ANNUAL REVIEW OF PHYTOPATHOLOGY

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PREFACE

With this, the first issue of the Annual Review of Phytopathology, we launch a new ship. We shall sail on new seas. We venture to hope that our scientific colleagues will approve.

Originally, the American Phytopathological Society intended to establish a journal of perspectives in plant pathology, but the society gratefully deferred to an older and more experienced organization when Annual Reviews, Inc. offered to publish it. At the request of Annual Reviews, Inc., the society at its 1961 meeting at Biloxi, Mississippi suggested names for an Editorial Committee as listed below. The nominees were appointed to the new Editorial Committee. One new name, R. A. Ludwig, was added at the first meeting, and K. F. Baker was chosen as Associate Editor at the second meeting.

We sail on a new ship on new seas as we launch a publication for synthesizing our science. A few texts have assayed this task for phytopathology but no periodical, heretofore.

The science of phytopathology has come of age. In 1946 W. B. Brierley the eminent British phytopathologist said, "Up to the present the study of plant disease has remained merely an increasing aggregate of data . . . . It has not attained the status of a science . . . for such an aggregate of data becomes a science in so far as it develops a consistent body of theory which correlates the facts into a logical and explanatory system." (*Ann. Appl. Biol.*, **33**: 336–37, 1946).

The establishment of the *Annual Review of Phytopathology* marks the emergence of our field as a science in Brierley's sense. We now have our own journal of synthesis, our own journal of critical review, our own journal of perspectives, in short, our own journal to "develope a consistent body of theory."

In this new publication we the phytopathologists of the world can take the assemblage of facts in the journals of original publication and meld them into an evocative and provocative intellectual structure, a symphony if you will.

The Editorial Committee has transmitted these aims and aspirations to the authors. We have asked the authors to synthesize new knowledge from the bits and pieces scattered through the literature. We ask for more than a critical evaluation and summary of the literature. We ask for an integration of knowledge.

We hope that the authors will look upon themselves as architects of a new structure. We ask them to design their own ideas of beauty into it.

We have said to them, "We would appreciate your personal perspective of your topic, your imaginative appraisal of it, not merely a synoptical summary. If you need to speculate, please do so, as long as you label it. If you

need to use your unpublished data to brick in a gap, please use them. We are confident that you will deliver a scholarly contribution to knowledge."

Thus, we hope that the *Annual Review of Phytopathology* will become a stimulating vehicle for our science. A stimulating vehicle for encouraging new research, imaginative research. We further hope that in so doing it will be useful to other scientists with similar views and similar problems.

We cannot close this preface without thanking Dr. J. Murray Luck, Dr. Laurence R. Blinks, and Dr. Windsor C. Cutting of Annual Reviews, Inc. for their encouragement along the way. Especially we thank Mrs. Prudence H. Whittemore, Assistant Editor, for her invaluable understanding of the problems involved, and enthusiastic support in preparation of this first volume. We also thank Mr. Rudolph E. Reichle, Department of Plant Pathology, University of California, Berkeley, California for his skill and generous cooperation in preparing the subject index.

K. B. A. K.
C. H. R. L.
J. H. G. P.
W. S.

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THE FUTURE OF PLANT PATHOLOGY

By J. C. Walker

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One hundred years ago there was no science of plant pathology as such although crop losses from plant diseases had been occurring for centuries. While we are justified in pointing to the work of Prevost (6) (1807) as the first irrefutable evidence of the causative nature of fungi in plant disease and to the early paper by de Bary (2) (1853) as the next one to support this concept, it was not until the early 1860's that the real momentum built up which led to the crystallization of plant pathology as a distinct discipline.

It should not be forgotten that de Bary was the outstanding plant morphologist of his day, and that his investigations in plant pathology were not his only and possibly not his greatest contributions to science. He became much interested in fungus morphology and life cycles, but he apparently was influenced by the economic pressures brought on by plant disease epidemics. Thus, he gave some attention to such problems as those related to late blight of potato, and rusts and smuts of cereals. In any case, his classical researches in these areas were not only scientific monuments in themselves, but they provided the evidence and incentive which put plant pathology on the road and attracted many of the brilliant minds of the day to its cause.

It followed quite naturally, as it has in any new science, that the bulk of the attention of investigators in the next few decades was concerned with the recognition of many fungi old and new to science as causal agents in plant disease. Beginning in the 1870's, bacteria began to fall in line although de Bary regarded them as unimportant plant pathogens. In the 1880's, viruses began to be recognized as different from bacteria but still infectious. Although descriptions of new viruses and virus diseases piled up rapidly, the nature of the virus entity was the subject of controversy until as late as 1935.

We are accustomed to refer to these early decades as the "descriptive era" in plant pathology. In one sense this is true, but the label tends to fog the issue. We are inclined to forget that during these last few decades of the 19th century men were struggling to get across the fact to the general public that microorganisms were causal agents of plant disease. As late as 1884, de Bary (3) lamented in the introduction to his well-known *Comparative Biology, etc.*, that "there are some who still think that fungi and bacteria . . . are produced by spontaneous or heteromorphous generation. . . . " de Bary's book (3) was a landmark and Erwin F. Smith (8) reminiscing 42 years later remarked that this magnificent volume when it appeared "was our *vade mecum* albeit the German was rather tough reading." It is well to recall also that as late as 1901, Smith (7) was still defending his proof that brown rot of solanaceous plants, black rot of crucifers, and bacterial wilt of cucurbits were incited by bacterial organisms.

In the first four decades of the present century many virus diseases were newly described, while controversy went on as to what a virus really was. Most pathologists were quite unprepared and unwilling to accept Stanley's (9) pronouncement in 1935 that the tobacco mosaic virus was not an organism at all, but a chemical entity with the implication that it was a product of the metabolism of the "host" cell itself.

Fortunately, de Bary was not a describer of diseases and of fungi. When we glibly refer to the present decade as the dawn of the physiological era of plant pathology, we forget that de Bary was exploring this area in the 1880's when he studied the pathogenicity of *Sclerotinia*, demonstrated the action of an exoenzyme on the host, and postulated the influence of the host substrate on pathogenicity (4). Let us not forget that L. R. Jones (5) in a classical research initiated in 1901 demonstrated for the first time the bacterial pectolytic exoenzymes, and laid the basis for our understanding of the physiology of bacterial soft rot. Let us not forget that as early as 1882, H. Marshall Ward (10) emphasized the relation of environment to the epidemiology of coffee rust in Ceylon where the disease was wiping out the plantations. Let us not overlook the classical work of E. T. Bartholomew (1) (1915) on potato black heart, one of the very first pieces of research to elucidate the effect of abnormal environmental conditions on the metabolism of host cells. This work demonstrated most clearly how environment can bring on disease. After all, a so-called fungus or bacterial pathogen may merely tip-off (incite) a chain of reactions which leads to macroscopic signs of disease.

There are many basic and now classic researches over the last 50 years too often "buried" in the literature which serve as monuments of progress in physiology of plant disease. The same may be said for the nature of fungicidal action; the nature of host-parasite interaction; and the nature of plant viruses. The question before us is, where do we as plant pathologists stand today? What of the future?

In the past two decades many new techniques have developed in physiology, genetics, chemistry, and bacteriology which can be and are being used to pry more deeply into the unsolved mysteries of plant disease. These problems are of general biological interest on the one hand; their solutions on the other hand are essential to the future control of plant disease and the assurance of adequate supplies of food, feed, fibre, ornamentals, and of suitable recreation areas. It is obvious that beginners in plant pathology, as well as those established in the field, if they are to stay there, must adopt these techniques and, more important, adapt them to pathological problems. The consequence of this trend obviously is more and more specialization within plant pathology. Already we see cults developing who refer to themselves as plant virologists, plant disease physiologists, chemotherapists, disease epidemiologists, plant nematologists, microbial geneticists, and I presume just around the corner, plant disease molecular biologists. This is all to the good because to make basic progress we must not only specialize, but also reach out to gain the advantage of mingling in allied fields. I am

not so much concerned that plant pathology will disappear like the exploding atom. There will always be plant disease problems and crop losses from disease. What I am concerned about is that these "specialty" groups will lose plant pathology. There is a real danger of being cut off in space without landing gear. We are already showing signs of building a tower of Babel within our science, wherein plant pathologists will not understand each other's lingo, to say nothing of their techniques and their philosophies. This must not happen.

This leads me to emphasize the fact that the time is ripe, if not overripe, to launch an *Annual Review of Phytopathology*. This is really the only way in which we can keep ourselves informed and in which specialists in one area can offer ideas, and inspiration to those in another. This means that there must always be a few of us taking time from busy research and teaching programs to evaluate and summarize the current advances in given areas within plant pathology. I know the reviewers will take their responsibilities seriously. I hope that they will always keep in mind that they are writing for all plant pathologists and not for the specialists in their chosen areas. After all, those specialists should not need such reviews if they are on the job. Let us hope that these reviews will be written not as annotated bibliographies but as sound interpretations in language attractive to those in other areas of plant pathology and to those just outside the field. Remember that it is not necessary to use the technique of a science fiction writer to make this journal attractive reading for the extension plant pathologist and many a county agent.

de Bary and his contemporaries started us off on a fabulous journey in 1863. We have nothing to be ashamed of. There are many stimulating challenges in the century ahead. Plant pathology will be in the forefront of biological sciences in 2063, and 100 volumes of the *Annual Review of Phytopathology* will bear witness to steady progress all along the way.

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GROWTH REGULATORS IN PLANT DISEASE[1,2,3]

By Luis Sequeira

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The study of the relationship of growth regulators to plant disease is not much more than twenty years old; and most of the work on this problem has been on disease resistance rather than on the basic aspects of the role of growth regulators in pathogenesis. The present review will be concerned very largely with the latter. We hope to integrate the recent progress in the field of pathogenesis.

Certain ancillary areas have been the subject of recent reviews. Braun (1) has covered extensively the entire problem of tumor inception in the crown gall disease. Gruen (2) has reviewed in considerable detail the production of auxins by fungi and the effect on auxins of fungal growth. Comprehensive reviews are also available on the formation and action of gibberellins (3) as well as on certain practical aspects of growth regulators in relation to disease control (4). Since no useful purpose would be served by re-reviewing these various aspects of the problem, the present article has the objectives of complementing these studies and, hopefully, of contributing to a more integrated view of the relations between growth regulators and pathogenesis. The term growth regulator or auxin is used in this review in the broadest sense, to include the wide variety of substances of both natural and synthetic origin that promote, inhibit, or modify plant growth.

GROWTH REGULATORS IN DISEASED PLANT TISSUES

Growth substances have been found in tissues infected with bacteria, fungi, viruses, and nematodes. Interest in auxin phenomena in bacterial diseases has centered almost exclusively in the crown gall disease incited by *Agrobacterium tumefaciens*. This has been treated very well recently by Braun (1).

Although a specific role for auxin has not been clearly demonstrated in most wilt diseases, tomato plants infected by *Pseudomonas solanacearum*, exhibit positive growth responses such as leaf epinasty and adventitious root formation. Grieve (8, 9) first investigated the possible role of IAA in this syndrome,

[1] The survey of literature pertaining to this review was concluded in December, 1962.

[2] The following abbreviations are used: GA, gibberellic acid (K salt); IAA, indole-3-acetic acid; IAE, indole-3-acetic acid ethyl ester; IAN, indole-3-acetonitrile; IBA, indole-3-butyric acid; ILA, indole-3-lactic acid; IPyA, indole-3-pyruvic acid; MH, maleic hydrazide; NAA, α-naphthalene-acetic acid; PLRV, potato leaf roll virus; SBMV, Southern bean mosaic virus; TIBA, 2,3,5-triiodobenzoic acid; TMV, tobacco mosaic virus; TSWV, tomato spotted wilt virus.

[3] Certain of the author's studies reported in this paper were supported in part by a research grant (GB 125) from the National Science Foundation.

but could not demonstrate increased auxin levels in diseased tomato tissue although the bacterium produced large amounts of IAA in culture. More recently, Sequeira & Kelman (6) have shown by means of chromatographic separation, increased levels of IAA, an additional unidentified auxin, and a growth inhibitor in infected tobacco and banana tissues. Increases in these substances were detected within four days after inoculation. In tobacco, a 100-fold increase in IAA (from 0.03 μg/100 g in healthy tissue to 3.3 μg/100 g in diseased tissue) was detected. IAA levels were highest in the stems, where maximum multiplication of the pathogen occurred. Since IAA was obtained in sufficient amounts to permit its identification by means of appropriate chemical and biological tests, as well as by means of absorption spectra, there is little doubt that hyperauxiny occurs even at relatively early stages of infection.

The nature of the second auxin has not been elucidated, although it was suggested that it could be IAN, on the basis of Rf and color reactions. Although the presence of this auxin in the acid fraction may be open to question, an active growth regulator of similar Rf and properties was also detected in the neutral fraction. The increase in IAA in diseased tissue was attributed mainly to the pathogen, based on: (*a*) the ability of the bacterium to produce copious amounts of IAA in culture, and (*b*) the fact that levels of IAA continued to increase even after the plants had completely wilted. The validity of such arguments may be questioned on the basis of reported increases in IAA during initial stages of infection, when populations of the bacterium were relatively small. In addition, the use of a dry weight rather than a fresh weight basis for determinations of IAA levels would have been desirable, particularly at advanced stages of infection.

Infection by pathogenic fungi quite often results in the development of exaggerated growth responses by the host. These may vary from the simple thickening or elongation of plant parts, which otherwise maintain their structural organization, to the highly disorganized growth that is characteristic of galls and tumors. Auxin relationships in the first of these responses have been studied almost exclusively among the rusts. For instance, safflower hypocotyls infected with *Puccinia carthami* show marked elongation before other outward symptoms appear, but this response stops when sporulation takes place. Analyses of ether extracts by combined paper chromatography and bioassay techniques have given clear evidence of hyperauxiny in diseased hypocotyls (10). When compared on the basis of individual hypocotyls, a tenfold difference in auxin content was noted. Chromatographic separation indicated that IAA was the main auxin present, although young diseased tissue contained additional growth promoting substances not present in healthy tissue. Both healthy and diseased tissues showed a peak in auxin content 13 days after planting but, at this stage, areas of strong growth inhibition could be detected only on chromatograms of ether extracts from diseased hypocotyls. Perhaps the strongest evidence, therefore, for a causal relationship

between elongation of safflower hypocotyls and synthesis of growth regulators is the fact that auxin content increased during the period of maximum growth of the hypocotyl and rapid mycelial growth of the pathogen, while growth stopped when growth inhibitors accumulated and sporulation took place.

It is unfortunate that inhibitor assays were not carried out after 13 days, when growth had essentially stopped, but considerable amounts of auxin could still be detected. While the evidence appears to be clearly indicative of auxin imbalance at all stages of infection, the rationale for estimating auxin levels on the basis of individual hypocotyls or on a fresh weight basis is not entirely clear. It was stated that diseased hypocotyls weighed approximately 50 per cent more than did healthy hypocotyls; thus, the use of dry weights for comparison purposes would have been more accurate, in spite of the fact that fewer hypocotyls would have been represented per unit dry weight in the diseased samples (rather than in the healthy samples, as indicated by the authors). In this, as in other disease situations, it is extremely difficult to ascertain the reasons for hyperauxiny. Although the synthetic capabilities of the parasite are unknown, it was suggested that increased synthesis, retardation of breakdown or accumulation of IAA could all be involved at different times during development of the parasite.

The common houseleek, *Sempervivum tectorum*, normally grows as a rosette, with short ovate leaves. When infected with the rust, *Endophyllum sempervivi*, however, leaves are about twice as long as the healthy leaves. Infected leaves are also chlorotic and much narrower at the base. When crude extracts of such leaves were tested for auxin activity by means of a sensitive root inhibition test, Pilet (11) found growth-promoting activity which was considerably higher than in healthy leaves. The highest concentration of auxin in the leaves (10^{-4}M IAA eq) appeared to be well correlated with the localization of the parasite. No attempt was made to identify the auxins involved and the quantitative aspects of this work may be questioned, since inhibitory substances are commonly present in such crude extracts. Thus, growth inhibition of roots, as used for auxin assay in this case, may well have resulted from direct inhibition by substances other than auxins. It would be desirable to re-examine this problem and to separate the growth substances by means of modern chromatographic techniques.

Pilet (12) has studied also the physiological basis for the exaggerated growth responses characteristic of cypress spurge infected by the rust *Uromyces pisi*. These responses are characterized by the extreme elongation and lack of branching of the stems, the formation of thick, shortened leaves which drop prematurely, and the atrophied nature of the flowers. An examination of the auxin levels in crude extracts of leaves showed that auxin (as IAA eq) increased in healthy tissues from 10^{-10} to 10^{-6}M and then dropped off after 40 days, while in diseased leaves the concentration increased to 10^{-4} M and did not drop off. Such estimates of auxin concentration on crude extracts

may be questioned as indicated in the case of rusted houseleek. However, Pilet (12) has given additional indirect evidence in support of presumed hyperauxiny in rusted spurge plants. When placed in a solution of K indoleacetate (10^{-5} M), highly parasitized plants became positively geotropic while lightly infected ones remained negatively geotropic. When exposed to one-sided lighting, lightly infected plants showed positive phototropism while severely infected plants showed positive phototropism first and then negative phototropism.

In both instances, the results may be interpreted as evidence for the presence of inhibitory levels of IAA and other auxins in severely infected plants. Although indirect evidence of this type does indicate deranged auxin metabolism, no evidence was presented to indicate that hyperauxiny alone resulted in the altered tropic responses. Other factors affecting auxin movement and degradation within diseased tissues could greatly influence responses to gravity and light. The fact that infected leaves abscissed prematurely would seem to be in conflict with the demonstration of relatively high auxin content in the leaf tissues, since in most instances the maintenance of high auxin levels has been shown to delay leaf abscission (13).

To overcome certain of these objections, Pilet (14) has re-examined the auxin picture in rusted spurge by a combination of ether extraction of large amounts of tissue (app. 8,000 g), separation of auxins by paper chromatography, and bioassay by two methods: root inhibition and stem elongation of *Lens*. The results indicate quite clearly that diseased stems contain IAA, two unknown "accelerators," and an unidentified growth inhibitor, in amounts considerably greater than healthy stems. These substances increased as the disease progressed and at the aeciospore stage, the ratio of auxin in diseased/healthy was 39.7/7.8 μg IAA equivalents per 1,000 g of tissue. Auxin analysis of infected leaves (15) showed increases of approximately the same magnitude. Although the use of dry weight as a basis for comparison would have been desirable, Pilet's extensive investigations on *Euphorbia* leave little doubt that hyperauxiny is an important factor in pathogenesis by *Uromyces*.

Results obtained with other rusts causing hypertrophic responses give further evidence of hyperauxiny. Hirata (16) used diffusates from pieces of tissue or expressed juice from various rusts and estimated the auxin content by means of the *Avena* coleoptile curvature test. He reported 1.6-,5.0-, and 8.4-fold increases in auxin in aecial stages of *Gymnosporangium* on *Pyrus*, *Puccinia* on *Smilax*, and *Uromyces* on *Ranunculus*, respectively. The identity of the auxins involved is not known.

Other rust diseases, such as stem rust of wheat, cause no overt hypertrophic responses, but involve increases in auxin content similar to those previously described. Using the *Avena* straight growth technique, Shaw & Hawkins (17) found that at 10 days after inoculation, the acid fraction of alcohol extracts from rust-infected leaves of the highly susceptible wheat variety Little Club showed a 24-fold increase in IAA. Since reasonably large

samples of tissue were extracted, the evidence appears to be largely in favor of IAA as the major auxin in rusted wheat tissues. The evidence is not as convincing in other disease situations involving obligate parasites of cereals. In powdery mildew-infected leaves of Atlas barley, Shaw & Hawkins (17) obtained evidence in one test of a 5-fold increase in IAA in leaves bearing well developed lesions. In other tests, however, differences in IAA content between healthy and mildewed were small and probably of no significance. In barley mildew, at least, it would be desirable to re-examine the auxin picture before conclusions regarding the physiological effects of hyperauxiny can be drawn. However, it may be significant that in wheat and barley, unlike corn and most dicotyledonous plants, IAA causes no marked hyperplastic or hypertrophic responses. Therefore, from the morphological aspect as well as from other points of view, the data on IAA in rusted and mildewed cereal leaves would appear to be consistent.

The marked overgrowths that are characteristic of most smut infections have aroused the interest of plant pathologists for many years and several attempts to determine the physiological basis for tumor formation have been made. In the case of corn smut, it was shown by Link, et al. (18) that ether extracts of cultures of *Ustilago zeae* contained substances that gave a positive Salkowski reaction, and suggested that IAA could be a constituent of the extracts. These extracts were active on the *Avena* coleoptile test (19, 20) and later, IAA was shown to be one of the primary constituents (21). Tumors from corn leaves and stems yielded more substances active in the curvature test than did healthy leaves and stems (20, 22). On the other hand, Turian (23) was initially unable to demonstrate the presence of IAA in chromatograms of extracts from small amounts of tissue, and he doubted that this auxin could be implicated in the development of the galls. Using more modern, elaborate extraction techniques and considerably greater amounts of tissue, Turian & Hamilton (24) were able to isolate and characterize IAA in tumorous tissues. When corrected for losses incurred during extraction, young tumors were found to contain 5.0×10^{-7} M IAA in contrast to normal stalk tissues which contained 2.4×10^{-8} M IAA. These results were in contrast with Moulton's original results in which he indicated a curvature response equivalent to 15–30μg of IAA/g, or approximately 2×10^{-4} M (20). Such large discrepancies may be the result of additional auxins present in Moulton's crude extracts (25), but may also reflect differences in efficiency of extraction. Daly & Inman (10) have given clear evidence that extraction of auxins is less efficient as the amount of plant tissue increases. Also, Moulton's procedure involved long-term extractions of small amounts of tissue, which presumably resulted in recovery of free as well as bound auxin.

Systemic invasion of crucifers by the white rust fungus, *Albugo candida*, results in hypertrophy and hyperplasia of various organs. Abnormal growth is particularly conspicuous in the flower parts, where the sepals may become enlarged to several times their normal size. Efforts to determine the basis for

this abnormal growth have not resulted in a clear picture as to the nature of the auxins involved. Hirata (16) tested the activity of expressed juice from infected rape tissue on *Avena* curvature and concluded that high levels of IAA were responsible for the abnormal growth. However, IAA was not specifically determined in his tests.

In plants of *Capsella bursa-pastoris*, naturally infected by both *Albugo candida* and *Peronospora parasitica*, Kiermeyer (26) detected both IAA and IAN. He used a combination of chromatography and bioassay of extracts prepared from plant tops. Both healthy and diseased plants contained high amounts of IAN, as is characteristic of most crucifers. On the other hand, only chromatograms of extracts from diseased tissues contained IAA in sufficient amounts to give a color reaction with Ehrlich's reagent. Thus, it was concluded that IAA accumulation was responsible for the hypertrophied growth. Kiermeyer further substantiated these findings by demonstrating that the application of 1 per cent IAA in lanolin to *Capsella* stems resulted in bending and other symptoms characteristic of white rust. However, in a recent publication, Srivastava et al. (27) reported that *Albugo*-infected rape inflorescences contained lower amounts of both IAA and IAN than comparable healthy tissues. Two additional, unidentified auxins presented a similar picture. Since both IAA and IAN were identified by a series of chemical and biological tests, there is little reason to doubt that changes in levels of these auxins may be of importance in pathogenesis.

The quantitative aspects of this work, on the other hand, can be seriously questioned. The use of fresh weights as a basis for comparison may have led, as in so many other instances, to discrepancies in estimates of auxin content, since differences in hydration of tissues can easily result in erroneous interpretations. It was indicated that differences in auxin content could arise from the fact that Kiermeyer included stem and leaf tissues in his extractions, while the Canadian workers used only inflorescences (27). It is clear, however, that other factors, as indicated above, may be of greater importance. In addition, the presence of both *Peronospora* and *Albugo* in the tissues used by Kiermeyer undoubtedly resulted in complications which are difficult to assess.

The best example of a specific role for auxin-like substances in plant diseases is the well-known "bakanae" or foolish seedling disease of rice caused by *Gibberella fujikuroi* (*Fusarium moniliforme*). Affected rice plants sometimes grow quite rapidly and become conspicuous because of their size and etiolated appearance. The discovery by Kurosawa (29) that cell-free culture filtrates of the fungus could reproduce the bakanae effect led to the eventual discovery of gibberellins and to the study of their role as natural growth regulators.

The similarity of effects on plant growth caused by virus infections and those obtained by the application of growth substances has long been noted. This similarity has led to the study of the auxin content of virus-infected tissues. With few exceptions, there is general agreement that growth stunting

characteristic of many virus infections is correlated with a lowered auxin content. In an early study of the potato degeneration problem caused mainly by potato leaf roll virus, Jahnel (31) noted that virus-diseased tubers contained up to 53% less auxin than healthy tubers. Auxins were detected by the *Avena* coleoptile curvature test on simple diffusates from tuber slices.

Jahnel concluded that auxin deficiency was responsible for the tendency of infected tubers to germinate by means of numerous long, thin shoots, distributed at random over the tuber. These findings were confirmed and expanded by Lucas (32), and by Söding & Funke (33), who showed a marked decline in auxin content in virus-infected plants, particularly in the stem tips and young foliage. Petioles of diseased plants responded less actively than those from healthy plants to dilute concentrations of IAA, thus indicating a lower endogenous content of auxin in the diseased tissues.

Later, Baumeister (34) reported that virus-affected tissues not only have a lower auxin content but a higher content of at least two growth inhibitors as well. The nature and possible role of these inhibitors in the leaf-roll virus syndrome have remained undetermined, but it is possible that these and other substances affecting the response of host cells to auxins may be of far greater importance than auxins per se. For instance, increases in fluorescence of potato leaves and tubers infected by PLRV have been shown to be due largely to the accumulation of scopoletin (35). Although the exact role of SC in auxin metabolism is not entirely clear, this substance competitively inhibits enzymatic destruction of IAA in vitro, induces photooxidation of IAA under other conditions (36), inhibits the elongation of *Avena* roots (37), and is toxic to tomato plants when supplied in high concentrations (6). It is difficult, therefore, to determine the specific role or roles of SC in the leaf-roll syndrome, but its presence in considerable amounts during early stages of infection undoubtedly affects both auxin content and the response of host cells to auxin.

Tomato plants infected with the spotted wilt virus either stop growing completely or grow at a much lower rate after the appearance of symptoms, suggesting interference with the hormonal control of the host. Grieve (38) has presented evidence for such a mechanism based on three different approaches: (*a*) less auxin was present in ether extracts of diseased plants, (*b*) fewer adventitious roots developed in diseased than in healthy plants treated with IAA, and (*c*) less auxin was transported through a diseased stem section than through a healthy section. Grieve reasoned that the stunting symptoms produced by virus infection result both from the reduction in auxin content in the leaves and from increased inactivation of auxin during translocation. It is apparent, however, that differences in auxin transport could have resulted from phloem degeneration, which typically accompanies infection by tomato spotted wilt virus (TSWV). In addition, a re-examination of the auxins present in the expressed juice from healthy and TSWV-infected tomato plants failed to detect any significant lowering of auxins in the diseased tissues (39). In fact, higher levels of IAA were detected in virus-infected plants in some instances. Although these results may be questioned