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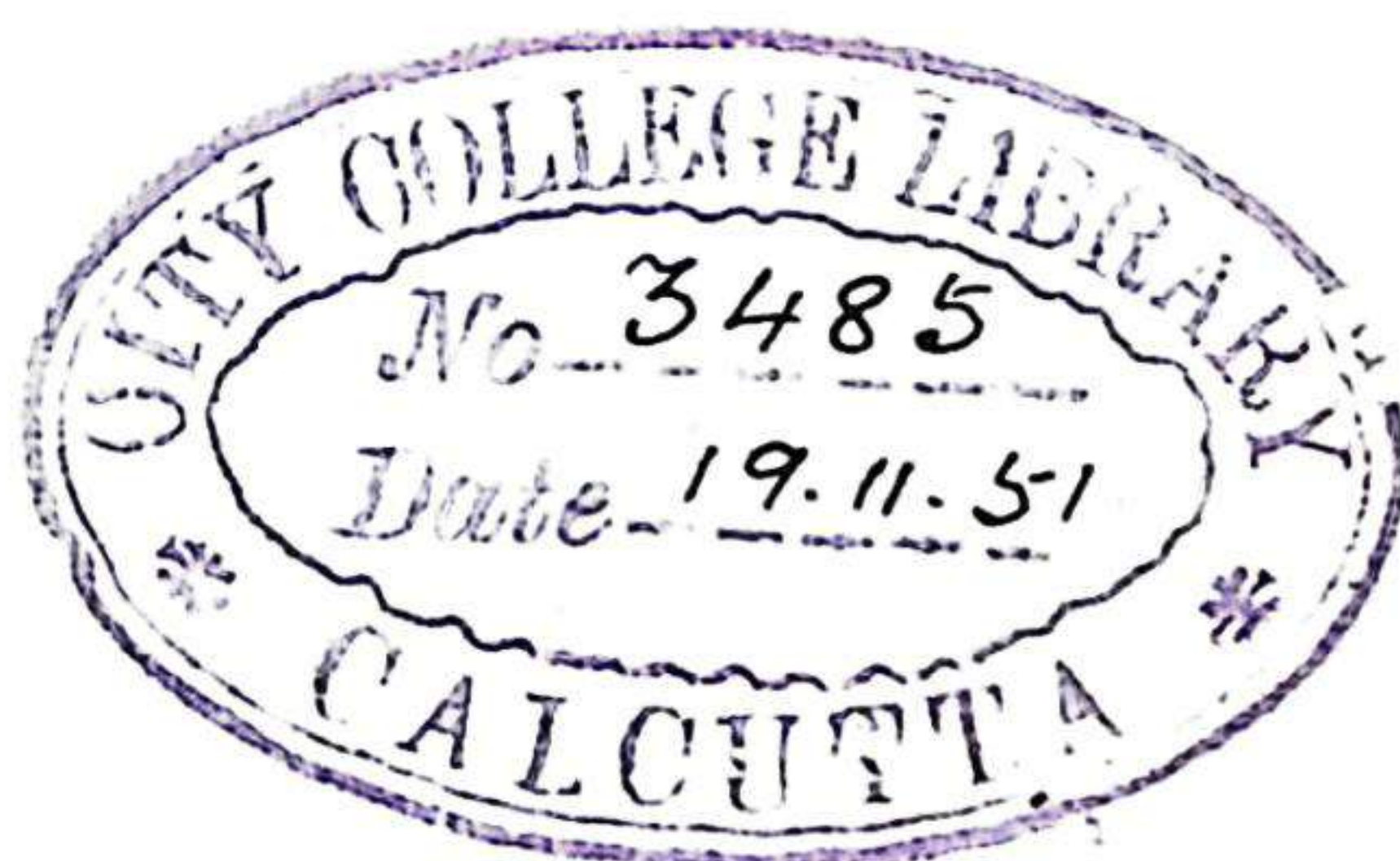
PLANT PATHOLOGY

BY

JOHN CHARLES WALKER

*Professor of Plant Pathology
University of Wisconsin*

FIRST EDITION



NEW YORK TORONTO LONDON
McGRAW-HILL BOOK COMPANY, INC.

1950
PROVAT BOOK COMPANY.

Book Seller & Publisher.
52/B, Russa Road.

PLANT PATHOLOGY

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To My Wife

EDNA DIXON WALKER

PREFACE

The purpose of this text is to provide the basic facts whereby the student may be introduced to the scientific foundations of plant pathology. It is not a manual of disease descriptions nor a handy compendium of remedial measures. It presents rather comprehensive treatments of better known representative diseases which may serve as guides to the general principles underlying the science of plant pathology. Since the background of any science is basic to a full understanding of its present trends, a chapter on the history of plant pathology is included. Following the chapters on specific disease groups, consideration is given to environmental factors, host-parasite relations, and the methods of disease control.

In the preparation of this book the writer has profited greatly by the help and interest of his colleagues. Among those who have given their advice on various chapters and furnished illustrative materials are Deane Arny, M. P. Backus, J. G. Dickson, D. J. Hagedorn, P. E. Hoppe, James Johnson, G. W. Keitt, R. H. Larson, J. D. Moore, G. S. Pound, A. J. Riker, and M. A. Stahmann. Others who have provided photographs are Eubanks Carsner, John C. Dunegan, Donald Folsom, M. W. Gardner, G. F. Gravatt, R. E. Hartman, L. M. Hutchins, G. B. Ramsey, and C. Audrey Richards. The drawings were all copied from the original by Barbara Blumenfeld. I am deeply indebted to Eugene Herrling, who made many of the original photographs and carried out all of the final preparations of the illustrative material.

JOHN CHARLES WALKER

MADISON, WIS.
October, 1950

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CHAPTER 1

INTRODUCTION

Plant production is essential to the maintenance of life of man and animals upon this planet. Food, raiment, many luxuries, and some essential drugs are dependent directly or indirectly upon growing plants. Continuous change in crop requirements is brought about by shifts in population, in trends in consumer preference, and in relative costs of land, labor, and equipment. American agriculture is, indeed, dynamic and is increasingly dependent upon progress in underlying sciences.

Plant pathology is concerned with the health and productivity of growing plants. Disease losses are hazards which can be minimized only by a continuous process of research and education. Each change in variety of crop, each turn in the climatic cycle, and each regional shift in the growing of a crop brings new problems of health and disease. It is the responsibility of plant pathology to evaluate and solve new disease problems, to train research investigators and extension specialists, to brief agricultural educators and county agents in applied phases of the science, and to work out practical procedures which growers can adapt to their needs.

DEFINITION OF DISEASE IN PLANTS

For any variety or species of plant there is a set of environmental conditions which favors its optimum development. In nature, however, such an ideal situation seldom exists, and in consequence every variety of plant is subjected to the vicissitudes of the environment. When one or more factors in the environment become particularly unfavorable, the development of the plant is altered in some way, and in comparison with plants growing in the usual range of environment, it appears to have abnormal characteristics. Diseased plants are distinguished by changes in their structure or physiological processes which are brought about by unfavorable environment or by one or another parasitic agency. There is no clearly defined line of distinction between **normal** or **healthy plants** and **abnormal** or **diseased plants**. **Plant disease**, therefore, is, like many biological phenomena, difficult to circumscribe or define. We may, however, think of diseased plants as those which have become altered in their physiological and their morphological development to such a degree that signs of such effects are obvious. These external signs on the plant which are characteristic of a given disease are known as **symptoms**. Whether all deviations from the normal in structure and/or function constitute disease is largely a matter of definition and depends

in any case upon what one chooses to define as normal for the particular variety or species in question.

The development of man's concept of plant disease during the past 20 centuries is told in the next chapter on History of Plant Pathology. Recognition of abnormalities in plants goes back to the early historical records. In general they were attributed to the acts of supernatural beings, to vicissitudes of weather, to insects, or to parasitic seed plants. Only since the middle of the nineteenth century have parasitic microorganisms been accepted as causal agencies in plant disease. In the last two decades of the nineteenth century viruses were recognized as infectious causal agencies, and only since 1935 has considerable evidence been brought forward to show that viruses are nonliving but infectious proteins.

Since we now recognize that disease in plants may be brought about by unfavorable environment, or by infectious agencies which may or may not be living, it is well that we define at the outset certain terms which will be used repeatedly in this book.

DEFINITIONS AND TERMS

First of all it is well for the student to have a clear concept as to what constitutes the **cause of disease**. This is particularly true because the term is used rather loosely in much of the literature on plant pathology. It is relatively simple to conceive of one or more, or a combination of several, environmental factors becoming the cause of a disease when they alter the physiological processes and morphological development of the plant concerned. When a parasitic microorganism enters into the complex, as is the case in a large percentage of plant diseases, there is a common tendency to refer to the microorganism as the cause of the disease. Since we now know that variation in the environment is sometimes necessary to predispose the plant, *i.e.*, cause it to become susceptible to the microorganism, one can readily see that the microorganism is not the sole causal factor. Moreover we also recognize that the microorganism, also being a biological entity, is subject to the influence of environment, and it often, if not always, requires a certain range of favorable environment to produce infective organs and for such organs to infect the plant. After a microorganism has successfully infected a susceptible plant, the subsequent interaction of plant and microorganism which results in a disease of the former is also subject to environal influences which determine whether disease develops at all and, if so, at what rate and to what degree. It is evident that in the case of a disease in which a bacterium or fungus is concerned there are various other factors besides the latter which enter into the causal complex. Strictly speaking, therefore, it is not correct to refer to a given microorganism as the cause of a given disease, since this

implies that it is the sole cause. However, such usage is common in current literature on the subject. It has been suggested that the organism be referred to as the **incitant**, implying that it incites the disease under the influence of other factors. The term **causal organism** is also proper since it implies that the organism is a part of the causal complex.

Most causal organisms are parasites. In current usage a **parasite**, as applied to an organism, is one which subsists in whole or in part upon living tissue. Conversely, a **saprophyte** is an organism which subsists upon dead organic matter and inorganic materials. A **pathogen** is an agency which incites disease. While the term might be applied to any or all of the causal factors described above, it is practically always used to denote the living entity in the causal complex. The terms pathogen and parasite are not synonymous, however. In some cases organisms are parasitic without becoming causal factors in disease. In other cases the by-products of a strict saprophyte are an important part of the causal complex, and thus a saprophyte may indirectly become a pathogen. **Pathogenicity** is the property of a microorganism whereby it may become a part of the causal complex. **Pathogenesis** is the process or chain of events whereby disease development takes place.

A not uncommon habit among plant pathologists is to refer to the disease concerned by the scientific name of the causal organism. This is, of course, strictly incorrect. The student of plant pathology should at the beginning develop a habit of referring to the disease and to the causal organism as distinct entities.

Diseases of economic plants have usually acquired one or more **common names**. As a rule such names are descriptive of some important phase of the disease, *e.g.*, late blight of potato; wilt of tomato; black stem rust of wheat; clubroot of cabbage. Sometimes the generic name of the causal organism is used as a descriptive adjective in the common name, either because a sufficiently descriptive common word is not available or because it is necessary to distinguish between similar appearing diseases with different causal organisms. Thus wilt of tomato may be the result of the action of distinct causal organisms; hence, *Fusarium* wilt of tomato, *Verticillium* wilt of tomato, bacterial wilt of tomato. The *Rhizoctonia* disease of potato is another example of a case where the genus of the parasitic phase of the causal organism is used in the common name of the disease. This organism has a wide range of pathogenetic effects upon the potato plant. Under certain conditions basal stem cankers are formed, the result of which may be upward rolling of leaves, abnormal purpling of the foliage, deposition of starch in axillary buds of the stem to form aerial tubers. Quite apart from the disease on aerial parts of the plant, there are formed superficial black resting bodies (**sclerotia**) on the surface of

the potato tuber. Because the last symptom is a very obvious one, the disease is often referred to as black scurf. However, since the last term describes only one phase of a wide range of distinct disease reactions, the first term mentioned, *Rhizoctonia* disease, is also used.

The causal organism, if fully described, has been given a specific name within a given genus according to the accepted system of plant nomenclature. *Since the generic name and specific name are Latin words always printed in italics, they should always be underlined in any handwritten or typed manuscript.* This name of the organism is referred to as the **Latin binomial** of the organism. The species as originally described may be later transferred to another genus by the same or another investigator. In such a case the species name is retained unless it has been declared invalid for some legitimate reason. The name, or an abbreviation thereof, of the investigator who first described the species follows the binomial. If the species is referred by another investigator to another genus, unless the transfer is from a form genus to that of a perfect form, the name or abbreviation of the one making the original description is retained in parentheses immediately after the binomial, while the name or abbreviation of the one responsible for the current binomial follows.

Since there is not always agreement as to the proper genus to which a species belongs or as to whom the first authentic description of a species is properly attributed, there may be found more than one binomial for a given organism in current literature. On this account it is customary in a paper dealing with a given disease to give the complete binomial with the proper authority when the organism is first mentioned. This indicates clearly to the reader which authority the writer chooses to follow. Subsequently in the paper the authority may be omitted, and some writers prefer to make subsequent reference to the causal organism by using the common name of the disease as a descriptive phrase modifying "organism," "fungus," "bacterium," etc., e.g., the crown-gall organism, the potato-late-blight organism, the potato-late-blight fungus, the fire-blight bacterium.

In certain cases usage has resulted in exceptions to the general rule, in that the same word is applied to the disease and to the causal organism. Most common examples are found in the rusts. The large group of diseases known as rusts acquired this name long before organisms were recognized as causal agencies, and the term came into use because it described a prominent characteristic of the diseases as a group. Likewise the organisms were known, and many were described and named, long before they were recognized as pathogenic agencies. Because of their characteristic color and the consistency of the spore mass, this group of organisms was also referred to as rusts. Thus the same word came into use for disease and

fore
for pathogen long before their relations were fully understood. The same chronological history is true for the smuts. It is the more important, therefore, to connote whether the term is being used to apply to the disease or to the causal organism.

In the case of diseases in which parasitic organisms are a part of the causal complex the diseased plant is commonly referred to as the **host**. While usage has made this the most widely used term, objection has been raised that it does not imply the actual relation which ordinarily exists between the plant and the pathogen. The term **suscept** is a newer word coined to apply to the plant which is subject to a given disease brought about by a given causal complex.

The above terminology does not always strictly apply in the case of virus diseases. While the inanimate nature of viruses is not accepted in all quarters and practically nothing is known about their mode of increase or reproduction, they have above all in common with pathogenic micro-organisms the property of being transmitted from plant to plant to become a part of the causal complex. While several schemes of nomenclature for plant viruses have been proposed, none has been fully accepted as yet. Therefore in current literature the student will find a great diversity among authors in method of reference to a given virus. They are not universally referred to as pathogens, and their disease-producing property is not commonly referred to as pathogenicity, largely because of the doubt that they are living entities. One system known as the Holmes system sets up a scheme of genera and species whereby each described virus is given a binomial. While this system has many admirable features, it is nonconformist in that it uses a system set up for living organisms to classify what may well be nonliving organic compounds, which cannot be defined on the bases used in the binomial system for living organisms. An earlier suggestion by Johnson is to give each virus described from a given suscept or host consecutive numbers, *e.g.*, potato virus 1, potato virus 2, cucumber virus 1, etc. A modification of the Johnson system, proposed by Smith, substitutes the generic name of the host for the common English name, *e.g.*, *Solanum* virus 1, *Solanum* virus 2, *Cucumis* virus 1, etc. Some writers follow one, some another, of these systems, while still others avoid all three systems and refer to the virus concerned by means of the disease with which it is associated, *e.g.*, common-tobacco-mosaic virus, potato-ring-spot virus, tobacco-ring-spot virus, spotted-wilt virus, potato-witches'-broom virus, etc.

When a pathogen is a part of the causal complex, the organism is first present in the environment in some overwintering (or oversummering) form. The overwintering stage may be in or on seed of the host, in perennial wild hosts, in debris of infected plants of the previous season, as

dormant spores, sclerotia, or other organs in the soil, in the form of an inhabitant of the soil flora, and in various other forms. Whatever this source, it becomes the **source of primary inoculum**. It may serve as the infective agent and thus become the **primary inoculum**, or it may multiply as a saprophyte (or as a parasite on a perennial host) to produce spores which become the primary inoculum. When the host becomes diseased, the organism commonly produces another crop of spores or infective bodies, which are known collectively as the **secondary inoculum** and serve to cause **secondary infection**. This last cycle may then be repeated several times during the current season.

The first step in the relation of the inoculum to the host consists of penetration. This may take place in one of a number of ways which are characteristic of the organism and are influenced sometimes by the environment. The various methods of penetration will be discussed in later chapters. It is important to recognize here that there is a distinction between penetration and infection. **Penetration** refers to initial invasion of the host by an organism. We know that many organisms penetrate cells of plants which are not susceptible and which do not respond with any sign of disease; moreover they die without proceeding beyond the stage of penetration. Thus we may have many cases of penetration without infection. **Infection** implies the establishment of the pathogen within the host following penetration. *It does not imply the production of disease symptoms.* There may be a long period between infection and appearance of disease, but ordinarily one follows rather closely upon the other. The interval is known as the **incubation period**. Just as penetration and infection are to be regarded as distinct, so infection and disease development are distinct. **Disease development** refers to the chain of events which occur between the time of infection and the complete expression of disease. In general, disease development is a protracted process which involves production of secondary inoculum and progression of external evidences of disturbance of the morphological and physiological processes of the host plant; these external evidences are referred to as **symptoms**. The chain of events in disease development is referred to as the **disease cycle**. While, when a pathogen is involved, the disease cycle is intimately associated with the organism, it is distinct from the **life cycle of the organism**. In the case of many causal organisms only part of the life cycle is passed in association with the host, the remainder being in association with dead host tissue, independently as a saprophyte, in association with an alternate host, or in association with an insect vector.

An **infectious organism** is one which may be transmitted from a diseased host plant to a healthy plant and which is capable of inciting the disease under favorable environment. An **infectious disease** is one in

which a transmissible causal organism is a part of the causal complex. Virus diseases are also usually referred to as infectious since the inciting agency can be transmitted from a diseased to a healthy plant and initiate the disease in the latter. In the light of present knowledge it is not proper to group them with infectious organisms, since there is no evidence that they reproduce themselves, whereas it is entirely possible if not probable that the infective entity acts as a catalyst which stimulates the formation of virus protein instead of normal host protein during the period of disease development.

Various **levels of parasitism** among microorganisms have long been recognized. In many cases two living organisms may live in association with one another, and in a sense each may be parasitic upon the other. However, they are not only nonpathogenic to one another but also are each essential to the other's development or at least each is beneficial to the other's development. This phenomenon is known as **symbiosis**, and the participating organisms are known as **symbionts**. Lichens are classic examples of symbiosis: a fungus and an alga living in close association with one another. The alga through its photosynthetic property converts kinetic energy of the sun's rays into potential energy of carbohydrates useful to its own development and essential to the life of the fungus. The latter through its property of producing hydrolytic enzymes breaks down materials in the substrate, making energy available and providing mineral and organic nutrients essential to itself and the alga. Another case of symbiosis is that of the nodule-forming bacteria in relation to their respective leguminous hosts. Here the bacterium penetrates and infects the root hair; the host responds by abnormal proliferation of cells to cause a gall, or root nodule. Up to this point the process might be considered pathogenic. However, the bacteria are capable of fixing the free nitrogen of the air and converting it into the essential nitrogenous compounds in their cells. Presumably, as the bacterial cells disintegrate, the nitrogenous compounds become available to the legume plant. Since higher plants are generally incapable of utilizing free nitrogen, an element essential to protein formation, this symbiotic phenomenon is a great economic source of available nitrogen. That symbiosis is a phase of parasitism and is very close to pathogenesis is shown by further study of the legume-bacterium phenomenon. All strains of the bacterium adapted to a given legume host are not equally efficient in nitrogen assimilation. In fact, some strains show little or no evidence of such a property. When such a strain is allowed to infect its legume host, nodules develop as usual, but in the absence of nitrogen assimilation the usual benefits to the host are lacking, and the latter shows certain disease symptoms when compared with uninoculated plants. Thus the legume bacterium is in this case not

a symbiont but a pathogen, and the parasitism involved results in pathogenesis rather than symbiosis.

Contiguous to parasitism but distinct from it is the phenomenon referred to herein as **epiphytism**. An **epiphyte** is a plant growing upon another plant, and, as the term is usually applied, it refers to cases where no parasitic or truly symbiotic relation is involved. Particularly in tropical regions, many higher plants grow on shrubs or trees, not in association with soil. They secure essential mineral nutrients from the dead bark tissue of the substrate plant but do not penetrate the living tissue of the latter and, apparently, have no beneficial or detrimental effect upon the latter. While some parasitic microorganisms are largely epiphytic, they are not considered as members of this group. Opposed to epiphytes are **endophytes**, which are contained entirely within the substrate plant. Such organisms may be either parasitic or symbiotic.

Having delineated symbiosis and epiphytism in relation to **parasitism**, we may now consider various grades of the last. After the relation of microorganisms to plant disease became fully recognized shortly after the middle of the nineteenth century, it soon became apparent that pathogenic organisms vary widely in the manner in which they bring about pathogenesis under the influence of favorable environment. Parasitism is a very complex phenomenon which is basic to the understanding of plant disease. Its various phases will be referred to repeatedly in subsequent chapters. Here it is essential only to explain the important terms used throughout the book. Some pathogens, so far as is shown, cannot complete their life cycles except in a parasitic relation to their respective hosts. They may persist as dormant spores away from the host, and such organs may germinate and produce secondary spores away from the host. Thus a part of the life cycle may proceed apart from the parasitic relation. Infection must occur, however, and parasitism obtain for the completion of the life cycle. Such organisms are known as **obligate parasites**, and they are found in various groups of the fungi; none are known among the bacteria. Viruses have properties in common with obligately parasitic organisms in that they increase in amount only in association with living susceptibles. Common examples of obligate parasites are the rust fungi, the powdery-mildew fungi, the downy-mildew fungi, the crucifer-clubroot organism, the potato-black-wart fungus. **Obligate saprophytes** are those microorganisms which have no relation with living cells and secure their nutrients from dead organic tissue or from available inorganic materials. A large share of the pathogens, however, have parasitic and saprophytic phases of their life cycles. The fact that an organism is in part a saprophyte does not in any sense detract, necessarily, from its potentiality as a pathogen. Many organisms can live for indefinite periods

as saprophytes in pure culture or otherwise and still remain highly virulent pathogens. There are, however, many organisms which are in the main saprophytes but have weakly pathogenic properties which are ordinarily expressed only when particularly unfavorable conditions have predisposed the host in such a way that it is unusually susceptible and when such environment is particularly favorable to the expression of the weakly pathogenic properties of the organism. Such organisms are called **facultative parasites**, which term implies that they are ordinarily saprophytic but under certain conditions may become parasitic. Many damping-off organisms are typical facultative parasites. At the other end of the wide range of parasites that are not obligate parasites in the strictest sense are the **facultative saprophytes**. These organisms ordinarily have life cycles similar to the obligate parasites, in that they pass most of the cycle in association with the host and do not subsist as saprophytes. They may, however, subsist as saprophytes for part of their life cycle but do not complete their life cycle away from the host. Many of the smut fungi answer the requirements of this definition of a facultative saprophyte. It should be borne in mind, however, that there are no well-defined limits between facultative saprophytes and facultative parasites. It should be emphasized that they represent the extremes in the range of microorganisms which may have parasitic and saprophytic phases in their life cycles and *particularly that, as defined, the facultative saprophyte has a much higher potentiality as a pathogen than does the facultative parasite.*

Predisposition may be defined as the effect of one or more environal factors which makes a plant vulnerable to attack by a pathogen. It is a process which antedates penetration and infection. **Susceptibility** refers to a condition of a plant in which it is normally subject to attack by a given pathogen. The susceptibility of a given individual, variety, or species may be increased or decreased by environal factors. It may also be higher or lower as between individuals, varieties, or species because of differences in the inherited characteristics which affect susceptibility. **Resistance** may be regarded as the counterpart of susceptibility and also may be influenced in its expression by environal factors. ✓

CLASSIFICATIONS OF PLANT DISEASES

Plant diseases may be grouped in various ways. The method of classification depends to a considerable extent upon the purpose of the treatment. In the chapter on History of Plant Pathology it will be seen that before the acceptance of microorganisms as causative agencies in plant disease (about 1850), many writers speculated more or less on possible causal factors, but they based their schemes of classification upon **effect** rather than upon **cause**. After 1850 books upon plant diseases grouped

them according to causal factors rather than according to effects or symptoms. Classification on the basis of cause has many advantages in a general text, one of the chief of which is that the most satisfactory approach to a basic understanding of disease is through an unraveling of the various causal factors and their interrelations, as well as their interaction with the host. For this reason such a scheme is used in this book.

In a manual of diseases of a certain group of plants, the consideration of or classification of diseases according to the host plant has decided advantages, and such a scheme is often used, *e.g.*, cereal diseases, truck-crop diseases, corn diseases, flax diseases, etc. In other treatises diseases are advantageously treated according to symptoms or effects, *e.g.*, root rots, powdery mildews, wilts, leaf blights, overgrowths, or hyperplastic diseases, hypoplastic diseases, necrotic diseases, etc.

Parasitic and virus diseases are often classified, in relation to their occurrence, into three groups: **endemic**, **epidemic**, or **epiphytotic**, and **sporadic**.

Endemic Diseases. When a disease is more or less constantly present from year to year in a moderate to severe form, it is classed as endemic. This is usually based on the fact that the pathogen or virus is well established and long persistent through its ability to survive from one crop season to the next in soil, on crop refuse, or in wild hosts. It is also implied that the environal conditions are usually favorable for inoculum to develop, infection to take place, and disease development to ensue. Diseases due to pathogens which persist for long periods in the soil, such as those of cabbage yellows and onion smut, are commonly endemic. When the infectious agent readily survives in prevalent wild hosts, as is the case with cucumber mosaic in milkweed or pokeweed plants, the disease is also usually endemic.

Epidemic, or Epiphytotic, Diseases. An epidemic disease is one which commonly occurs widely but periodically. The term "epidemic" is derived from a Greek word meaning "among the people" and in the strictest sense applies to diseases of man. The term "epiphytotic" has been coined to carry the same connotation with reference to plants. Epidemic diseases are often very responsive to variations in environment. The pathogen may be more or less constantly present, as in endemic diseases, but the environment favorable to development occurs only periodically. It is thus evident that the environment in such a case may well be the major determining causal factor. Epidemic diseases may also occur where the environment is usually favorable but where the pathogen is irregular in its occurrence. This may be because of the inability of the organism to survive always from one crop to the next. It may be dependent upon seed or other propagative parts of the host for

introduction. Insect vectors may be necessary for introduction or spread. Epidemics of black rot of crucifers are commonly dependent upon introduction of the pathogen with the seed. Late blight of potato is commonly epidemic in nature because of its sensitivity to fluctuation in temperature and humidity. Curly top, a virus disease of sugar beet in the Rocky Mountain states, is dependent upon the introduction of the virus by leaf hoppers which overwinter in more or less distant areas. If the winter reduces greatly the overwintering insect population, infection and spread are minor; if the conditions are the reverse, early and continued infection results in severe disease development.

Sporadic Diseases. (Sporadic diseases in reality belong to the epidemic group.) The term is applied to those which occur at very irregular intervals and locations and in relatively few instances.)

It should be borne in mind that a given disease may be endemic in one region and epidemic in another. Late blight of potato is endemic in certain parts of northern Europe because the environment is practically always favorable at some time during the season. It is epidemic in the upper Mississippi valley and on the winter crop in the Gulf states, because some seasons are entirely unfavorable for its initiation and development.

DISEASES CLASSIFIED ACCORDING TO MAJOR CAUSAL AGENTS

There are two methods by which major disease classes may be set up according to cause. By one method, diseases are divided into two classes: infectious and noninfectious. In this case all diseases incited by microorganisms, parasitic phanerogams, nematodes, and viruses are regarded as infectious. However, it may be questioned whether certain of the insect-incited diseases are infectious, although they are parasitic. By the second method, diseases are divided into nonparasitic and parasitic classes. In this classification some authorities would place virus diseases in one class, and some would place them in the other. Thus these, like most or all methods of classification of biological entities, have obvious shortcomings at the outset. For purposes of utility, simplicity, and reasonable accuracy, the writer uses the latter scheme with the exception that the virus diseases are placed in a third distinct class.

I. Nonparasitic diseases.

Diseases incited primarily by

1. Low temperatures.
2. High temperatures.
3. Unfavorable oxygen relations.
4. Unfavorable soil-moisture relations.
5. Injurious atmospheric impurities.
6. Lightning injury.

7. Mineral excesses.
8. Mineral deficiencies.

II. Parasitic diseases.

Diseases incited by

1. Bacteria.
2. Plasmodiophorales.
3. Phycomycetes.
4. Fungi Imperfecti.
5. Ascomycetes.
6. Basidiomycetes.
7. Phanerogams.
8. Animal parasites.
 - a. Nematodes.
 - b. Insects.

III. Virus diseases.

In the United States injuries brought about by insects belong in the field of economic entomology. This does not mean that the effects upon the hosts are not regarded as disease effects. Such a division of the field is an arbitrary one and is entirely justifiable for numerous reasons.

ECONOMIC SIGNIFICANCE OF PLANT DISEASES

It is not uncommon to find, in a relatively small locality, that the loss within a given crop in a given season from a single disease amounts to \$100,000 or more. A very careful survey was made of some 6,000 acres selected at random from something over 100,000 acres of canning peas grown in Wisconsin in 1942. A very conservative estimate was that about 10 per cent of the crop was lost due to the inroads of one disease, *Aphanomyces* root rot, while another 10 per cent was lost due to the combined effect of all other diseases. Thus the yield of some 20,000 acres was nil. The cost of pea seed alone for 20,000 acres was approximately \$20 per acre, or \$400,000. The average gross return to the farmers concerned was about \$75 per acre. Thus diseases caused altogether a reduction in return to growers of \$1,500,000 in one crop in one state in one year. It may well be emphasized that this loss occurred almost entirely unnoticed by the public generally. There is no particular gain in adding up the annual loss in economic crops from disease which is sustained in this and in other countries. It is stupendous, but it would be difficult to determine accurately. Some is due to neglect; some is due to lack of education; much is due to lack of adequate information upon which to base corrective procedures.

It is also difficult to estimate how much loss is prevented annually by the application of remedial measures. Many crops are now dependent upon scientifically bred and carefully maintained varieties, or upon care-

fully grown seed stocks. Many crops are protected by carefully timed, scientifically devised fungicidal practices. As this is being written, a crop season is closing in the United States in which about 150,000,000 lb. of sulfur and 100,000,000 lb. of copper sulfate, in addition to various other fungicides, were used to protect crops from plant diseases. In spite of this, 10 million dollars' worth of tomatoes were lost in 1946 owing to one preventable disease in one portion of this country. In another part of the country, 2 million dollars was saved by watching the weather and withholding fungicidal applications because they were unnecessary.

Many such examples might be marshaled, but these are sufficient to show that plant-disease control is big business. Moreover, to serve the needs of crop protection, the science of plant pathology must continue to be a dynamic one. It can move forward only through research. Research rests upon understanding and initiative. A basic understanding of the principles of plant pathology is essential to the student in this field. The purpose of this book is to help inquisitive students to attain that goal.

CHAPTER 2

HISTORY OF PLANT PATHOLOGY

The earliest records of man reveal that as he began to depend upon cultivated food, feed, and fiber plants for his livelihood, the problems of crop losses, food shortages, and famine continually arose to plague him. We need only turn to the Old Testament (Gen. 41:23; I Kings 8:37; Amos 4:9; II Chron. 6:28; Deut. 28:22; Hag. 2:16-17) to find references in the history of the ancient Hebrews to blights and mildews of the cereal and vine crops upon which the people depended heavily. Greek philosophers, such as Theophrastus (84) (370-286 B.C.), noted the occurrence of crop maladies and speculated as to their cause and cure. Down through the Middle Ages we find that scholars were impressed by the appearance of plant diseases but were quite confused as to the factors which brought them about. Scientific experiments were not common, and the occasional keen observer of nature was hampered by the current custom of explaining natural phenomena by the process of deductive reasoning. Incorrect interpretations, based on unsound analysis of facts and influenced by superstition and religious dogmas, were passed from one generation to another until they became accepted as hard and fast laws of nature. At the close of the eighteenth century, about 150 years ago, there was no evidence that our modern concepts of the nature of disease inception in plants and the relation of environment to disease development had been adopted by those concerned with plant science and with plant culture.

DEVELOPMENT OF MYCOLOGY

Modern plant pathology did not develop alone; it grew along with and was influenced by related sciences. One of the most important of the allied fields was mycology. The fleshy fungi were thought by the Greeks and Romans to arise *de novo* from dead organic matter under the influence of the elements. This idea came naturally since all the early philosophers believed firmly in the idea of spontaneous generation of lower animals and plants. We shall find that the tenacity with which this theory was held until the middle of the nineteenth century was a dominant factor in the delay of the development of our present concepts of parasitism and disease.

In the sixteenth, seventeenth, and eighteenth centuries some prominent botanists were skeptical of spontaneous generation. Among these were Porta (64), Malpighi (56) (1628-1694), the pioneer in plant anatomy, and Jung (67) (1587-1657), Tournefort (86, 87) (1656-1708), and Jussieu (47) (1699-1777), pioneer plant systematists. On the other side were

such prominent and influential students of the plant world as Cesalpino (21) (1519-1603), another pioneer plant systematist, and Hooke (45) (1635-1703), an early microscopist. The discovery of bacteria (1683) by the Dutch draper, Leeuwenhoek (51), seems not to have influenced current thought on reproduction of lower organisms.

A contribution which stands out in this period, as we now look back upon it, is the work of Micheli (1679-1737). This Italian botanist studied both the higher and lower forms of fungi. In his "Nova plantarum genera" (16, 59) (1729), he described many new genera and illustrated their reproductive structures. He was the first to collect spores of fungi, sow them on organic substrate (nearly sterile pieces of melon), and watch the development of mycelium together with sporangia and spores characteristic of the species. He was convinced that fungi arose from their own spores. However, even these careful observations and the conclusions inductively arrived at were not accepted generally.

In 1748, Needham (17), an English clergyman and naturalist, upheld strongly the theory of spontaneous generation. To prove his point, he boiled meat in flasks, corked them, and set the containers away for a time, only to find that the contents had spoiled due to the microorganisms. In 1775, Spallanzani (79, 80), an Italian investigator, challenged Needham and set out to prove that decay of meat by bacteria and other lower forms of life could be prevented by heating the material in a flask which was sealed off in such a way as to exclude contamination from the air. His experiments supported this contention, but others failed to secure similar results consistently. These failures, we can now understand, were due to what would presently be regarded as faulty technique. This pioneer investigator likewise did not receive recognition and was successfully refuted for the time by his opponents.

The predominant attention of botanists from the time of Cesalpino (1519-1603) was directed toward naming and describing plants rather than searching into the phenomena whereby they grew and developed. By the turn of the eighteenth century the dogma of the constancy of species had become deeply ingrained in the minds of men, and this concept was fostered by the ecclesiasts, who found it to coincide well with the prevalent religious teachings. For 150 years or more, men had struggled to find a rational basis of naming and classifying plants. Linnaeus (1707-1778) was the leader in the eighteenth century who established the presently used Latin binomial system, his work culminating in the publication of "Species plantarum" in 1753 (55). This Swedish botanist had a wide influence. His students and followers were collectors and classifiers, but they were not experimenters. Micheli had been interested in morphology and taxonomy of the fungi, but he had also given attention to

their physiology and reproduction. He was ahead of his time. Linnaeus was a product of scholasticism and a firm believer in the dogma of constancy of species. He assented to the view that fungi arose through spontaneous generation.

While Linnaeus did not devote much time to the fungi, some of his followers specialized in this field. Prominent among these were Persoon (63) (1755–1837), who worked chiefly in France; Schweinitz (69) (1780–1834), in eastern United States; Fries (39) (1794–1878), in Sweden; Nees von Esenbeck (60) (1776–1858), in Germany; Léveillé (52–54) (1796–1870), in France. Persoon's "Synopsis methodica fungorum" (63), published in 1801, and Fries's "Systema mycologicum" (39), published in the years 1821 to 1832, became the foundation stones of the modern application of the Linnaean system to the taxonomy of the fungi. In the main, these men were concerned with the naming of fungi and with their classification, and they were not concerned with their reproduction and with their relation to plant disease. Although Persoon gave the name *Puccinia graminis* to the fungus associated with the black stem rust of wheat in 1794, he did not regard the fungus as the incitant of the disease. In 1818 he expressed the opinion that some fungi grew from spores, but that others arose by spontaneous generation.

In 1807, Prévost (65) observed germination of the spores of the wheat-bunt organism and conceived the idea that this organism penetrated the young wheat plant and was the actual cause of the disease. His revolutionary idea was refuted and did not gain favorable attention until 40 years later. Although predominant attention was given by most mycologists to taxonomy, there began about 1820 a renewed interest in the type of study that Micheli had undertaken 100 years earlier. Ehrenberg brought together known information on propagation of fungi and contributed further observations of his own on spore germination, as well as the first description of sexuality in fungi. Nees von Esenbeck repeated Micheli's experiment by sowing bread-mold spores on bread and later obtaining ripe sporangia. Dutrochet (1834) connected the sporophores of fleshy fungi with the subterranean mycelial strands which had been considered up to that time as distinct fungi. Trog (1837) confirmed earlier claims that fungi were disseminated by means of air-borne spores (67).

About this time there began to appear more discrete studies of the morphology and life history of fungi. The "Icones" of Corda (22), Bohemian botanist (1809–1849), published from 1837 to 1854, were the first result of the application of the compound microscope to the description of fungi. They were followed by the even more exacting and equally well illustrated studies of fungi by the French botanists, Louis René

Tulasne (1815-1885) and Charles Tulasne (1817-1884), whose works extended from about 1841 to 1865 (88-91). Contemporary with these men was Berkeley (1803-1889), the pioneer British mycologist.

PREMODERN PLANT PATHOLOGY

During the seventeenth century, plant diseases were increasing in importance in Europe with increase in population and in intensity of plant culture. Farmers, by and large, accepted them as inevitable concomitants of unfavorable soil and climate. Some growers, however, tried to do something to alleviate the damage, for we know that treatment of wheat seed with salt brine to control bunt began in that century (92). The association of the barberry with stem rust of grain had been recognized by farmers for centuries. It has been claimed by some that in 1660 farmers in the vicinity of Rouen, France, secured passage of a law which required destruction of the barberry bushes in that area. The authenticity of this report has never been firmly established. We are certain, however, that colonists took the barberry to New England with them and that wheat rust became increasingly destructive after 1660. A law was passed in Connecticut in 1726 which authorized town meetings to adopt barberry-eradication measures. Similar legislation was enacted in Massachusetts in 1755 and in Rhode Island in 1766. It should be emphasized that in the development of the control practices mentioned above there is no evidence that anyone had the present-day concept that many diseases are brought about by separate infective entities under the influence of favorable environment.

Theophrastus recorded his observations on the effects of various diseases of plants and in the main assumed that the basic causes lay in the vicissitudes of the weather. Pliny the Elder wrote upon the diseases of trees and of the cereal grains, but his work is little more than a translation of that of Theophrastus. Giambattista della Porta's discussion of diseases of plants (64), consisted largely of quotations from Theophrastus and Pliny, with some observations of his own. In 1705, the French systematist Tournefort (86) (1656-1708) classified plant diseases, dividing them into two major groups, one due to external causes, the other to internal causes. There followed a period of 150 years in which most writings on plant diseases consisted in descriptions of their appearances or symptoms and an arrangement of them into classes, genera, and species. The influence of the Linnaean binomial system was evident in each of these compilations. Adanson (1) (1763) in his work on "Familles des plantes" enumerated 23 species of plant disease, which were divided into two classes depending upon whether they were due to internal or external causes. The nature of the causes was, however, primarily a matter of speculation.

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In Rozier's "Cour complet d'agriculture" (66) the list was expanded to 32 species. Plenck divided diseases into eight classes: external injuries, flux of juices, debility, cachexias, putrefactions, excrescences, monstrosities, sterilities (66). In 1773, Zallinger (101) (1731-1785) placed plant diseases in five classes: phlegmasiae (inflammatory diseases), paralyses or debilities, discharges and drainings, cachexia, or bad constitution of the body, chief defects of different organs. Fabricius (35), a Danish botanist who studied under Linnaeus and was for a time on the staff of the Royal Botanical Garden in Copenhagen, published a classification of plant diseases in 1774. He spent much time in Paris and evidently drew much of his material from Adanson's book. His treatise is an arrangement of diseases into classes, genera, and species on the basis of their symptoms. That Fabricius was not satisfied with current explanations of certain plant diseases is indicated by his statement that "certain it is that the cases and symptoms of smut can never be better explained than by assuming something organized to be the cause."

In 1755, Tillet (85) (1714-1791), director of the mint at Troyes, France, and amateur botanist and agriculturalist, published an important paper on wheat bunt. He found that opinion varied widely as to the cause of the disease—evil fogs, winds, excessive soil moisture, improper plant nutrients, soil texture, insects. In well-planned field experiments he tested the effect of dusting seed with the black mass contained in diseased kernels, and of treating such seed with water containing common salt and lime. He demonstrated that the disease was contagious, since dusted seed produced a much larger percentage of diseased plants at maturity than did undusted seed, while seed treated with salt and lime produced a lower percentage of diseased plants. Tillet did not recognize that the smut dust consisted of spores which were "seeds" of a parasitic fungus. He rather looked upon the spore dust as merely the carrier of a poisonous entity. However, he did establish for the first time that the disease was in some way contagious through the smut dust and that healthy seed produced only healthy plants. Furthermore, he demonstrated clearly that chemical treatment inhibited the contagious entity. In 1759, Count Francesco Ginanni (66) in Italy published a book on the same subject entitled "Trattato delle malattie del grano in erbo," in which he attributed the cause of bunt to minute insects. Another Frenchman, Tessier, working in the same period, published a paper entitled "Traité des maladies des grains," in which he attributed rust to a stoppage of the transpiration of the plant, caused by mists (83).

In 1766 an Italian, Giovanni Targioni-Tozzetti (66), put forward a theory that the rusts and smuts of wheat consisted of the vegetation of fungi arising under the epidermis of the plant. Fontana (38) (1730-1805)

an Italian physicist, published a paper the following year (1767) in which he expressed the view that the grain rust was a distinct parasitic entity. This new theory received some support, no doubt, but the weight of opinion was against it. In this same decade Anderson, an Englishman, wrote concerning the degeneration of potato varieties which we now know to be due to infectious viruses. In 1778 he published evidence to show that the malady was tuber-borne and likened it to smallpox in its infectiousness. Zallinger (101) in his book on plant diseases, "*De morbis plantarum*" (1773), upheld the prevalent concept that fungi found in association with plant-disease lesions were abnormal structures of the diseased plants.

BEGINNINGS OF MODERN PLANT PATHOLOGY

It is apparent that at the beginning of the nineteenth century when Persoon and, a little later, Fries were actively naming and compiling fungi without reference to their relation to plant disease, those who were writing about these maladies were still not cognizant of the true relation to them of microorganisms. The first investigator to bring forth the facts which clearly demonstrated the pathogenic nature of any microorganism was Prévost, a Swiss professor of philosophy at the Academy at Montauban, France. He studied the wheat-bunt disease for about 10 years beginning about 1797. He published in 1807 his epochal "*Memoir on the Immediate Cause of Bunt or Smut of Wheat, and of Several Other Diseases of Plants, and on Preventives of Bunt*" (65). After presenting an accurate description of the disease, he reported important results of experiments upon it. He studied and described the germination of the spores. He demonstrated, in confirmation of Tillet, that spores applied to the seed in some way resulted in infecting the plant. He studied the growth of the fungus within the growing wheat plant and its final development in the embryo where spores were formed, and where the life cycle of the fungus was completed. He showed that solutions containing copper sulfate prevented spore germination. He distinguished between fungicidal and fungistatic effects. From these facts he demonstrated the basis of control through seed treatment, a practice which had been carried on empirically and without any rational explanation for 100 years and more. The thorough hold of the theory of spontaneous generation on men's minds at this time was demonstrated by the fact that Prévost's results and interpretations were not accepted. A committee of the French Academy rejected his conclusions as unsound, and some 40 years had to elapse before others confirmed Prévost and pressed the adoption of the germ theory.

In the period from 1807 to 1845 the prevalent teachings are illustrated in the writings of Count Fillipo Ré, Franz Unger (1800-1870), and Franz

Julius Ferdinand Meyen (1804–1840). Ré was professor of botany and agriculture at the University of Modena, Italy. His treatise on the diseases of plants was published in 1807 (66), in the same year as the publication of Prévost's memoir. A second edition was published in 1817, and in 1849 Berkeley published an English translation of the latter in the *Gardener's Chronicle*, stating that it was the best book available on the subject. It is of interest to take more than casual note of Ré's book, because it portrays the increasing conflict of thought on the nature of plant disease in the beginning of the nineteenth century. Diseases were divided into classes and genera on the basis of their symptoms and their supposed causal factors. The plan was not fundamentally different from that of classifications presented in the last half of the previous century. The influence of current writings on the classification of human diseases is evident. We need mention here only the titles of the five classes: (a) diseases constantly sthenic (arising from excessive vigor, inflammatory); (b) diseases constantly asthenic (arising from debility); (c) diseases derived sometimes from the sthenic, sometimes from the asthenic state; (d) mechanical injuries, wounds; (e) indeterminate diseases. In the last class were those diseases which the author regarded as of uncertain origin. Among the diseases of that class were the rusts and smuts. Ré's comments on this group are quoted:

I have thus designated those diseases whose origin is either entirely unknown, or deduced from observations contradictory in themselves, or from hypotheses which, however brilliant, have no real foundation. It is indeed a mortifying thing to see amongst these diseases precisely those which from the earliest times have been the subject of the studies of philosophers and that they are those which produce the greatest havoc in this country, while Nature refuses as yet to disclose to us the secret of their causes. Nevertheless, as I have said in my preliminary discourse, there are no vegetable diseases on which so much has been written by men of sound doctrine, of exactness of observation, and of careful judgment. . . .

The principle opinions on the cause of rust, blight, melume, yellow blight, and carbuncle may be reduced to four. . . . The most generally received theory, first put forward in 1766, by the celebrated Dr. Giovanni Targioni-Tozzetti is, that these diseases consist in the vegetation of cryptogamic plants, arising under the epidermis of corn; and according to others, also on leaves and fruits of trees: and this has been confirmed by the microscopic observation of Fontana, Saussure, and more recently of Bauchs. . . .

The opinion of Targioni has been latterly illustrated by several others, but I confess that I cannot yet bring myself to adhere to it. Some difficulties occur to me in the fact that notwithstanding the microscopical researches of Bauchs, others see differently also through the microscope. It appears also that he was unacquainted with the chemical and microscopical investigations of Girod-Chantrous, which have been passed over in silence by all those who have written on these diseases. . . .

Another difficulty in the explaining these difficulties by their being the result of cryptogamic, as it were intercutaneous vegetation, is the necessity of having recourse to principles which will not so readily be admitted by impartial persons. In the case of rust, for instance, some will have it that it is one plant, some that it is two different ones; and the manner in which they explain its appearance is precisely the same as that which those adopt who consider it an animal production. It is agreed that the seeds of these plants attack the *Cerealia* externally; and to show how this can be, they say they are minute enough to penetrate through the organic pores of the epidermis, establish themselves underneath it, and take root in the cellular tissue or parenchyma of the plant. Gautieri, on the other hand, in a memoir on the means of destroying rust in wheat, gives a totally different explanation of its mode of production, though founded also on the observations of Bauchs and Carradori. With all due honour to these great men, I confess that they leave me still in doubt, and I have therefore classed all these blights amongst diseases of which the cause is not determined.

If to all these opinions it is wished that I should add my own, I should commence by remarking that all agree in one point, that is, in the circumstances under which rust usually shows itself. I say, usually because some exceptions observed have occasioned those doubts which prevent me from laying down new hypotheses. Fields in low situations are generally the most subject to the disease, although in some years elevated spots have been more infected than low ones; so also fields well-manured or naturally rich have been the most affected. The milder or hotter the season the more these blights abound, especially in hot and moist years. Fields adjoining irrigated or rice grounds have most rusty plants. In little mountain valleys, much subject to fogs, blights abound. The greater the vigour of vegetation in the plant, the more it has to fear from them. All this would induce me to lay down that the cryptogamic plants, the minute insects, or the exudations, whether dry or not, are rather symptoms of the disease itself, which is a result of excessive vigour or over-repletion. And I am the more induced to believe it, as I have observed that weak trees and corn growing in poor soils are less liable to blights and rusts. It may also be that the transpiration is checked by the particles of wet (and for that they need not act as lenses but as mere obstacles), and that there follows a disorganization occasioned by the matter of transpiration itself, which might easily happen, considering that it is chiefly oxygen, and that facilities are thus given for the development of extraneous matter, vegetable, animal or whatever it may be.

The pressure of the parasitic theory is evident from the remarks of Ré, who evidently did not see the work of Prévost before the second edition of his book was published in 1817. The experimental evidence was lacking, except that presented by Prévost, to clinch the germ theory. The adherents of the autogenetic theory of disease still had the better of the argument.

Franz Unger was born in Germany but spent most of his life in Austria-Hungary. He was trained in medicine but in early mid-life turned to botany, becoming a professor of botany in Graz in 1835 and professor

of vegetable physiology in Vienna in 1849. In 1833, while serving as a medical officer in the Tirol, he published a book entitled "Die Exantheme der Pflanzen" (93). He believed that the fungus organisms associated with disease were endophytes but not parasites, that they had no independent existence, and that under certain environmental conditions every plant had the potentiality of producing them, in some way, from the waste material or exanthema of the plant. The influence of human pathology upon the interpretation of plant disease was still strong.

Meyen, a professor of botany in Berlin, was concerned primarily with plant anatomy and physiology, but he wrote a book on plant pathology which was published in 1841, in the year following his death (58). He followed Tournefort's system of classification of plant diseases. His erroneous concepts were characteristic of his period. He considered smuts of cereals to be noninfectious and to be due to a rotting of the sap brought about by improper fertilization of the soil. Rusts and mildews were likewise regarded as the by-products of disease due to environmental factors. He described the formation of the pustules of white rust on shepherd's-purse in a unique manner. He claimed that as the disease attacked the plant, cells of the latter enlarged and stretched out in some way toward the epidermis and cut off several spores in succession. Thus this incomplete picture of the development of sporangia as we now know it (p. 211) was misinterpreted to strengthen the autogenetic theory of disease.

About 1830, a few decades after the culture of potato as a food crop had expanded rapidly in Europe and North America, the late-blight disease appeared in both continents. It reached epidemic proportions by 1845. In that year it received much attention by writers in agricultural papers in Europe and the United States. The editorials and comments by contributors in the *Gardener's Chronicle* of 1845 are particularly enlightening to the student of plant pathology because they reveal better than any other existing evidence the trend of thought of laymen and scientists on the nature and cause of this destructive disease. While the association of the causal fungus with the blight of foliage and the rot of tubers was recognized by all who studied the disease closely, most writers held, in accord with the autogenetic theory of disease upheld by Unger (94) and by Meyen (58), that the fungus was the result rather than the cause of the disease. Von Martius, in Germany, was one of the first to distinguish the fungus (1842). He ascribed the disease to the effects of the fungus and published drawings of the latter. In 1845, Morren, director of the school of agriculture at Liège, Belgium, where the disease had devastated the potato fields, described the disease and the fungus associated with it. He conducted crude inoculation experiments with the fungus and con-



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