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Durable resistance to plant diseases

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RESUMEN

Resistencia, en el contexto de resistencia a enfermedades en las plantas, es la habilidad del hospedero para detener el crecimiento del patógeno. En otras palabras, resistencia es el mecanismo genético mediante el cual la planta retarda o suprime la invasión de sus tejidos por parte del patógeno potencial. Es complementaria a virulencia y opuesta a susceptibilidad, que es la suma total de cualidades que hacen de una planta servir de hospedero a un patógeno. Susceptibilidad y resistencia son inversamente proporcionales. Desde el descubrimiento de la heredabilidad de la resistencia a la roya en trigo por Farrer en 1898 y de que la resistencia a la roya amarilla obedece a las leyes de Mendel por Biffen en 1905, se inicio una búsqueda extensa por la obtención de resistencia durable de las plantas a los patógenos. Las expectativas iniciales de durabilidad basadas en la presunción de evolución lenta del patógeno en su proceso por sobreponerse a la introducción de genes de resistencia en la planta fueron, sin embargo, dramáticamente eliminadas por la rápida adaptación de nuevas variantes patogénicas. Años mas tarde, el concepto de gene-por-gene en 1944 fue firmemente establecido por Oort en el sistemas Ustilago maydis - trigo y por Flor en *Melampsora lini – lino*. El descubrimiento de resistencia a los patógenos estimuló a los mejoradores de plantas a extender la búsqueda hasta el punto que mejoramiento por resistencia a los patógenos se convirtió en una de las medidas más exitosas en el proceso por controlar las enfermedades de las plantas. La práctica de obtener resistencia mediante la introducción de genes mayores (o cualitativos) en las plantas se ha constituido en la mas popular herramienta de los programas de mejoramiento por su especificidad, relativa facilidad de reconocimiento y manipulación. Este tipo de resistencia es durable hasta tanto un nuevo strain del patógeno para el cual el gene incorporado no confiere resistencia se establece, en cuyo caso un nuevo gene es introducido, siempre y cuando este disponible. Repitiendo el proceso de tiempo en tiempo, nuevas variedades con genes diferentes reemplazan las que han sucumbido por susceptibles. Debido a las limitaciones encontradas, nuevas estrategias para incrementar la durabilidad de la resistencia han sido propuestas. Pero como en términos de durabilidad el tipo de interacciones entre hospedero y patógeno son tan variadas, probablemente ningún modelo servirá para el establecimiento de una estrategia única. Resistencia durable es entendida como aquella resistencia que permanece efectiva luego de varios años de uso en extensas áreas. La revisión que aquí se presenta pretende sintetizar los descubrimientos alcanzados para ayudar a visualizar la magnitud de las interacciones hospedero-patógeno, en tanto se adelanta una discusión de los diferentes términos utilizados para relacionar el concepto de durabilidad con los procedimientos empleados para la obtención de resistencia durable a las enfermedades de plantas.

INTRODUCTION

RESISTANCE, in the context of resistance to pathogens, is the ability of the host to hinder the growth of the pathogen. In other words, resistance is the genetic mechanism for which a plant suppresses or retards invasion by a potential pathogen. It is complementary to pathogen virulence and the opposite of susceptibility, which is the total sum of qualities that makes a plant fit host for a pathogen. Susceptibility and resistance are proportionally inverse R. R.1969)» (Robinson, R. R.1969).

Since the discovery that resistance to rust in wheat is inheritable [Farrer, 1898 cited by Wit, P. J. G. M.1992)» (de Wit, P. J. G. M.1992)] and that resistance to yellow rust obeys Mendel's laws [Biffen, 1905 cited by Wit, P. J. G. M.1992)» (de Wit, P. J. G. M.1992)], the search has been on for long-lasting genetic resistance to plant pathogens. The initial expectations for durability based on slow evolution in the pathogen to overcome introduced resistance genes were, however, swept away by rapid adaptation of new pathogenic variants. Years later, the gene-for-gene concept was firmly established by Oort working with *USTILAGO MAYDIS* in wheat [Oort, 1944 cited by Wit, P. J. G. M.1992)» (de Wit, P. J. G. M.1992)] and Flor with *MELAMPSORA LINI* in flax H. H.1942)» (Flor, H. H.1942). The finding of

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resistance to plant pathogens encouraged plant breeders to expand the search.

Stated as «the host-parasite interaction in flax rust may be explained by assuming a relationship between rust reaction in the host and pathogenicity in the parasite», the gene-for-gene concept emerged as a tool to facilitate the development of resistant cultivars and to open new approaches to study on the origin of new races, mutations for susceptibility in the host, and pathogenicity in the parasite H. H.1945)» (Flor, H. H.1945). Resistance to plant pathogens is typically inherited in a simple Mendelian fashion and is generally dominant to susceptibility. Widely used in plant breeding programs, the development of resistant cultivars is one of the most successful means of controlling plant diseases.

Disease resistance is successfully achieved after the introduction of major (or qualitative) genes into cultivars. The most popular breeding strategy of choice because major genes are easily recognized by their specificity and can thus be relatively easy manipulated in a breeding program. This type of resistance is effective until a new strain of the pathogen to which the incorporated gene does not confer resistance becomes established. Given the circumstance, a new gene for resistance is incorporated into new cultivars, if it is available on time. By repeating this process at frequent intervals, new cultivars with different resistant genes replace varieties that have become susceptible.

Not very long after the initial success, only a few of the resistance genes introduced using such strategies were able to confer resistance that remained effective in the field. Because of this failure, different strategies for enhancing the duration of resistance have been suggested. As related to durability, the types of interactions between hosts and pathogens are so diverse that probably not a model will account for the establishment of a unique strategy. Durable resistance is understood as resistance that remains effective after years of intense use in large areas. This review is intended to summarize different findings that help to visualize the magnitude of host-pathogen interactions and to discuss different terms used to relate the concept of durability with the procedures employed to achieve durable resistance to plant diseases.

Plant recognition and disease resistance

The occurrence of a plant disease is determined by the encounter of a given susceptible host with a pathogenic strain at the appropriate time. Both host physiological state and environmental conditions must be favorable for host-pathogen compatible interactions to develop. Most plants are able to avoid infection by most plant pathogens simply because they posses an array of constitutive defense compounds or because they are capable of blocking the entry to particular pathogens. Pathogens multiply only in plants that contain particularly favorable metabolites for penetration, invasion, and reproduction. Lack of recognition is common in unsuccessful pathogen-host interactions that, due to the incompatibility involved, are regarded as examples of "non-host" resistance. By the same token, "escape" refers to the presence of the pathogen infective units at the host non-receptive stages or their absence during receptive stages. In other cases initial defense responses are observed in plant tissues after being confronted by potential pathogens that causes cell death near the tissue surrounding the penetration site. The later phenomenon is regarded as "hypersensitivity" and usually serves as a means of assessing the presence of resistance genes G. N.1997)» (Agrios, G. N.1997).

Although resistance is the result of an incompatible host-pathogen interaction, neither non-host resistance nor escape would be considered achievements of durable resistance. The possibility for positive interactions underlies the concept of durable resistance, which is a descriptive term and so does not provide further explanation for its causes. Nevertheless, understanding the mechanisms employed by pathogens to successfully colonize their hosts may provide insights for the establishing of possible control strategies. Knowledge on the unifying principle governing compatibility will be of prime value for achieving resistance that is more durable. However, based on the evidence so far accumulated, there is much concern that a single model will be enough to explain such diversity observed in the interactions between hosts and pathogens R.1984)» (Johnson, R.1984).

From the several types of resistance described in plant-pathogen interactions, two of them have been explored in detail: resistance due to gene-for-gene interactions and resistance due to the plant ability to divert the activity of pathogen-produced compatible factors. Whereas in order to overcome resistance based on a gene-for-gene system the pathogen must lose the function of a gene, overcoming resistance of intervening compatible factors may only be achieved by the gain of a factor to render useless the defensive system of the plant R.1994)» (Chasan, R.1994).

The first resistance gene ever to be cloned (*Hm1*) is a gene that participates in the inactivation of a toxin (HC) produced by some strains of the fungus *Cochliobolus (Helminthosporium) carbonum* in maize. All susceptible maizes contain a non-functional *hm1* allele, compared with most plants that are resistant and contain the *Hm1* gene. It is likely that a single mutational event converted maize into a potential host for what was essentially a non-pathogen. The chromosomal region where the gene that encodes for toxin production in pathogenic strains is located segregates as a single gene. It appears to be duplicated in *tox+* strains and completely absent in toxin non-producers. Non-pathogenic *tox-* isolates have shown being equally pathogenic in susceptible maize after exogenously providing the toxin G. S., Gray, J., Gruis, D., and Briggs, S. P.1995)» (Johal, G. S., Gray, J., Gruis, D., and Briggs, S. P.1995).

Similarly, other relatively well-known pathogens take advantage of host-specific or non-specific toxins to circumvent resistance in the host, such is the case with *C. maydis* race T in cytoplasmatically inherited male sterile maize or *C. victoriae* in oats. Durable resistance to these organisms would be obtained by interfering with the pathogen's ability to suppress the plant defenses, either by: 1) altering the structure of the pathogen target; 2) introducing a factor that interferes with the toxin and its target or blocks further steps in the signal cascade; 3) degrading or modifying the toxin before its interaction with the target M. B.1995; Johal, G. S., Gray, J., Gruis, D., and Briggs, S. P.1995)» (Dickman, M. B.1995; Johal, G. S., Gray, J., Gruis, D., and Briggs, S. P.1995).

The gene-for-gene concept

The gene-for-gene concept was conceived as the simplest explanation for results obtained from studies on the inheritance of pathogenicity of the flax rust fungus *Melampsora lini*. Varieties of flax (*Linum usitatissimum*), with one gene for resistance to parents of avirulent races, were inoculated with *F2* cultures of the fungus that segregate at monofactorial ratios. Varieties having two, three or four resistance genes segregated with bi-, tri- or tetrafactorial ratios. The results suggested that, for each gene that conditions susceptibility in the host, there is a corresponding gene that conditions pathogenicity in the

parasite. Being so, either member of the system may be identified by its counterpart in the host-parasite relationship. The concept so defined may be utilized to: 1) establishing the means of variation in pathogenic fungi; 2) identifying major genes conditioning resistance; 3) elucidating the physiology of resistance and susceptibility; 4) explaining the co-evolution of host-parasite systems and; 5) developing resistant cultivars H. H.1971)» (Flor, H. H.1971).

Following Flor's procedures, intraspecific variation (virulence) of plant pathogenic fungi (races) has been largely determined based upon specialization towards different genotypes within a host species. The number of races (probably better termed "pathotypes") that can be found depends on the number of resistance factors in a set of differentials (host cultivars that possess a single, unique resistance gene). Identity of genes involved in virulence towards differentials solely defines a race without further consideration on the pathogen's genome. Two isolates can have very distinct genomic configurations but similar virulence reactions on a set of differential hosts. This means that a new differential introduced to the set can separate previously unified isolates into two new races, as it has been commonly observed. Therefore, making the determination of "races" entirely dependent on the set of differentials used Wit, P. J. G. M.1992)» (de Wit, P. J. G. M.1992).

Resistance to plant pathogens is known primarily from selective breeding of crop species while naturally occurring pathogenic events have been less documented. Nevertheless, a better understanding on the mechanics of pathogen resistance is now becoming available with the isolation and sequencing of putatively interacting plant and pathogen genes. The genetics of specificity in pathogens has turned out to be more complex than expected. In wheat alone, more than 90 genes that condition isolate-specific resistance to three rust species and powdery mildew have been identified. One of them is believed to be also involved in the recognition of more than one species. Even more, alleles with different pathogen specificity have been identified and there is evidence that genes expressing identical specificity are present at different loci and in different species. Gross similarities have been observed even in hosts with highly divergent DNA sequences. To some extent, it appears that resistance genes are members of multigene families well conserved among taxa. These families are further discriminated in their evolution by recombination or gene-conversion events. Members of these gene families may be linked, entirely unlinked, or occur in both linked and unlinked clusters I. R. and Pink, D. A. C.1996)» (Crute, I. R. and Pink, D. A. C.1996).

As an example of resistance gene complexities, *Pto*, the gene that confers resistance to *avrPto*-strains of *Pseudomonas syringae* pv *tomato* in a gene-for-gene fashion in tomato has been cloned and found to belong to a family of related genes. *Pto* is also successfully expressed in related species like tobacco. *Fen*, another member of this family maps to the same locus as *Pto* and confers resistance to the insecticide Fenthion and its genomic sequence has 80% similarity to *Pto*. A third gene (*Prf*) is required for successful responses either to the bacterial virulence or to the insecticide. It is believed that *Prf* acts as a receptor that binds both gene products and feeds them into two different signal transduction pathways G. B., Brommonschenkel, S. H., Chunwongse, J., Ganal, M. W., Spivey, R., Frary, A., Wu, T., Earie, E. D., and Tanskley, S. D.1993)» (Martin, G. B., Brommonschenkel, S. H., Chunwongse, J., Ganal, M. W., Spivey, R., Frary, A., Wu, T., Earie, E. D., and Tanskley, S. D.1993).

The finding that cloned resistance genes from diverse plant species to a wide array of viral, fungal and bacterial pathogens often encode structurally similar proteins, has indicated a high degree of mechanistic conservation to defense in plants. Others show interesting complexities like the locus of *Rp1*, a gene conferring resistance to the rust fungus *Puccinia sorghi* in maize, which contains a cluster of distinct *Rp* specificities that probably share sequence similarities. Frequent unequal crossing over has been observed at this locus and one recombinant has been shown to recognize not only every rust race but also non-rust pathogens A. F.1996; Chasan, R.1994)» (Bent, A. F.1996; Chasan, R.1994).

The nature and mechanisms controlled by disease resistance genes indicate an indirect participation of the gene products in the defense response. Resistance genes simply dictate whether or not a normal common defense response is activated. Genes in the pathogen coding for the recognized product by resistance genes play a negative role in pathogenesis and are believed to be prone to be lost. Deletion of avirulence genes has shown to be involved in avoiding recognition so promoting compatible interactions. Gene interference with specificity, one resistance gene modified by another or a susceptible allele affected by the activity of another allele of a different gene, has been also observed. Contrary to expectations for independence, weaker resistance phenotypes (strong susceptible reactions) were expressed by plants bearing two resistance genes to a combination of two isolates that promote strong and weak resistance reactions. A phenomenon observed in arabidopsis with two isolate-specific loci for *Peronospora parasitica* R.1994; Johal, G. S., Gray, J., Gruis, D., and Briggs, S. P.1995)» (Chasan, R.1994; Johal, G. S., Gray, J., Gruis, D., and Briggs, S. P.1995).

Horizontal resistance

Any plant exhibits certain level of unspecific resistance that promotes differential interactions after challenge with individual pathogenic strains, a type of resistance that is based on the assumption of multiple gene control. Each of the genes alone may not confer a high level of resistance but once combined apparently play an important role in exerting the numerous processes that constitute plant defense. In general, this so-called horizontal resistance is not believed to exclude infection but does play a role in slowing the development of epidemics in the field. The level of infection conferred by this type of resistance appears to be greatly environmentally influenced. Horizontal resistance is also known by the terms nonspecific, general, quantitative, adult-plant, field and durable resistance. The term adult or mature resistance has a horizontal resistance meaning although it has also been used to indicate changes in susceptibility observed at different ages of the plant. Field resistance is equated with horizontal resistance but its proper use is to describe resistance shown by plants in a field situation without implying what is the nature of this resistance. The terms minor gene, oligogenic, partial, polygenic, and race non-specific resistance have also been used to mean horizontal resistance G. N.1997; Nelson, R. R.1978; Robinson, R. R.1969; van der Plank, J. E.1963)» (Agrios, G. N.1997; Nelson, R. R.1978; Robinson, R. R.1969; van der Plank, J. E.1963).

The term horizontal resistance was proposed and discussed by van der Plank (1963) to distinguish it from "vertical" responses to virulence when drawn in a chart. The definition, in his own words "...when resistance is evenly spread against all ra-

ces of the pathogen ..." has been advocated as having an epidemiological and genetic framework. Breeders would like to have a genetic meaning behind concepts dealing with disease resistance and in fact, horizontal resistance affect the apparent infection rate and is probable polygenic in origin. Others argue that resistance can not be recognized or decided by the degree of its effect on disease epidemiology. It has been suggested that cultivars that become less diseased when grown in a field favorable situation are those likely to be useful sources of horizontal resistance. This is usually linked with certain characters, for example the cultivars are more difficult to infect, the period from inoculation to sporulation is longer and sporulation is less abundant R.1984; van der Plank, J. E.1963)» (Johnson, R.1984; van der Plank, J. E.1963).

Infection rate (r) is a measure of the epidemic speed. It gives an overall picture of how rapidly a population of the pathogen builds up on a population of the host. For many host/pathogen systems, it has proved a useful parameter for assessing horizontal resistance. Other parameters are inoculum efficiency, latent period, lesion type and sporulation. A given host will show horizontal resistance when more conidia are required to produce a lesion, it takes longer for a lesion to develop, and it produces smaller lesions and lesser spores. In the field, a combination of these factors leads to a low infection rate. Inoculum efficiency, which is the ratio of the number of lesions formed to the number of conidia applied, lesion size and sporulation were assessed on rice cultivars after inoculating with *Pyricularia grisea*. The results suggested differences in resistance type between the cultivars. Prediction models built using multiple linear regression statistics analysis confirmed the results from the greenhouse as predictors of infection rate in the field. Inoculum efficiency was also found the most important and commonly observed component of horizontal resistance to rice blast in the Philippines. Cultivars that have shown relatively little disease in the field for several years displayed lower inoculum efficiency and smaller lesions with lower sporulation than others did. Other components of resistance assessed, latent period and lesion size were not as reliable in establishing a correlation between the test conducted and the experience in farmers' field. Assessing horizontal resistance by using latent period is difficult, particularly on deciding whenever exactly lesions start sporulating. Small lesions in rice blast disease produce fewer conidia per night than typical diamond shape lesions and their presence has been reported as an expression of horizontal resistance. There is a great deal of difficulty however, associated to estimating lesion size. Sporulation has been proposed as the parameter that takes account for all resistant mechanisms in rice to blast R. R.1978; Villareal, R. L., Nelson, R. R., MacKenzie, D. R., and Coffman, W. R.1981; Yeh, Z. H. and Bonman, J. M.1989)» (Nelson, R. R.1978; Villareal, R. L., Nelson, R. R., MacKenzie, D. R., and Coffman, W. R.1981; Yeh, Z. H. and Bonman, J. M.1989).

There is the general assumption that horizontal resistance is durable. Durability of horizontal resistance traits has been suggested to be occurring in several diseases of plants, or nearly so, but has been difficult to probe useful in different environments and other diseases. Despite wide use, corresponding races were not observed for years towards monogenic resistance in cabbage (*Fusarium oxysporum* f. sp. *conglutinans*), in cucumber (*Cladosporium cucumerinum*) and in maize (*C. carbonum*) which are examples of durable resistance as conferred by single "specific" genes. Being race-non specific would be of particular interest to breeders. Resistance gene combinations may contribute to the durability of resistance by several potential mechanisms. In general, the probability that a pathogen can simultaneously mutate to virulence at loci corresponding to combined resistance genes is lower than mutations at a single gene, specific combinations of virulence in the pathogen may have fitness disadvantages and, pathogen fitness is more difficult to combine at multiple virulence mutations. Enhanced durability is expected to result less from the inability of the pathogen to mutate than from the time required for recombination to generate the necessary gene combinations to unmatch the combined resistance genes. Based in part on these assumptions, most breeding programs rely on gene pyramiding (combining resistance genes) to enhance durability but these are major genes and in a sense, horizontal resistance refers to resistance conferred mostly by the cumulative effects of minor genes. Though such distinction between major and minor genes is not entirely accepted, so being the result of minor effects of genes otherwise major in a different genetic background. A sort of host-pathogen equilibrium which allow certain degree of infection but do not severely damage the host. Chances are that single resistance genes or combinations of a small number of resistance genes will give higher than average resistance and factors other than gene number may also be closely associated with resistance durability C. C.1991)» (Mundt, C. C.1991) R. R.1978)» (Nelson, R. R.1978).

There has been a lack of knowledge on genetically complex and polygenic forms of disease resistance, which does not fit to simple Mendelian ratios. Quantitative genetic analyses of host-pathogen interactions are difficult to perform when dealing with obligate parasites. Continuous variation of traits showing quantitatively inherited resistance have been studied by statistical analysis for some plant resistance characters like slow rusting and slow mildewing in cereals. Inheritability, number of relevant loci, degree of dominance, additivity, heterosis and the role of "gene x gene" and "gene x environment" interactions have been analyzed from controlled crosses between parents of dissimilar resistance phenotypes. The few studies performed so far may contribute to the impression that disease specificity is controlled by simple systems with only one or a few components. The number of effective genetic factors towards disease resistance being in a lower range than in other quantitative traits likes growth, height or fertility. Several well-studied systems have shown a more complex phenomenon however, such as in barley mildew where the specialization of different isolates to a group of cultivars carrying the same resistance gene can be discerned. More recently, Quantitative Trait Loci (QTL) have been mapped using approaches that are more effective with DNA markers. Significant progress have been made on this regard in mapping QTL for resistance towards the blast fungus in rice, to late blight in potato and to gray leaf spot in maize among others M. B.1995)» (Dickman, M. B.1995) N. D.1996)» (Young, N. D.1996).

Conclusions

Durable resistance can be recognized only after a cultivar has been extensively grown for years by farmers in regions where culture conditions and environments are favorable for the disease. The term by itself is only descriptive and the association of durability to any breeding strategy is an extrapolation based mostly on partial results. Durable resistance has been associated to horizontal resistance becau-

se of the effects of the latter in slowing the progress rate of epidemics. Techniques for distinguishing horizontal resistance in breeding lines have been difficult to apply. Some success however, has been achieved by screening breeding lines using differential isolates and testing successive progenies in the field using parameters like infection rate. However, cultivars exhibiting resistance that has been maintained for long periods in certain regions have become susceptible when planted to different environments. Whether race specificity or environmental factors have been responsible for this change has not been conclusively determined.

Marker-assisted selection strategies are now available and can improve the efficiency of breeding by incorporating novel or particularly valuable genes to well-adapted commercial cultivars. Availability of resistance genes sequences and prospective resistance genes conservation among taxa has made an impact on building expectations for their use in related and unrelated species. However, demonstration of functionality using these strategies will demand substantial efforts in performing co-segregation analyses. It seems clear that no single breeding strategy would confer durable resistance to all pathogens but achievements so far attained are strongly in favor for accumulation of resistance genes. Even susceptible hosts could possess genes that might be effective when transferred to a new genetic background and crosses between individually susceptible parents have led to gene interactions conferring enhanced resistance in the progeny. The results may be considered less predictable when the pathogens freely recombine in the field. However, it is in the function of the entire plant genome and from its interaction with the pathogen that the principle governing resistance and, perhaps, durability lays.

BIBLIOGRAPHY

Agrios G. N. *Plant Pathology.* 4 ed. San Diego, CA: Academic Press Limited. i-xvi-635 1997.

Bent A. F. Plant disease resistance genes: function meets structure. *The Plant Cell* (1996): 1757-71.

Chasan R. Plant-pathogen encounters in Edinburgh. *The Plant Cell* (1994): 1332-41.

Crute I. R. and D. A. C. Pink. Genetics and utilization of pathogen resistance in plants. *The Plant Cell* (1996): 1747-55.

de Wit P. J. G. M. *Molecular characterization of gene-for-gene systems in plant fungus interations and the application of avirulence genes in control of plant pathogens.* Annual Review of Phytopathology. nº. 30. Palo Alto, Calif.: Annual Reviews Inc., 1992.

Dickman M. B. *The molecular biology of plant-fungal interactions.* Chap. in *Disease analysis through genetics and biotechnolgy.* Iowa StateUniversity Press, 1995. 149-95.

Flor H. H. The inheritance of pathogenicity in a cross between physiologic races 22 and 24 of *Melanospora lini. Phytopathology* 5 (1942): Abstracts

Flor H. H. Host-parasite interaction in flax rust - its genetics and other implications. *Phytopathology* (1945): 680-5.

Flor H. H. *Current status of the gene-for-gene concept.* Annual Review of Phytopathology. nº 9. Palo Alto, Calif.: Annual Reviews Inc., 1971.

Johal G. S., J. Gray, D. Gruis, and S. P. Briggs. Convergent insights into mechanisms determining disease and resistance response in plant-fungal interactions. *Canadian Journal of Botany* Suppl. 1 (1995): S468-S474

Johnson R. *A criticl analysis of durable resistance.* Annual Review of Phytopathology. nº 22. Palo Alto, Calif.: Annual Reviews Inc., 1984.

Martin G. B., S. H. Brommonschenkel, J. Chunwongse, M. W. Ganal, R. Spivey, A. Frary, T. Wu, E. D. Earie, and S. D. Tanskley. Map-based cloning of a protein kinase gene conferring disease resistance in tomato. *Science* (1993): 1432-6.

Mundt C. C. Probability of mutation to multiple virulence and durability of resistance gene pyramids: further comments. *Phytopathology* 3 (1991): 240-2.

Nelson R. R. *Genetics of horizontal resistance to plant diseases.* Annual Review of Phytopathology. nº 16. Palo Alto, Calif.: Annual Reviews, Inc., 1978.

Robinson R. R. Disease resistance terminology. *Review of Applied Mycology* 11-12 (Dec 1969): 593-605.

van der Plank J. E. *Plant Disease: Epidemics and Control.* New York: Academic Press. 1-349. 1963.

Villareal R. L., R. R. Nelson, D. R. MacKenzie, and W. R. Coffman. Some components of slow blasting resistance in rice. *Phytopathology* (1981): 608-11.

Yeh Z. H. and J. M. Bonman. Assessment of partial resistance to *Pyricularia oryzae* in six rice cultivars. *Plant Pathology* (1989): 319-23.

Young N. D. *QTL mapping and quantitative disease resistance in plants.* Annual Review of Phytopathology. nº 34. Palo Alto, Calif.: Annual Reviews Inc., 1996.