol. 9: 805–822.
Loegering, W. Q., 1971. Application of interorganism genetics to mutation breeding for disease resistance. pages 25–30. In: Mutation breeding for disease resistance. IAEA, Vienna: 249 pp.
Luke, H. H., Barnett, R. D. & Pfahler, P. L., 1975. Inheritance of horizontal resistance to crown rust in oats. Phytopathology 65: 631–632.
Lund, B. M., 1973. The effect of certain bacteria on ethylene production by plant tissue. pages 69–84. In: R. J. W. Byrde & C. V. Cutting (Eds), Fungal pathogenicity and the plant's response. Acad. Press, London. 499 p.
Lyall, L. H. & Wallen, V. R., 1958. The inheritance of resistance to *Ascochyta pisi* Lib. in peas. Can. J. Pl. Sci. 38: 215–218.
Maeda, K. M., 1970. An ultrastructural study of *Venturia inaequalis* (Cke) Wint. infection of *Malus* hosts. Masters Thesis. Purdue Univ., Lafayette, Ind.
Martin, J. T., 1964. Role of cuticle in the defence against plant disease. A. Rev. Phytopath. 2: 81–100.
Mastenbroek, C., 1960. A breeding programme for resistance to anthracnose in dry shell haricot beans, based on a new gene. Euphytica 9: 177–184.
Murphy, H. C. & Meehan, F., 1946. Reaction of oat varieties to a new species of *Helminthosporium*. Phytopathology 36: 407.
Nelson, R. R., 1973. Breeding plants for disease resistance. The Pennsylvania St. Univ. Press. 401 pp.
Nelson, R. R., MacKenzie, D. R. & Scheifele, G. L., 1970. Interaction of genes for pathogenicity and virulence in *Trichometasphaeria turcica* with different numbers of genes for vertical resistance in *Zea mays*. Phytopathology 60: 1250–1254.
Peng, J. H. & Black, L. L., 1974. Increase in activity of proteinase inhibitors in tomato plants inoculated with an incompatible race of *Phytophthora infestans*. Proc. Am. phytopath. Soc. 1: 63.
Person, C., 1966. Genetic polymorphism in parasitic systems. Nature Lond. 212: 266–267.
Person, C. & Sidhu, G., 1971. Genetics of host-parasite interrelationships. pages 31–38. In: Mutation breeding for disease resistance. IAEA, Vienna: 249 pp.
Person, C. & Ebba, T., 1975. Genetics of fungal pathogens. Genetics 79: 397–408.
Pilet, P. E., 1960. Auxin content and auxin catabolism of the stems of *Euphorbia cyparissias* L. infected by *Uromyces pisi* (Pers.). Phytopath. Z. 40: 75–90.
Pitt, D. & Galpin, M., 1973. Role of lyosomal enzymes in pathogenicity. pages 449–465. In: R. J. W. Byrde & C. V. Cutting, (Eds) Fungal pathogenicity and the plants response. Acad. Press, London. 499 pp.
Plank, J. E. van der, 1963. Plant diseases: epidemics and control. Acad. Press, New York. 349 pp.
Plank, J. E. van der, 1968. Disease resistance in plants. Acad. Press, New York. 206 pp.
Plank, J. E. van der, 1975. Principles of plant infection. Acad. Press, New York. 216 pp.
Pound, G. S. & Cheo, P. C., 1952. Studies on resistance to cucumber virus I in spinach. Phytopathology 42: 301–306.
Prakken, R., 1974. Inheritance of colours in *Phaseolus vulgaris* L. IV. Recombination within the 'complex locus C'. Meded. Landb.hogesch., Wageningen, 74-24, pp 36.
Pringle, R. B. & Scheffer, R. P., 1964. Hostspecific plant toxins. A. Rev. Phytopath. 2: 133–156.
Riggle, J. H., 1974. Development of *Peronospora manshurica* in leaves of susceptible, intermediate, resistant, and non-host plants. Proc. phytopath. Soc. 1: 129.
Riley, R., 1973. Genetic changes in hosts and the significance of disease. Ann. appl. Biol. 75: 128–132.
Robinson, R. A., 1969. Disease resistance terminology. Rev. appl. Mycol. 48: 593–606.
Ross, H., 1958. Virusresistenzzüchtung an der Kartoffel. Europ. Potato J. 1: 81–99.
Rouselle, G. L., 1974. Variation of the hypersensitive reaction of the Vf gene for apple scab resistance. Rutgers Univ. State Univ. New Jersey. In: Doctoral Research on Agriculture 1975, 12.
Ryder, E. J., 1970. Inheritance of resistance to common lettuce mosaic. J. Am. Soc. hort. Sci. 95: 378–379.
Saxena, K. M. S. & Hooker, A. L., 1974. A study on the structure of gene RP3 for rust resistance in *Zea mays*. Can. J. Genet. Cytol. 16: 857–860.
Shimomura, T. & Dijkstra, J., 1975. The occurrence of callose during the process of local lesion formation. Neth. J. Pl. Path. 81: 107–121.
Skoog, F. & Armstrong, D. J., 1970. Cytokinins. A. Rev. Pl. Physiol. 21: 359–384.
Slusher, R. L., Haas, D. L., Carothers, Z. B. & Sinclair, J. B., 1974. Ultrastructure at the host-

parasite interface of *Phytophthora megasperma* var. *sojae* in soybean rootlets. Phytopathology 64: 834–840.
Stahmann, M. A. & Demorest, D. M., 1973. Changes in enzymes of host and pathogen with special reference to peroxidase interaction. pages 405–420. In: R. J. W. Byrde & C. V. Cutting (Eds), Fungal pathogenicity and the plants response. Acad. Press London. 499 pp.
Strobel, G. A., 1973. Biochemical basis of the resistance of sugarcane to eyespot disease. Proc. Natn. Acad. Sci. USA 70: 1693–1696.
Strobel, G. A., 1975. A mechanism of disease resistance in plants. Scient. Am. 232: 81–89.
Tjallingii, F., 1952. Onderzoekingen over de mozaiekziekte van de augurk *(Cucumis sativus* L.). Meded. Inst. plziektenk. Onderz. No. 47, 117 pp.
Turner, M. F. & Hart, K., 1975. Field spore production of *Helminthosporium turcicum* on *Zea mays* with and without monogenic resistance. Phytopathology 65: 735–736.
Ullstrup, A. J. & Brunson, A. M., 1947' Linkage relationships of a gene in corn determining susceptibility to a *Helminthosporium* leaf sport. J. Am. Soc. Agron. 39: 606–609.
Valent, B. & Albersheim, P., 1974. A protein from the fungal pathogen *Colletotrichum lindemuthianum* inhibits an enzyme of its host capable of attacking the pathogen. Proc. phytopath. Soc. 1 : 52.
Walker, J. C. & Stahmannn, M. A., 1955. Chemical nature of disease resistance in plants. A. Rev. Pl. Physiol. 6: 351–366.
Walker, J. C. & Williams, P. H., 1973. Cucurbits. pages 307–325. In: R. R. Nelson (Ed.) Breeding plants for disease resistance. Pennsylvania St. Univ. Press, London 401 pp.
Watkins, J. E. & Statler, G. D., 1974. Histological studies of the post-penetration phase of infection of resistant and susceptible wheat by *Puccinia recondita tritici*. Proc. Am. phytopath. Soc. 1: 131.
Watrud, L. S., Baldwin, J. K., Miller, R. J. & Koeppe, D. E., 1975. Induction of a sensitive response to *Helminthosporium maydis* race T toxin in resistant mitochondria of corn (*Zea mays* L.) by removal of the outer mitochondrial membrane. Pl. Physiol. 46 :216–221.
Watson, I. A. & Luig, N. H., 1966. Sr15 – a new gene for use in the classification of *Puccinia graminis* var. *tritici*. Euphytica 15: 239–248.
Webster, J. A., Smith, D. H., Rathe, E. & Cress, C. E., 1975. Resistance to cereal leaf beetle in wheat: density and length of leaf-surface pubescense in four wheat lines. Crop Sci. 15: 199–202.
Williams, P. H., 1975. Genetics of resistance in plants. Genetics 79: 409–419.
Yen, D. E. & Cruickshank, I. A. M., 1957. The breeding of peas resistant to *Fusarium* wilt. N.Z. Jl. Sci. Technol. 38: 702–705.
Yoder, O. C. & Gracen, V. E., 1975. Segregation of pathogenicity types and host-specific toxin production in progenies of crosses between races T and O of *Helminthosporium maydis (Cochliobolus heterostrophus)*. Phytopathology 65: 273–276.
Zadoks, J. C., 1972a. Reflections on disease resistance in annual crops. Proc. of a NATO-IUFRO Advanced Study Institute. Misc. Publ. U.S. Dep. Agric., Forest Serv., 1221: 43–63.
Zadoks, J. C., 1972b. Modern concepts of disease resistance in cereals. Pages 89–98: In: F. G. H. Lupton et al. (Eds). The way ahead in plant breeding. Proc. 6th Eucarpia Congr., Cambridge, 1971.
Zalewski, J. & Sequeira, L., 1974. A genetic approach to determine the role of a bacterial growth inhibitor from potato tissues in resistance to bacterial wilt. Proc. Am. phytopath. Soc. 1:74.

Address

Instituut voor de Veredeling van Tuinbouwgewassen (IVT), P.O. Box 16, Wageningen, the Netherlands.