Plenary Session

An Agenda for Phytopathology

Robert M. Goodman

Calgene, Inc., 1920 Fifth Street, Davis, CA 95616

ABSTRACT

Plant pathology has a long and distinguished history of incorporating knowledge and assimilating people from other disciplines of science. The rapid developments in molecular biology and the interests shown in plant pathology as a research field by molecular biologists are the latest external development that help shape our field. Plant pathology also has played a central role in modern agriculture. Some of the practices that have become accepted in agriculture, for environmental or economic reasons or both, must change. These changes include disease management practices. With new tools, including those of recombinant genetics, the field of plant pathology is better equipped than ever to respond to the challenge of improving on agricultural practices. Already, molecular biology is providing answers to some of the classic problems in plant pathology and helping form the basis of new disease management options.

It is a historical fact that plant pathology, from its origins in 19th century natural history to its contemporary role as a cornerstone of modern agriculture, has assimilated new knowledge, new technology, and people from other areas of science. The choice of theme for this annual meeting indicates a clear recognition that once again new knowledge is changing plant pathology. Some unfortunately see in these changes a threat to the discipline. Luis Sequeira (29), in his presidential address to the society last year, spoke about the attitude that still prevails in some quarters that regards molecular biology as some harmful fad. That view is wrong. To the contrary, the new biological knowledge base represents perhaps the greatest opportunity yet to approach and get answers to some of the classic problems in plant pathology. In change, I see nothing new for plant pathology. I wish to address the challenges and opportunities ahead and relate my view of the future both to the disciplinary traditions of our past and the agenda I see for agriculture in the coming decades.

People

A creative response to the forces of change depends ultimately on how we invest in people. Plant pathology has a past that is distinguished in its recruitment and assimilation of people. I can illustrate from my own experience how important the influence of people can be. My paternal grandfather was Alpheus Mansfield Goodman, an agricultural engineer, Cornell professor, and international agriculturalist. From him and my "Cornell" upbringing I learned many things, including about the great plant pathologists H. H. Whetzel and Cynthia Wescott and the great horticulturalist Liberty Hyde Bailey. These influences presaged later decisions, making plants, agriculture, and international interests predictable if not inevitable choices. But it was a high school science project fueled by the generosity of Tom Kruk in Bill Mai's lab at Cornell that four years later brought me to Carl Boothroyd's office as one of his many undergraduate advisees.

Through undergraduate, graduate, and postdoctoral study I was influenced by many others. These included George Kent, John Chen, Andre Jagendorf, Ray Wu, Bob Horne, Roy Markham, Bill Rochow, and Frank Ross. When I was a young faculty member at the University of Illinois, it was David Gottlieb and Jim Gerdemann who for me set the high standards of scholarship, clarity of purpose, and interest in the professional and personal development of students, to which I aspired.

Plant pathology has been rich with distinguished men and women of the caliber of the few I have mentioned. As I reflect on the influential people in my past, I cannot help thinking that, in view of the other forces at work on the field of plant pathology, academic departments of plant pathology and the society have a major task ahead in preserving and refreshing the strong traditions and values of the past as represented by these people.

What was it about these people that made a difference? One feature of them all is that, whether from within or outside the field of plant pathology, they saw its breadth and its depth. Some had been trained in our field, but many were recruits by virtue of where their interests led them after postgraduate education. They had great personal attributes as well. Their hand on the inquiry of their students was light but sure. They were not merely broad-minded, though they certainly were that. They combined broad-mindedness with high expectations. They were also alive to much more than plant pathology. They read widely and expected their students to do the same. I shall never forget discussions with Roy Markham about the ideal acoustic speaker for faithful reproduction of music, with Bill Mai about his passion for gardening, with Jim Gerdemann about rhododendrons, with Dave Gottlieb about his lust for exotic travel, with Bill Rochow about the future of the Adirondacks preserve in upstate New York.

Plant pathology has had its share of great men. It is a notable development of the past 20 years or less that the field has also recruited and become the career vehicle for women. This happy development has occurred despite the lack of enthusiasm toward women in graduate school shown in the 1960s by some of the influential men I have mentioned. It is not too early to judge what the dramatically increased census of women has done to the field of plant pathology. We have seen rapid career development and major contributions by women in many departments of plant pathology around the country.

The record for bringing minorities into plant pathology, on the other hand, is not good. This is a situation I do not understand. I think it may have less to do with the reception minority individuals receive in our field than with the historical unattractiveness of agriculture to minorities. There certainly is a perception, entirely justified by statistics and demographics, that plant pathology does not provide an attractive or proven career path for Hispanics, blacks, Asians, and other traditional minorities in North America.

Technology

Within the applied plant sciences, plant pathology has a historical and perhaps unique centrality. The study of plant interactions with pathogens draws on virtually every biological discipline. And it extends well beyond, for example, from atmospheric sciences to applied mathematics and to many branches of chemistry. It is the only applied plant science discipline having strong affiliations with both vertebrate (via immunology) and invertebrate zoology. It is the meeting ground of genetics, development, physiology, ecology, and (considering insect vectors of plant pathogens) behavioral biology. It has been historically strong in systematics.

Plant pathology has also always been a field that is responsive to new technology. Though practiced by a generally conservative group of people, plant pathology has at times quite quickly adopted (and occasionally contributed to) innovative methodologies. Plant pathology is a field rich with opportunity to bring the concepts, tools, and people from many disciplines together. It is no surprise, then, that our field is one the new tools of genetic manipulation make extraordinarily attractive to scientists in other disciplines. Plant pathology must, for its future vitality and relevance, recognize its scientific centrality as a distinguishing hallmark of its past—and it must maintain and strengthen its commitment to importing knowledge, methodology, and people from other fields. We must resist use of a restrictive definition or narrowly "professionalize" what it means to be a plant pathologist. We must maintain our traditions, our values, and our organizational identity. To do so, as I gave argued, means to welcome and encourage migration from other disciplines. We must likewise be flexible with the curricula of the graduate schools of our leading research universities and draw widely from the pool of talent available when we employ new faculty.

An Agenda for Agriculture

Modern production agriculture faces major crises—environmental and economic—that must be addressed in the next several years. Underlying all of the concerns over land values, export markets, and temporary surpluses are some fundamental issues that are not receiving adequate attention. It is unacceptable that we are eroding the quality of the natural, renewable resources on which agriculture depends. Soil loss from erosion, increases in salinity, groundwater contamination due to farm chemicals, and eutrophication of lakes and ponds due to fertilizers affect too much of our prime farm land. It is unacceptable that there is high pressure on use of marginal land for farming. Around the globe, rain forests are threatened and desertification is expanding. In the United States, farm policy continues to promote use of marginal lands to support base acreage for commodity support payments. It is unacceptable that germ plasm resources are being lost through neglect and as the result of the encroachment of the modern crop varieties created from the land races and noncultivated wild relatives that are now threatened. And it is unacceptable that agricultural policy in much of the developed and developing world reinforces that status quo by providing serious disincentives for change.

The agenda I see is to bring back a focus on management and increase the emphasis on genetics in the practice of agriculture. We must learn to manage the agricultural enterprise in a manner consistent with sound ecological principles. In a well-managed system, what is taken from the land in the form of product is returned, and the inputs stay where they are put until called upon for production. The full potential of the biological, physical, and chemical system is used in a manner that is *sustainable*. A sustainable system managed for high productivity will have as a major ingredient plants and animals with high genetic potential. In plants, the focus will be, as it has always at some level been in plant pathology, to do with genetic modification what would otherwise be done with chemicals or tolerated as loss.

Concerns over the environmental implications of modern production agriculture aside, the agenda I see of improved management and expanded use of genetic as opposed to chemical solutions is today driven by economic considerations. At contemporary farm prices, growers simply cannot afford the level of purchased inputs to which many have become accustomed. A new balance must be struck. The time is right for new approaches and for the use of new, more powerful tools for genetic improvements that avoid the economic and environmental costs of many present day practices.

This period of economic turbulence, low prices, and excess production presents a real opportunity to plant pathologists and to everyone in agriculture. It is a breathing space. It is time—perhaps only a brief time before the urgency of increasing production to avert famine returns—to get things right, to set a new agenda. Time to answer some fundamental questions about the genetic basis of disease and resistance. Time to evaluate more completely our germ plasm collections of major and minor crops for resistances and other useful traits. Time to attend to the sorry state of systematics and the neglected field of agricultural ecology. Time to develop new more powerful diagnostic tools. And time to integrate with our sister disciplines and begin to test the usefulness of new knowledge in the field and apply it on the farm in promoting the agenda of sustainable agriculture.

Impact of the New Genetics: Some Contemporary Examples

Recombinant DNA methodology is already having a profound effect on plant disease research. Incorporation of recombinant DNA tools into the research of plant pathologists is intense. So also is the migration of molecular biologists to the field of plant pathology from other traditions and backgrounds. Recombinant DNA technology is moving across the plant pathology landscape like a brushfire across drylands, renewing and revitalizing the science and the scientists that it touches.

I wish now to turn to a selected set of examples of contemporary research using the new biology. My examples are chosen from the work I know best. While biased towards virology, they are chosen to represent what is underway throughout much of our discipline. They address a broad range of topics—from gene transfer to understanding virulence and resistance, to controlling diseases in new and unexpected ways.

The Tools

The study of plant pathogens has made two major contributions to the development of and applications of recombinant DNA technology to plant improvement (11). The first is *Agrobacterium tumefaciens*, the crown gall pathogen. One of the elegant stories in contemporary biology has been the dogged pursuit, over 50 years, of the etiology of crown gall disease. It is a story that began in plant pathology, was solved largely outside the traditional realm of plant pathology and even of agriculture, and has now returned to wide use and further investigation in plant pathology.

Agrobacterium tumefaciens (19) harbors a large plasmid, called Ti (tumor-inducing), part of which (the T-DNA), is transferred to and integrated into the chromosomes of susceptible plants. Transfer is induced by diffusible chemical signals produced by the plant when wounded. These signals induce expression of other genes on the Ti plasmid, outside the T-DNA region, whose functions *in trans* accomplish the transfer and integration in a way that is not completely understood. Recent indications suggest analogies to the mechanisms of DNA transfer in bacterial conjugation. *Agrobacterium* is the only case we know of in which transfer and integration of DNA from a foreign source occurs naturally in plants.

Crown gall disease is the result of expression in the transformed plant of genes encoded by the T-DNA (23). Three of these genes encode enzymes involved in biosynthesis of the hormone-like growth substances indole acetic acid (an auxin) and iso-pentenyl adenine (a cytokinin). By removing these genes, useful gene transfer vectors, the so-called disarmed vectors, have been created. That is, it turns out that the causes of disease and the transfer functions are independent. Moreover, the T-DNA can be put on a second, or binary, plasmid, where it can be more easily manipulated in *Escherichia coli*. The plasmid carrying an engineered T-DNA is then transferred back to *A. tumefaciens*. Such vectors can be used for gene transfer in a number of plant species. In some it is very routine. For example, in tomato, transgenic plants by the hundred or thousands can be routinely obtained. Transgenic plants can be selected directly and can be rooted and in soil within eight weeks or less from the initiation of the experiment.

A second important contribution of plant pathology has been the detailed understanding we now have of the two groups of DNA plant viruses, the cauliflower mosaic virus (CaMV) group and the geminiviruses (20). One of the transcriptional regulatory signals that governs gene expression and replication of CaMV—the 35S promoter—is proving to be a remarkably versatile gene expression tool in many plants. It is a constitutive regulatory element, and it works well in plant species outside the host range of the virus. It is the highest level and most versatile regulatory sequence available today to the plant genetic engineer.

Results That May Matter

I now wish to briefly illustrate some examples of contemporary research that bear on our understanding of disease and resistance thereto or on the future of plant disease control, or both, and that derive from the use of recombinant DNA technology.

Virus resistance in transgenic plants expressing viral coat protein genes. First with tobacco mosaic virus (1,4,24) and now with several other virus groups (30), it is turning out that the simple step of producing in the cells of the susceptible plant the coat protein of a virus protects, in some cases completely, against infection by the virus and its close relatives. This phenomenon is being interpreted as, and may well be, molecular cross-protection (1). The concept is that the coat protein already in the cell when the infective virus particle arrives occupies and perhaps saturates some receptor, as yet unknown, that the virus must find to begin a productive infection.

Virus resistance in transgenic plants by expression of satellite RNAs. Satellite RNA associated with infections of plant viruses were first discovered as physical entities "contaminating" virus preparations of tobacco ringspot virus. Subsequently, and increasingly over the past several years, many small, dependent satellite RNAs have been found associated with RNA plant viruses. Most, if not all, such RNAs have an effect on the course of disease, and particularly on symptom expression, of the virus with which they are associated. In many instances, the effect is symptom suppression. Several laboratories have now produced transgenic plants that produce these satellite RNA sequences. The interesting result is that when such a transgenic plant is inoculated with a satellite-free isolate of the virus, the satellite RNA sequences begin to replicate and effect the same changes in the disease caused by the virus as would be seen if nontransgenic plants were inoculated with a satellite-containing inoculum of the virus (2,9,13).

A virus resistance factor that interferes with a critical step in the pathogen's life cycle. A third example from the annals of recent molecular virology is that of resistance in Arlington cowpeas to cowpea mosaic virus, a comovirus. A key feature of the comovirus life cycle is the translation from viral messenger RNA of polyproteins. These are large precursor protein molecules that are then processed by proteolytic cleavage to the mature functional proteins. In comoviruses, the processing enzyme is also encoded by the virus (8). What Bruening and co-workers (26) have found is that the cowpea line, Arlington, which is highly resistant to the virus, encodes an inhibitor of the virus-specific proteolytic enzyme. The resistance segregates as a single dominant genetic trait. In extracts of near-isogenic lines supplemented with viral translation products, that from the recessive homozygous (susceptible) plant processes a 95 kD precursor protein to its 60 and 48 kD products, whereas that from the dominant homozygous (resistant) plant does not. It does not take much to imagine the potential applications of this result for virus control were it to turn out that the inhibitor is a protein, as some protease inhibitors are, and one had the gene encoding it in hand. The significance of this result looms large in agriculture when you consider that two virus groups of major agricultural importance, the potyviruses and nepoviruses, use the same posttranslational processing strategy.

Acquired resistance to plant pathogens induced by avirulent strains, hypersensitive responses, and some chemicals. Systemic acquired resistance (SAR) is one of the classic problems in plant pathology. Recombinant DNA tools are now being applied by numerous laboratories to get at the basis of this phenomenon. A better understanding of the SAR is of interest, not the least because of the generality of the resistance that is induced and also because the expression of this type of resistance in the field has proven to be disappointingly variable. In the classic work of Frank Ross (27,28), tobacco mosaic virus applied to a hypersensitive-responding tobacco variety was used to induce the resistance. Several days later, challenge inoculation to distant parts of the plant showed substantial protection, as indicated by reduced lesion size and number. Others (21,22) showed that such tissues also displayed resistance against other pathogens, and even an aphid. Other protocols, including avirulent strains of pathogenic fungi and even certain chemicals can induce a similar effect.

Recent results from the literature (10,15–17,31) show that correlated with induction of resistance is the induction of certain mRNAs that we now know encode a group of acid-soluble proteins that had earlier been associated with the SAR phenomenon, the so-called b- or pathogenesis-related (PR) proteins. The actual cause of the resistance is still unclear as are the mechanisms by which the resistance and the PR proteins are induced and the role if any of the PR proteins in causing resistance. An interesting recent result in work on PR proteins has been the discovery—through sequencing the genes (25) and by molecular histology (5)—that these proteins are exoproteins. That is, they are transported through the plasmalemma into the apoplast.

Understanding pathogenicity and virulence. Work in this area is proceeding on a broad from, from viroids to bacteria to fungi. I shall cite just one brief example where the simplicity of the system would seem to lend itself to making life easy, but it's not. Viroids are as small as the aforementioned satellite RNAs, but unlike the satellite RNAs, they are independently able to replicate and they can cause severe disease. Their origins and the mechanisms by which they cause disease are still mysteries. Site-specific mutagenesis (12,32) and sequence analysis of naturally occurring variants (18) are being used to try to deduce answers to the functional questions of replication and disease. For such a small entity to have functional domains, as this type of analysis is resulting in the definition of, is quite surprising.

Mapping and cloning genes for disease resistance. The DNA-cutting specificity of restriction endonucleases and our knowledge about mobile elements (controlling elements or tranposons) provide two new powerful approaches to identifying, tagging, and cloning genes that condition disease resistance (and numerous other genetically well-defined traits). Just as isozyme markers for particular enzymes found use in following segregation of certain genetic traits, polymorphism in the lengths of restriction fragments, which are characteristics of the DNA itself, can be used to correlate with genetic traits and locate on chromosomes the genes responsible for those traits (3,14).

A genetic map based on restriction fragment length polymorphism analysis for tomato has been produced (33). Three restriction fragment length polymorphism markers have been identified that are very closely linked to a genetic locus, Tm2, conditioning resistance in tomato to tobacco mosaic virus (S. D. Tanksley, N. D. Young, and D. Zamir, *personal communication*). Use of such clones to identify genomic clones in a library near or spanning the gene of interest thus becomes a realistic strategy for cloning genes encoding such a specific trait as disease resistance.

Mobile genetic elements (6) can also be used to identify and tag a gene so that it can then be isolated (7). The concept is to derive, through genetic crosses or by gene transfer, a population of plants containing such a mobile element and then to screen for the loss of a dominant trait (such as disease resistance or susceptibility). A proportion of such phenotypically altered plants is expected to harbor the genetic element in or very near the gene encoding the trait. The specific DNA sequence of the tag can then be used to follow the gene in confirmatory genetic experiments (for example, to rule out tagging of distant *trans* acting factors on gene expression) and as a tag to identify and isolate specific clones of genomic DNA that carry the tagged gene. Sequences flanking the tag are then used to go into a library from a nonmutant plant and clone out a native copy of the gene (7).

Conclusions

My examples have been a small sample of exciting developments. There is already a plentiful supply of similar advances, for example in understanding pathogenicity and virulence in plant pathogenic bacteria and fungi. Diagnostic plant pathology is also taking advantage of new advances in technology. Systematics and epidemiology are not yet "in the loop" but the opportunities are so profound as to not remain untouched for long. Molecular plant pathology is as ready a tool for practical work as it is for basic science. Our challenge is to place it in its proper place and then work to achieve the new balance—of people, of resources, and of the research agenda. The APS has taken important steps. A notable move by the society is the introduction of the new journal, *Molecular Plant-Microbe Interactions*. Another is the theme of this national meeting. As Luis Sequeira pointed out last year, we still have to get right the appropriate level and balance of funding. Internal squabbles over how to divide up the dime, however, when what should be being spent is a dollar, are counterproductive. What we need to do is exercise the leadership needed to see that something closer to the dollar is available.

I wish to close with a reiteration of my view that we in plant pathology are central to agriculture in ways that rival all and surpass most other fields of agricultural science. We must take this position seriously, both in light of agriculture's needs in the closing decade of the 20th century and in light of the new powerful technology that is now available to do our work. So I put to you the question, what will plant pathology do? Will it address the major challenges of sustainable agriculture? Will it seek alternatives to the damaging practices that it has at times in the past helped to promote? Will it adjust its niches in higher education and research to reflect future opportunities and needs? Will it take the lead in the renovation that is called for—both from need and from opportunity—of the public research agenda?

I am convinced that plant pathology can be a stronger, more influential, and more satisfying science—and a more useful public service—in the future even than it has been at times in the past. It will take successful integration of new technology and a steady assimilation of new people into the field to make it so. As I have tried to argue, that is our intellectual tradition. We can and must do it again.

ACKNOWLEDGMENTS

I thank David Baulcombe, George Bruening, Roger Beachy, Kim Chonoles, Rosemarie Hammond, Robert Owens, Steve Tanksley, Nevin Young, and Dani Zamir for providing access to data and illustrations used in the presentation of this address, some of it then unpublished, and Christine Facciotti for assistance in preparing the manuscript.

LITERATURE CITED

1. Abel, P. P., Nelson, R. S., De, B., Hoffman, N., Rogers, S. G., Fraley, R. T., and Beachy, R. N. 1986. Delay of disease development in transgenic plants that express the tobacco mosaic virus coat protein gene. Science 232:738-743.
2. Baulcombe, D. C., Saunders, G. R., Bevan, M. W., Mayo, M. A., and Harrison, B. D. 1986. Expression of biologically active viral satellite RNA from the nuclear genome of transformed plants. Nature (London) 321:446-449.
3. Beckmann, J. S., and Soller, M. 1986. Restriction fragment length polymorphisms in plant genetic improvement. Oxford Surv. Plant Mol. Cell Biol. 3:196-250.
4. Bevan, M. W., Mason, S. E., and Goelet, P. 1985. Expression of tobacco mosaic virus coat protein by a cauliflower mosaic virus promoter in plants transformed by Agrobacterium. EMBO J. 4:1921-1926.
5. Carr, J. P., Dixon, D. C., Nikolau, B. J., Voelkerding, K. V., and Klessig, D. F. 1987. Synthesis and localization of pathogenesis-related proteins in tobacco. Mol Cell Biol. 7: 1580-1583.
6. Doring, H. P., and Starlinger, P. 1986. Molecular genetics of transposable elements in plants, Annu. Rev. Genet. 20:175-200.
7. Fedoroff, N. V., Furtek, D. B., and Nelson, O. E. Jr. 1984. Cloning of the *bronze* locus in maize by a simple and generalizable procedure using the transposable controlling element *Activator* (Ac). Proc. Natl. Acad. Sci. USA 81:3825-3829.
8. Garcia, J. A., Schrijvers, L., Tan. A., Vos. P., Wellink, J., and Goldbach, R. 1987. Proteolytic activity of the cowpea mosaic virus encoded 24K protein synthesized in *Escherichia coli*. Virology 159:67-75.
9. Gerlach, W. L., Llewellyn, D., and Haseloff, J. 1987. Construction of a plant disease resistance gene from the satellite RNA of tobacco ringspot virus. Nature 328:802-805.
10. Gianinazzi, S., and Ahl, P. 1983. The genetic and molecular basis of b-proteins in the genus *Nicotiana*. Neth. J. Plant Pathol. 89:275-281.
11. Goodman, R. M., Hauptli, H., Crossway, A., and Knauf, V. C. 1987. Gene transfer in crop improvement. Science 236:48-54.
12. Hammond, R. W., and Owens, R. A. 1987. Mutational analysis of potato spindle tuber viroid reveals complex relationships between structure and infectivity. Proc. Natl. Acad. Sci. USA 84:3967-3971.
13. Harrison, B. D., Mayo, M. A., and Baulcombe, D. C. 1987. Virus resistance in transgenic plants that express cucumber mosaic virus satellite RNA. Nature 328:799-802.
14. Helentjaris, R., King, G., Slocum, M., Siedenstrange, C., and Wegman, S. 1985. Restriction fragment polymorphisms as probes for plant diversity and their development as tools for applied plant breeding. Plant Mol. Biol. 5:109-118.
15. Hooft van Huijsduijnen, R. A. M., Cornelissen, B. J. C., van Loon, L. C., van Boom, J. H., Tromp, M., and Bol, J. F. 1985. Virus-induced synthesis of messenger RNAs for precursors of pathogenesis-related proteins in tobacco. EMBO J. 4:2167-2171.
16. Hooft van Huijsduijnen, R. A. M., van loon, L. C., and Bol, J. F. 1986. cDNA cloning of six mRNAs induced by TMV infection of tobacco and a characterization of their translation products. EMBO J. 5:2057-2061.
17. Jamet, F., and Fritig, B. 1986. Purification and characterization of 8 of the pathogenesis-related proteins in tobacco leaves reacting hypersensitively to tobacco mosaic virus. Plant Mol. Biol. 6:69-80.
18. Keese, P., and Symons, R. H. 1985. Domains in viroids: Evidence of intermolecular RNA rearrangements and their contribution of viroid evolution. Proc. Natl. Acad. Sci. USA 82:4582-4586.
19. Klee, H., Horsch, R., and Rogers, S. 1987. *Agrobacterium*-mediated plant transformation and its further applications to plant biology. Annu. Rev. Plant Physiol. 38:467-487.
20. Kridl, J. C., and Goodman, R. M. 1986. Transcriptional regulatory signals from plant viruses. BioEssays 4:4-8.
21. Lovrekovich, L., Lovrekovich, H., and Stahmann, M. A. 1986. Tobacco mosaic virus-induced resistance to *Pseudomonas tabaci* in tobacco. Phytopathology 58:1034-1035.
22. McIntrye, J. L., Dodds, J. A., and Hare, J. D. 1981. Effects of localized infections of *Nicotiana tabacum* by tobacco mosaic virus on systemic resistance against diverse pathogens and an insect. Phytopathology 71:297-301.
23. Morris, R. O. 1986. Genes specifying auxin and cytokinin biosynthesis in phytopathogens. Annu. Rev. Plant Physiol. 37:509-538.
24. Nelson, R. S., Abel. P. P., and Beachy, R. N. 1987. Lesions and virus accumulation in inoculated transgenic tobacco plants expressing the coat protein gene of tobacco mosaic virus. Virology 158:126-132.
25. Pfitzner, U. M., and Goodman, H. M. 1987. Isolation and characterization of cDNA clones encoding pathogenesis-related proteins from tobacco mosaic virus infected tobacco plants. Nucl. Acid Res. 15:4449-4465.
26. Ponz, F., Glascock, D. B., and Bruening, G. 1987. An inhibitor of polyprotein processing with the characteristics of natural virus resistance factor. Mol. Plant-Microbe Interact. 1:25-31.
27. Ross. A. F. 1961. Localized acquired resistance to plant virus infection in hypersensitive hosts. Virology 14:329-339.
28. Ross, A. F. 1961. Systemic acquired resistance induced by localized virus infections in plants. Virology 14:340-358.
29. Sequeira, L. 1987. Facing the issues in plant pathology. Phytopathology 77:24-28.
30. Van Dun, C. M. P., Bol, J. F., and Van Vloten-Doting, L. V. 1987. Expression of alfalfa mosaic virus and tobacco rattle virus coat protein genes in transgenic tobacco plants. Virology 159:299-305.
31. van Loon, L. C. 1985. Pathogenesis-related proteins. Plant Mol. Biol. 4:111-116.
32. Visvader, J. E., and Symons, R. H. 1986. Replication of *in vitro* constructed viroid mutants: Location of the pathogenicity-modulating domain of citrus exocortis viroid. EMBO J. 5:2051-2055.
33. Young, N. D., Miller, J. C., and Tanksley, S. D. 1987. Rapid chromosomal assignment of multiple genomic clones in tomato using primary trisomics. Nucl. Acid Res. In press.