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CHAPTER 2

The Nature, Origin, and Evolution of Parasitism

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Any interference with the normal metabolism of a plant that prevents it from making full use of the factors available in its environment for growth and reproduction must be considered as a disease. The agent that interferes with the physiological processes of a plant is a pathogen regardless of whether it is living or inanimate.

The living pathogens have been of much more concern to plant pathology than the inanimate ones because of their ability to multiply and create epidemics. Most of the living pathogens are parasitic because they invade one or more plant tissues to procure food but parasitism is not a prerequisite to pathogenicity. As a matter of fact the processes of parasitism and pathogenicity in an organism often are entirely different and specialization for one attribute is often made at the expense of the other.

I. Interrelationship of Parasitism and Pathogenicity

The interrelationship of parasitism and pathogenicity can best be interpreted by considering the pathogen and the injured plant in a disease association as symbionts. In the classical sense employed by De Bary (1879), there is symbiosis whenever two organisms occupy the same habitat. In a few unique associations the symbionts are mutually independent and nonresponsive to each other but usually they affect each other's development. The physical contact of symbionts ranges from disjunctive to fully conjunctive and their effects upon each other vary from mutual or unilateral antagonism to synergism. The following illustrations of unilateral, antagonistic symbiosis will serve to define the range of pathogenic processes.

Very few, if any, organisms can occupy the same habitat as another without influencing its growth. For example, the sugar beet inhibits the germination of seed of peppergrass in its immediate vicinity presumably because of the toxic materials it secretes such as *p*-hydroxybenzoic acid, vanillic acid, ferulic acid, and *p*-coumaric acid (Massart, 1957).

The soil sickness resulting from the growth of wheat, rye, and other crops is caused by picolinic acid and other chemicals in the soil (Schreiner and Sullivan, 1909). The glycosides and phenolic substances from an apple or peach root (Patrick, 1955) prevent replanting of younger trees in an old orchard. Juglone and its trihydroxy naphthalene analog from roots of the black walnut tree poisons the soil for many other forms of plant life such as tomato and cabbage which become chlorotic and die (Massey, 1925). Such plants cause disease in other plants and, hence, must be considered as pathogens even though they do no more than foul the environment with by-products of their metabolism. This is pathogenesis by independent association of two subjects, but it is not parasitism.

Most pathogens, however, have a much more intimate relation with the infected plant based upon a nutritional affiliation in which one serves as the host to the parasitic endeavor of the other. A plant parasite is any form of animal or plant life that invades a plant and multiplies or grows

at its host's expense without contributing much, if anything, in return. The arbitrary removal of water, mineral elements, or synthesized food from the metabolic pool of the host usually will prove detrimental to its further development, so parasitism in most plants automatically leads to pathogenesis.

For example, the mistletoe of ponderosa pine is a pathogen even though it is fully capable of synthesizing its own foodstuffs. It creates disease by interfering with movement of minerals and nutrients to the foliage and possibly by adding injurious materials to the tissues of the pine tree.

Many parasitic establishments do not become pathogenic if the parasite compensates for the damage done by its presence. The mycorrhizal fungus certainly is a parasite on the root of trees but it may be relatively noninjurious or even may be beneficial in some soils by increasing the absorbing area of the root so as to expedite uptake of mineral nutrients from the soil (Burges, 1936; Hatch, 1937; Funke, 1942). By strict definition this is a commensal association rather than parasitism since the invader contributes to the welfare of its host.

There may be other auxiliary benefits from parasitism that completely compensate for the damage done to the host. The legume nodule bacterium (*Rhizobium leguminosarum*) causes a disease reaction in the nature of a proliferating gall which undoubtedly diverts foodstuffs from normal cell metabolism and orderly growth. However, the extra atmospheric nitrogen fixed by the nominally parasitic bacteria eventually proves to be so beneficial to host development that the relationship must be considered to be a mutually beneficial symbiosis or commensalism of bacterium and higher plant.

It follows that the symbiosis between the bacterium and its host is more likely to be beneficial to a legume growing in a nitrogen-deficient soil than in one supplied with adequate amounts of nitrogen. As a matter of fact, if soil nitrogen were present in optimum supply for growth of the legume throughout the season, it is conceivable that the nodule bacteria might be considered as a gall forming, pathogenic parasite rather than as a commensal symbiont.

These brief examples illustrate the basic facts regarding pathogenesis. Plant diseases so caused are the product of an interplay between the metabolism of the plant, physiology of the parasite or other symbiont, and the environment. In a few exceptional associations, there can be parasitism without severe pathogenesis, and disease induction without parasitism. Parasitism usually is an attack on organic food reserves by a chlorophyll-deficient form of plant life but it certainly is not restricted to these primitive forms of life or this one class of nutrients. The intensity

of the disease reaction will be determined by the physiological balance existing between the two symbionts as mediated by the environmental forces operating upon their symbiosis.

II. The Physiology of Pathogenicity

In one broad sense, plant pathology is an aspect of plant ecology. Like all other ecological sciences, it must turn to physiology and biochemistry for the ultimate answers as to why plant societies behave as they do. However, a plant disease is something more than the sum total of the physiology of the host and the pathogen. It is the physiology of a symbiotic state in which the biochemical activities of pathogen and host are completely consolidated.

A. *Appropriation of Nutrients by Pathogen*

There are considerable data on the physiology of the fungi (Foster, 1939, 1949; Wolf and Wolf, 1947) and bacteria (Dowson, 1957) that cause plant diseases. Unfortunately, however, there are all too few data on the biochemistry of their parasitic activities and pathogenic capabilities while in the host (Gäumann, 1950; Allen, 1954; Lilly and Barnett, 1951). The measure of their parasitic activity should be ability to remove different classes of foodstuffs from the host.

B. *Toxin Formation by Pathogen*

The loss of food materials undoubtedly constitutes a drain on the metabolic activities of the host and leads to inefficiency in its growth and reproduction. However, most parasitized plants are damaged much more than would be expected from the drain on their metabolism in supplying nourishment to the invader. This comes from increased respiratory activity of the invaded tissues, disintegration or collapse of the adjacent tissues, and various physiological stimuli that cause wilting, cell proliferation, cell elongation, abscission of leaves, and degeneration of chlorophyll.

These effects are the results of the extracellular materials secreted by the pathogen or incited to production by the infected cell. Gäumann (1954) refers to these materials as toxins but they could be appropriately subdivided into more specific classes such as extracellular enzymes, auxins, wilt toxins, and cell stimulants. Those materials that regulate disease processes are attracting more attention but there is not enough known about them to permit the classification of disease according to the physiology of the pathogens.

C. *The Six Major Processes Affected*

Attention may be directed more profitably to the activities of the host as a basis of understanding disease processes because the basic knowledge on the physiology of higher plants is fairly well understood. It revolves around the synthesis, transport, use and/or storage of food materials, and the auxiliary services thereto. In its simplest terms, the chronological sequence of events in the six major processes is as follows: (1) A food reserve consisting primarily of energy sources in carbohydrates or lipids and secondarily in nitrogenous materials is used to nourish the embryonic tissues of seed, tuber, root, or bud after the dormant period. (2) Juvenile tissues that are dependent upon these reserves develop as seedlings or shoots. (3) Roots are established to procure water and mineral elements essential to growth, photosynthesis, and protein metabolism. (4) The nutrients and water supplies are transported to the leaves and growing points through the tracheal tubes. (5) The green leaves assimilate a new supply of carbohydrates by photosynthesis so the young plant becomes independent of the food reserve. (6) The products of photosynthesis are transported to the area of cell utilization where they are incorporated into new tissues or else are stored in seeds, buds, roots, or stems.

Thus, there are six vital steps in the life of the host, six vital processes. In this chapter we shall refer often to these six vital processes and the diseases thereof. Any one of the six may be injured or even wholly blocked by a pathogen.

If any one of the processes is brought to a stop, the plant succumbs but this is the exception rather than the rule. Ordinarily, the function is injured but not destroyed so only the efficiency of the plant is affected. A major objective of plant pathology is to learn how these processes are injured and to design methods of preventing or circumventing that injury. The science, therefore, has as its applied objective the conservation of foodstuffs and the promotion of maximum efficiency in their synthesis and utilization by the plant.

If the pathogens that disrupt each of these six processes are grouped together irrespective of their taxonomic derivation, a surprising degree of order is obtained. The ones in class 1 that do no more than attack and destroy food reserves are good pathogens but very poorly developed parasites. They are essentially opportunistic saprophytes. Likewise, the soil saprophytes that attack seedlings (class 2) and roots (class 3) are good pathogens and relatively poor parasites. They become harmful only when conditions handicap the host so as to keep it in a susceptible

state. The vascular or wilt parasites in class 4 are facultative parasites with very definite parasitic specialization.

Some of the leaf invaders of class 5 in the Fungi Imperfecti are only slightly better parasites but a high state of parasitism is attained by some, such as the downy mildews. The powdery mildews and rusts are obligate parasites on foliage. They have lost all capability for an alternate existence as a saprophyte and have become so dependent upon the host's physiology that parasitic races are necessary for their survival. The pathogens in class 6 that have come to reside inside the host cells and regulate cell activities in the metabolism of proteins, cell division, and cell differentiation show an appreciable degree of commensalism regardless of whether they are obligate or facultative parasites.

Here, then, is a system for organizing the types of pathogenesis. It is based on disruption of physiological processes of the host. There is enough order and logic to this to warrant its further study. By so organizing the knowledge on plant diseases into an integrated system depending upon function, certain natural laws of pathogenesis can be evolved that are valuable in guiding research into fruitful channels and in organizing knowledge so it can be learned from principles rather than by rote.

The basic tenets of this system are that: disease is an abnormal physiological process in plants; the physiological processes are constantly in a state of flux; there is an orderly progression of parasitism depending upon which function of the host is under attack; and the system is natural enough to permit development of some general laws and principles for understanding disease and its control. Various aspects of these concepts are examined in the following sections.

III. The Natural Processes of Pathogenesis

A plant is diseased whenever its normal functions are impaired so it cannot operate at maximum efficiency under prevailing environmental conditions. Inefficiency (i.e., disease) comes from the loss of an organ, destruction of food reserves, impairment of essential functions such as photosynthesis and translocation of foodstuffs, or abnormal utilization that results in abnormal growth. For example, the abscission of a leaf attacked by a blight organism or the severance of roots by a rot organism destroys the normal balance between the water-supplying and food-synthesizing capacity of a plant. As soon as one is destroyed, the other cannot operate at maximum capacity so there is a loss of efficiency.

Disease is a physiological process regardless of whether a tissue is decayed or not. The blocking of photosynthesis, interference with translocation or induction of abnormal cell respiration is disease even though

these abnormal processes may not be visible directly to the eye of the diagnostician.

A. *Interference with the Six Vital Processes*

The interference with the six vital processes of the host mentioned in the preceding section leads to six classes of pathogenesis. It is well to examine the nature of the pathogens and the six classes of disease they cause before proceeding to other considerations.

1. *Destruction of Food Reserves*

The pathogens that attack storage organs and organic material cause seed decay, timber rots, soft rot of vegetables, and fruit breakdown (see also Chapter 5 of Volume I). Most of the fungi and bacteria involved are very poor parasites and ordinarily live as saprophytes upon decaying organic matter. Most of them are habitual soil-inhabitors where organic matter provides carbohydrates and cellulosic materials but some are primarily aerial in habitat.

With very few exceptions their parasitic capabilities are limited to invasion of the storage organ through a wound or other unprotected tissue. Once inside the host tissue, they digest starches and cellulose by virtue of ability to secrete amylases and cellulase. In addition, many of them have ability to break down plant tissues by use of protopectinase and pectinase (Thornberry, 1938) as was first demonstrated for *Erwinia carotovora* by Jones (1909).

Among the typical examples of this class are the soft rots of vegetable caused by *Erwinia carotovora*, mold of drupe fruits caused by *Rhizopus nigricans*, soft rot of potato caused by *Erwinia atroseptica*, storage rot of lily bulbs caused by *Rhizopus necans* and dry rots of woody plants. In each of these, the host tissue is destroyed as a general cortical necrosis. As each cell succumbs it provides food to the parasite for further aggression. The host is essentially inert and does not respond to the aggression to any appreciable extent. These are potent pathogens but poorly developed parasites.

2. *Prevention of Seedling Metabolism*

The seedling blights are likewise caused by general soil saprophytes. Sometimes a parasite that attacks the seed will also invade the radicle or epicotyl through the wounds created by the emergence of adventitious or secondary roots. The pathogens on seedlings are one step more advanced parasites than the soft rot agents, since they do invade a rapidly growing, metabolically active tissue. As a rule, they are aggres-

sive in juvenile tissue but may be restricted as soon as the adjacent tissue begins to achieve more mature differentiation.

Among the pathogens in this class are such common damping-off and seedling blight fungi as *Pythium ultimum, Pythium debaryanum, Rhizoctonia solani,* and *Diplodia zeae.* It should be noted that the latter two also can attack more mature plants. *D. zeae* is very aggressive on the seedling and often becomes established in the root crown of the corn plant where it remains relatively inactive until the plant is past its prime period of growth. The pathogen spreads aggressively again after the corn plant reaches maturity and begins to enter into senility.

3. *Interference with Procurement*

The root rotting diseases are caused by a wide variety of fungi and bacteria that are capable of either colonizing and enduring in soil or of invading it temporarily. They show some advance in parasitism over seedling pathogens since they can invade well-formed roots through lignified, mature tissue. Still, most of them are poorly specialized as plant parasites but they are quite aggressive pathogens. The routes of invasion are usually through wounds created by emergence of secondary roots, abrasion with soil particles, nematode penetration, or feeding of insects. However, some of them such as the cause of black root rot of tobacco, *Thielaviopsis basicola,* will form mycelial mats on the surface of the root and force a penetration directly through the epidermal wall by an infection peg that secretes enzymes or exerts direct pressure. Roots may be preconditioned to invasion by excess moisture or nutrient conditions such as are found in the brown root rot of wheat (Vanterpool, 1940). Apparently under anaerobic conditions during prolonged heavy rainfall, an accumulation of salicylic aldehyde predisposes the root to invasion by the fungus (Graham and Greenberg, 1939).

Many other factors in the rhizosphere influence the transition of the soil saprophyte into a pathogen. Apparently the organisms are stimulated to grow along the diffusion gradient of carbohydrates from roots. The mycelium grows more rapidly toward the source of food supply in the roots. However, the exudates from some roots, such as those from varieties of peas resistant to *Fusarium oxysporum* f. *pisi,* may be toxic to the pathogen and prevent its reaching the site of invasion (Buxton, 1957). Even the carbohydrates secreted by a root may encourage the growth of antagonistic soil saprophytes such as those that protect the cotton root from invasion by *Ozonium omnivorum* (Eaton *et al.,* 1947). The roots of cotton seedlings appear to be quite resistant to invasion because of the substantial amounts of carbohydrates in the roots, but

quickly become susceptible as the sugar concentration declines during the period of rapid growth. It then seems to be resistant again in the advanced stages of its development (Blank, 1940) as sugar concentration increases sufficiently to affect the rhizosphere population.

In any evolutionary scheme, the root pathogens might be classified as the first strongly aggressive plant invaders. They include such destructive fungi as the species of *Pythium* on sugar cane, maize, and wheat; *Aphanomyces* spp. on sugar beets and peas; *Fusarium* spp. on pea, bean, and other crops; and *Ophiobolus graminis* on wheat. The ones of these that are natural soil inhabitants are difficult to avoid and are chronically destructive but the soil invaders that are poorly adapted to survival apart from their hosts in competition with other microorganisms (Reinking and Manns, 1933, 1934; Garrett, 1944, p. 13) may be avoided by proper treatment of the soil to favor their antagonists (King, 1940; Hildebrand and West, 1941).

The root rot organisms are poorly specialized parasites even though some have become adjusted to the rhizosphere environment and thrive best on or in roots. They are general cortical invaders with very little specialization in preference of tissue. The host has very poorly developed resistance. Most of the resistance exploited agronomically is in the nature of disease-escaping tactics where a particular variety has ability to develop new roots to replace those destroyed by the pathogen or has a favorable seasonal development that avoids the infection period.

4. *Interference with Upward Transport*

The vascular diseases or wilt diseases interfere with upward movement of water and mineral nutrients. They are caused by xylem parasites (see also Chapter 9 of Volume I). Some of them live in soil and have habits similar to those of the root rot fungi. They invade directly through root hairs or natural wounds, traverse the cortical tissues and become established in the tracheal tubes. Although they are only facultative parasites, they show a much higher order of specialization than the root rots in their preference of a specific tissue and for certain varieties of crops. They may also be considered more highly specialized because the typical disease symptoms are induced by response of the host to special causative chemicals.

Two classes of wilt organisms have to be considered, the soil-borne and the insect-borne. Many of them such as the *Fusarium* spp. on cabbage, cotton, tomato, watermelon, and banana are normal soil inhabitants. Others, such as the bacterial wilts of maize caused by *Bacterium stewartii* and of cucumber caused by *Erwinia tracheiphila* and the fungal

wilts of oak caused by *Endoconidiophora fagacearum* and elm *Ceratostomella ulmi,* are insect-borne but they have essentially the same habits of invasion as the soil colonizers.

The masses of mycelium and bacterial cells present in the tracheal tubes interfere with the movement of water and nutrients and some tubes respond to invasion by forming tyloses that further complicate free movement of materials to the leaves. These mechanical impediments will not explain fully all the wilting symptoms so attention is focused upon the metabolic by-products of the parasite. There are a number of wilt-inducing toxins formed in cultures of a fungus such as *Fusarium lycopersici,* the cause of tomato wilt, which has been studied by Gäumann (1957); Davis and Dimond (1954), and Dimond and Waggoner (1953). The principal problem confronting these investigators has been to prove that enough of any one or any combination of these materials is present in the tracheal tubes at any one time to cause the wilt symptoms (see also Chapter 9 of Volume I).

The wilt organisms are fairly well specialized facultative parasites. There is a definite mechanism of pathogenesis above that induced in their normal parasitic activities. There are very specific forms of resistance in the host and it is possible to breed for such types of resistance. In any evolutionary scheme, these pathogens would rate rather high both as parasites and as pathogens with specific abilities; but there is no evidence that they and the host have any tendency to establish a commensal state. They cause general necrosis and frequently rupture the tracheal tubes and overrun nearby parenchyma cells.

5. *Destruction of Food Manufacture*

The leaf-invading pathogen (Group 3) include at least four diverse groups ranging from poorly specialized facultative saprophytes to obligate parasites. Most of the general leaf-blight organisms have ability to infect and destroy the stems, flowers, and fruits, as well as the leaves. Thus they can cause substantial loss in food manufacture by stem cankers, flower blasts, and fruit rots, scabs, anthracnose, etc.

This overlapping of parasitic activity on different organs is not surprising because the tissues involved are morphologically analogous. The flower, stem, and fruit may be considered as modified leaves that have assumed special functions other than photosynthesis. They have comparable epidermal cells and cuticle—at least while in the juvenile condition. The nectaries, lenticels, and hydathodes may be considered as having modified stomata.

The primary mechanism of pathogenesis is destruction of the food-synthesizing potential of the plant. This results from local necrosis of the leaf blade, which may become very extensive in some diseases such as late blight of potato, caused by *Phytophthora infestans.* In many other diseases, the damage is entirely out of proportion to the amount of necrosis because plants are defoliated by abscission-inducing substances such as those found in shot hole of peach caused by *Xanthomonas pruni* or are rendered chlorotic as in cherry leaf spot caused by *Coccomyces hiemalis.* Extensive rupture of the cuticle by the sporulating fungus may disturb the water economy of the plant with serious consequences such as often occur in wheat fields affected with *Puccinia graminis.*

a. *The Leaf Blights, Cankers, and Rots.* The leaf blights and stem cankers are caused by fungi and bacteria that range from very poorly adapted to highly specialized parasites but all are facultatively saprophytic. Taxonomically they are Schizomycetes, Fungi Imperfecti, and Ascomycetes. The lower members of the series are analogous to the root rot fungi in their parasitic potentials. As a matter of fact, some of them do attack roots and underground parts of the plants. The fire blight organism (*Erwinia amylovora*) may cause root cankers in the Pacific Northwest of the United States. Conversely, some of the root rot fungi may splash upon the stems of plants and cause stem cankers.

In spite of these overlapping tendencies it would seem well to separate the root rots and canker diseases because the mechanism of action is largely different, the habitat is usually entirely different, and there usually is more host specialization in the leaf invaders. There is no comparison, for example, in the parasitic specialization of *Pythium debaryanum* and *Venturia inaequalis.* In between these two are all degrees of specialization but usually there is one very significant difference. The root parasites have a well-developed mycelial stage whereas the leaf parasites have very little mycelial development apart from the host. The aerial parasite exists as a fungous spore or bacterial cell that penetrates directly or through a very restricted infection hypha. This becomes a major consideration in determining the nature of the parasitic establishment and the methods to be employed in controlling the two classes of disease agents.

b. *The Downy Mildews.* The downy mildews and their allies are caused by much better specialized parasites than the general leaf blights. The members of the Peronosporaceae, Albuginaceae, and Phytophthora are facultative to obligate parasites that establish liaison with the leaf tissue and maintain a more or less compatible relationship until the fungus begins to sporulate.

They usually have rather specific host requirements and there are sharp differences in the varietal reaction of plants to their invasion. It is possible to breed for disease resistance. The use of haustoria by the

pathogen to procure food with a minimum of injury to the host appears for the first time in this group. In general, this group of pathogens has a much more highly developed type of parasitism than any of those in the preceding group, both as to host specificity and a restrained type of feeding which avoids immediate destruction of the invaded tissue.

c. *The Powdery Mildews.* The powdery mildews are caused by obligate parasites (Erysiphaceae) with well-developed haustoria that permit them to feed on the cell contents without extensive injury to the supporting tissue. The dense growth of mycelium and sporophores on the leaf surface accelerates respiration and undoubtedly decreases photosynthesis by screening out the sun's rays. However, infected leaves persist for weeks and may show only the slightest traces of necrosis. This could be attributable to the fact that relatively little of the parasite's body is inside the host tissue where its toxic secretions would be added to the cells of the host.

The specificity for hosts is very marked. The physiological races that differ in varietal host preference (which were first encountered in the more specialized leaf blights such as *Colletotrichum* spp.) are found regularly in the powdery mildews. The significant feature about the parasitism of the powdery mildews is that the parasite has no saprophytic existence apart from its host and must depend entirely upon specialization in parasitism for its survival. With this change, there appears some evidence of a very compatible establishment in the host even though the host tissue is not stimulated to excessive growth.

d. *The Rusts.* The rusts are caused by parasites (Uredinales) that are very similar to the powdery mildews in their parasitic ability. The two might be grouped together as obligate leaf parasites except that the rusts are even more highly specialized. In addition to physiological specialization of races for certain crop varieties there often is further specialization of the haploid and diploid stages for entirely different families of host plants. Heteroecism has been established as an essential aspect of the life cycle in many species. One can only conjecture as to how or why heteroecism came into existence and is actually obligatory for most of those species of rust which have no autocyclic stage.

The rusts have achieved remarkable progress in approaching a commensal relationship with the host. A hypersensitive reaction of the host cell that ends in its death is actually an immune reaction in this type of establishment. The more adept races of rust invade the tissue intercellularly without causing such injury. The haustorium or occasional intracellular mycelium approaches the nucleus or else attracts the nucleus to it so the two often adhere closely to each other. The parasite

may be suspected of either finding special nutrients at this center of cell activities or else stimulates host metabolism from this location.

The susceptible host cell enlarges rapidly and begins to undergo division so a new type of metabolically active tissue is formed that is composed of the fungus and host cells in a very compatible relationship. There is a change in the rate of respiration and in the respiratory quotient indicating that a new type of metabolism has come into existence. This is different from normal host activity (Samborski and Shaw, 1956). No one knows exactly what transpires but there is a definite change in the citric acid metabolism of the tissue and there is strong indication that succinic acid affects the respiration in uredospores of *Puccinia graminis* (Staples, 1957a,b). As the rust pustule ruptures the cuticle to expose uredospores or spermogonia, there may be considerable necrosis of the host cells at the base of the lesion in the more resistant varieties and there is an appreciable loss in the water economy of the host.

In most susceptible species the leaf tissue remains active and functional while the rust spores are developing. A gall may be produced when infection occurs in cortical meristematic tissues such as those of the stem of red cedar parasitized by *Gymnosporangium juniperi-virginianae.* In such disease establishments the parasite has been able to regulate the host cells, without otherwise injuring them, to such an extent that the pathogenesis could very readily be classified in the sixth (following) type of parasitism. This type of pathogenicity is preeminent in such rusts as *Peridermium* spp. on pine but tendencies in this direction are to be found throughout the Uredinales.

6. *Diversion of Foodstuffs to Abnormal Uses*

The interference with utilization of food materials is encountered in many types of parasitism such as those discussed above in the rust fungi. Certain types of pathogens have the ability to invade the host cell either directly or by means of haustoria and alter its metabolism most drastically. Such tissues are stimulated to abnormal uses of foodstuffs in synthesizing materials that cannot be of value to the host, since they contribute nothing to the host's essential functions. There are three conspicuous groups of parasites in this classification: the smuts, gall-forming parasites, and viroses.

a. *The Smuts.* The smuts would seem to belong in the class of gall diseases because the parasites, with very few exceptions, convert host tissue into tumors, then assimilate the tumor tissue. The physiology of corn smut caused by *Ustilago zeae* is representative of this class. The

chlamydospores produce sporidia that lodge in a leaf whorl, complete their sexual process, and invade the tissues by direct penetration. The mycelium becomes somewhat systemic as it infects nearby axillary buds and flowering organs before the internodes elongate. The various invaded tissues are not converted into smut galls until there is a generous supply of nourishment, according to Davis (1936). Because of this, galls are most commonly observed on the ear and lower portions of the stalk to which carbohydrates have been transported. The smut mycelium grows with the stimulated host tissue and eventually produces a mass of chlamydospores.

The process in the floral smuts is very similar to corn smut even to the point of the mycelium growing in the vegetative organs for relatively long periods without causing growth or necrosis in such diseases as bunt caused by *Tilletia tritici*. The state of restrained parasitism may endure for weeks or even months before the parasite becomes aggressively pathogenic. Pathogenesis awaits the proper stage of nutritional development in the host which, in this case, apparently occurs when the leaves begin to supply food materials to the young ovary. The food materials that would have enriched the endosperm and developed an embryo are diverted into nourishing the parasite and overgrown gall tissue.

b. *The Galls.* The gall diseases are incited by bacteria, lower fungi in the Chytridiales, and other fungi. The parasite apparently thrives by stimulating host metabolism. There is a richer pool of metabolites from which it can draw materials for its own growth and reproduction. As mentioned previously, the nodules produced on legumes by *Rhizobium leguminosarum* constitute a form of parasitism that definitely approaches commensalism.

The overgrowths caused by the crown gall and hairy root bacteria (*Agrobacterium tumefaciens* and *A. rhizogenes*) are special examples of this type of pathogenesis (Braun, 1947). The bacteria may multiply inside the host tissue without causing tumors but once a wound is created in the presence of growing bacteria the host cells proliferate a neoplastic tissue. The host must be exposed to the bacteria within a few hours after the wound is created but the bacteria may be eliminated later without jeopardizing tumor development. Under natural conditions, the bacteria may be restricted to the surface of the gall far removed from the site of hyperplasia. The exact nature of the growth incitant produced by the bacterium has not been determined but it is known to be thermolabile (Braun and Mandle, 1948) and the suggestion has been made that it is a polypeptide or nucleoprotein.

The principal feature of this gall development is that it seems to be definitely autocatalytic once the initial reaction is induced by the bacterium. The diseased tissue may be isolated in pure culture (White and Braun, 1941), separate from the bacteria. The aseptically grown tissues will induce a new gall when implanted in tissue of the host. This disease is almost unique in the annals of plant pathology in that the casual agent soon becomes incidental to the development of the disease. The processes of pathogenesis are autocatalytic and independent of parasitism after the initial period of induction.

There are several overgrowth diseases caused by obligate parasites related to the Chytridiales. These fungi penetrate a root either by way of the root hair or other tender epidermal cells or through a wound. Some produce an intracellular protoplast and others, the mycelial forms, send a haustorium into the cell. The host cell responds by enlarging and dividing. The naked protoplast in the host subdivides and continues its growth in the two new daughter cells so there is essential compatibility between host and pathogen.

Rather extensive studies have been made on the nutrition of crucifers affected with *Plasmodiophora brassicae*. For many years increasing the soil pH to 7.2 with lime was recommended as a preventive (Chupp, 1928) but there is good reason to suspect that the effect of the calcium ion was about as important as hydrogen ion concentration becauses the balance of calcium and potassium in the host cell will determine the severity of infection (Pryor, 1940; Gries *et al.*, 1944). Abundant supplies of potassium increase gall development and it has been shown by Palm and McNew (unpublished data) that potassium accumulates in the club tissue to such an extent that the normal growing points of the plant may be starved into inactivity. The potassium is essential for synthesis of inorganic nitrogen into amino acids and, undoubtedly, tissue rich in amino acids will promote the growth of both tumor cells and the plasmodium of the parasite.

c. *The Viral Diseases.* The viruses divert essential amino acids and nucleotides into synthesis of virus nucleoprotein. Insofar as known, these nucleoproteins can never be digested or converted into host protoplasm again even when the normal host tissue is being starved for nitrogen (Spencer, 1941). This loss of organic nitrogen to virus multiplication constitutes a drain on the host's metabolic processes.

There is much more to the pathogenic activities of the virus than the drain on nitrogen metabolism by the mere synthesis of nucleoproteins. A plant may support virus multiplication without any appreciable evidence of disease reaction. The most striking example is the so-called healthy or X virus of potatoes. It is essentially noninjurious to growth or physical appearance; but it accentuates the injury by other viruses in a synergistic fashion and it is virulent when transferred to other hosts.

This situation reminds one of the acquired resistance of tobacco for the ring spot virus. As described by Price (1932) and others, the established young leaves on an inoculated plant show the typical chlorotic oak-leaf pattern but leaves developed subsequently appear normal green. If the leaf primordia while still in the embryonic tissue are preconditioned by the virus, they do not produce symptoms even though the virus multiplies readily in them and is capable of causing symptoms on a new, unconditioned host.

It is now known that higher plants may synthesize heavy molecules of protein such as those resembling the tobacco mosaic virus (Takahashi and Ishii, 1952) without becoming diseased. The protein becomes viruliferous only when carrying the proper nucleic acids. One is forced to conclude that ability of the nucleoprotein to induce a disease reaction apparently resides in some secondary reaction not directly associated with abnormal synthesis of proteins.

There is one mechanism of pathogenesis by viruses that seems to be rather obvious. Many of them are known to interfere with the translocation of synthesized food materials. Phloem necrosis is very conspicuous in some of the virus diseases (curly top of beets and net necrosis of potatoes) and undoubtedly the presence of large molecules in the phloem tubes increases the viscosity of the phloem sap and may interfere with its passage through the sieve plate.

As shown by Bennett (1932, 1937), viruses move in the direction of translocation of synthesized foodstuffs to food-deficient areas. Therefore yellow mosaic virus in raspberry, for example, moves downward into the crown in the fall and upward into the young, actively growing shoot in the spring. Any interference by the virus would lead to inefficient metabolism as carbohydrates accumulated in the leaves or storage organs and could not be moved expeditiously to the growing tip or into storage as required. These interferences with movement of foodstuffs could justify a separate classification for some viruses but this has not been done because so little is known about the real nature of their pathogenesis either in phloem or parenchyma cells.

B. *Resumé of Natural Processes of Pathogenesis*

This brief resumé shows that there is a rather general correlation between the type of parasitism and the physiological function of the host that is interrupted. The causal agents range from facultative saprophytes, in which parasitism is incidental to their ordinary activities, to obligate or facultative parasites with commensal tendencies. Not much in the way of physiological specialization is required of a fungus or bacterium that digests a food reserve when it happens to lodge in a wound through the

protective covering. Even other forms of tissue that are actively growing may be destroyed by parasites that are only slightly more specialized.

However, when parasites begin to adjust their activities to special tissues such as the tracheal tubes or to send their cells into a living host without digesting the cell or otherwise poisoning its activities, they must have a tight control of their pathogenic activities. Finally, if the parasite in the cell supervises and directs the activities of that cell so that its own multiplication is enhanced, it must produce growth-regulating chemicals that induce new reactions or retard differentiation of cells into tissues. The alteration of host activities to the benefit of the parasite may be considered as an ultimate development in parasitism because it is only one step removed from commensalism in which two subjects share the same pool of metabolites without injuring each other. Few parasites, however, make a unique contribution to the partnership which would enhance the strength and vitality of the host so that the symbiotic state would be definitely superior to independent existence for both members.

As one progresses through this series of physiological disturbances caused by parasites, he becomes aware of certain basic changes in their habits. There are three well-marked trends. The first is suppression of a tendency to destroy cells by cytolytic enzymes. The second is a progressive change from indiscriminate general invasion of all cells to establishment between cells or intracellular penetration with haustoria to feed inside the cell while the major body resides outside and, finally, to a true intracellular existence where proteins of host and pathogen are freely associated. The third change is the growing tendency for the host cell to respond actively to the parasitic establishment and for the parasite to regulate this response. However, the parasite sacrifices autonomy when it prolongs and expands a favorable activity in the host.

The concepts of this section are based upon the assumption that there must be some set of basic principles behind pathogenesis. These seem to emerge as one begins to classify the physiological effects involved. If there is much validity to this arrangement, it should be possible to reinforce its concepts by interpreting, in its light, such things as the evolutionary processes that are operating in pathogenesis, and the response of the diseased state to changes in the nutrition and heredity of the host.

IV. The Evolution of Parasitism and Pathogenicity

From a strictly teliological viewpoint, a parasite has very little to gain from unbridled exercise of pathogenicity. If food procurement is so destructive that the host is eradicated, the parasite usually will have defeated its own purposes in life. The more compatible it can be with the host cell, the better are its chances of propagating itself indefinitely.

There is little advantage to be gained from making a plant body into a corpse that can be overrun by every saprophyte in the neighborhood.

Can evidence be found that parasitic specialization does improve and progress toward a state of mutual tolerance as outlined in the preceding section? Would it be possible for a soil saprophyte that ordinarily survives by digesting miscellaneous organic matter to become adapted by evolution to a specific host, to specialize its attack on certain tissues of the host, eventually to forsake the freedom of saprophytism and become so specially adapted to its host that it depended entirely upon it?

There is no ironclad evidence of such complete evolution, but anyone who has worked with plant pathogens knows that their virulence is a tenuous thing. Pure cultures usually become avirulent when maintained for long periods on culture media generously provided with all nutrients necessary for growth. There is ample evidence that new parasitic races may arise in culture under aseptic conditions or in the host through somatic mutation or segregation of factors for pathogenicity.

Fortunately, there are some observations that phytopathogenic bacteria may be arising under field conditions from saprophytes that first acquire parasitic ability and then proceed to develop chemicals to promote their pathogenesis. Furthermore, there is excellent experimental evidence on other species that host and parasite eventually establish a tolerable level of parasitism in which host and parasite can coexist. Unfortunately there is no comparable evidence of how parasites forsake saprophytism entirely or assume a compatible conjunctive relationship.

A. *The Tobacco Wildfire Group of Bacteria*

The wildfire of tobacco is caused by a green fluorescent bacterium known as *Pseudomonas tabaci.* Whenever weather conditions favor the dispersal of this pathogen by driving rain while the leaves are water-soaked, epiphytotics erupt with explosive damage. Somewhat less virulent in its effects on tobacco is a second pathogen known as *Pseudomonas angulata,* the cause of angular leaf spot. In addition to these two species, there is a third green fluorescent bacterium present in tobacco fields. It is the very widely distributed *Pseudomonas fluorescens* that is a general soil saprophyte in many areas both in the tobacco growing region and elsewhere. These species are normal soil inhabitants that multiply profusely in the vicinity of wheat and grass roots where they apparently find nourishment from root exudates (Valleau *et al.,* 1943).

There is good reason to suspect that these three species are variant strains of the same thing in spite of their different names. The only real substantial reason for giving them separate names is that they differ in pathogenicity. If the soil saprophyte *Pseudomonas fluorescens* is sprayed on a tobacco leaf, it does not invade the tissue. However, certain strains isolated from healthy-appearing leaves, will infect and cause lesions on weakened plants (Reid *et al.,* 1939). If *Pseudomonas angulata* is sprayed on a plant whose leaves have been preconditioned by exposure in a moist chamber, a necrotic spot is formed ranging from a minute speck less than 1 mm. in diameter to an angular spot bordered by veins surrounding the locus of invasion. *Pseudomonas angulata* is a reluctant parasite that invades leaf tissues readily only when they have been water-soaked. There are strains of this bacterium that are absolutely non-aggresive and will not invade the leaf tissue or cause lesions. These avirulent strains soon come to predominate in a culture of the pathogen maintained on nutrient agar. To all intents and purposes they are identical to *Pseudomonas fluorescens* in cultural and physiological characteristics (Reid *et al.,* 1942).

The wildfire organism causes a larger lesion than *Pseudomonas angulata* and it usually is bordered by a chlorotic halo ranging from 5 mm. to 2 cm. in diameter. This halo is due to the production of an exotoxin by *Pseudomonas tabaci* that has been identified as a new *a*-amino acid by Woolley *et al.* (1952a, b) who gave it the name tabtoxinine. This toxin weakens the tissue and predisposes it to more extensive invasion by the pathogen. Variant strains can be isolated from pure cultures of *Pseudomonas tabaci* that do not produce toxin. They are identical in all respects with *Pseudomonas angulata.* There can be little doubt that these two so-called species are identical because they have identical cultural characteristics, morphology, serology, and physiological properties according to Braun (1937).

Either with or without the toxin, the *Pseudomonas tabaci* complex is a poor parasite. The host must be preconditioned to infection by water-soaking the leaves as described by Clayton (1936). Leaves exposed to extreme root sap pressure from wet soils or water-logged by prolonged driving rains may be invaded extensively, particularly if the tissues have been weakened by inadequate supplies of potassium during their growth.

Thus we have in the tobacco fields of Kentucky, Pennsylvania, and undoubtedly elsewhere a complex mixture of green fluorescent bacteria. The omnipresent soil saprophyte is encouraged to grow by exudates from the roots of wheat, barley, and other plants. Undoubtedly if enough of them splash onto the lower surface of tobacco leaves injured by inclement weather, an occasional one might multiply (Reid *et al.,* 1942). After two or three host passages it should become aggressive enough to cause angular lesions on weak plants. Those strains that acquired the ability to produce toxin would have much more serious pathogenic

abilities. The sequence of changes is known to occur in reverse order so it is logical to assume that increase in virulence could be developed as outlined here.

B. *The Bacterial Wilt of Maize in Resistant Hosts*

Parasitism has not evolved beyond this stage in wildfire. The parasite is still a facultative saprophyte that destroys the tissue indiscriminately whenever the host is weakened by unfavorable weather and soil conditions. The next stage of progress can be illustrated by well-documented experiments with the sweet corn wilt bacterium (*Bacterium stewartii*) which shows a primitive ability to adjust its activities to its host.

This bacterium also produces variant strains in culture that may be either more or less virulent than the parent cell. One weakly virulent strain was found to have lost its ability to use inorganic nitrogen. Since the nitrogenous materials of the tracheal sap are primarily inorganic in nature, the loss of ability to reduce these materials to organic forms would handicap the survival and growth of the bacteria (McNew and Spencer, 1939).

When such weak strains are inoculated into the host in massive dosages, they rarely cause infection, but small chlorotic lesions may occur on an occasional leaf. The bacteria from such lesions will progressively gain in virulence as they are transferred from plant to plant. Eventually they become fully aggressive. These virulent progeny from avirulent strains simultaneously regain the ability to assimilate inorganic nitrogen. The proper interpretation of these data is that the avirulent bacteria have all the attributes necessary for virulence except ability to use the primary nitrogen sources available in the host tissue, and once the bacterium has developed the proper enzymes to reduce nitrate and ammonium ions, it attains a full complement of parasitic abilities.

It follows that if any progeny of this strain should acquire the ability to use inorganic nitrogen in culture it should be more virulent in the host. This was demonstrated in two types of experiments (McNew, 1938). When massive transfers were made from peptone agar to a mineral nutrient agar containing only inorganic nitrogen, an occasional colony began to grow. After several transfers on the inorganic nitrogen medium to fix this attribute firmly, all of the new cultures were found to be fully virulent for sweet corn. This demonstrates that organisms may gain virulence while in pure culture under the proper conditions.

A comparable evolution of parasitism was obtained on organic nitrogen medium by mechanically segregating variant strains as they appear by dispersing the cells in peptone-beef agar dilution plates, isolating the colonies, and testing subcultures of each for virulence. The process of selection was repeated with the most virulent member. After four such mechanical selections, a fully virulent strain was isolated from cultures maintained constantly on peptone agar. The virulent strain had concomitantly gained ability to assimilate inorganic nitrogen. The simultaneous restoration of virulence and ability to use inorganic nitrogen was due to selection of natural mutants rather than to development of adaptive enzymes since the parent avirulent type and its progeny had never been exposed to either the host environment or inorganic nitrogen in medium before the final test for virulence was made.

Undoubtedly there are dozens of attributes such as ability to use inorganic nitrogen that are prerequisites for parasitic establishment in the tracheal tubes. The resistant varieties of maize may be suspected of having some specific difference in physiology that prevents full parasitic development. If so, bacterial cultures injected into hosts with different resistance would be exposed to different selective environmental pressures. When a weakly virulent culture is injected into a susceptible host it ordinarily gains in virulence as is witnessed by the example just discussed; but would the highest state of virulence be attained in the most susceptible variety of maize? When Wellhausen (1935, 1937) investigated this by making serial passages through resistant and susceptible inbred lines of maize he found that the bacteria from the more resistant lines were more virulent than those from susceptible lines when tested for virulence on the same host. Extensive tests with several different cultures prove that each of these in a given line of maize attained a comparable maximum degree of virulence after nine or ten serial passages and remained constant thereafter depending on the innate resistance of the host. Wellhausen (1937) and Lincoln (1940) were able to demonstrate that the more virulent cultures from resistant lines were readily attenuated by serial passage through a more susceptible line. Irrespective of initial virulence of the culture, they came to have essentially identical ability to cause wilt when maintained in a host of specified resistance.

C. *Variations in Michigan Wilt of Tomato*

There is a limit to the operation of this principle of host-regulated virulence. When different species of hosts for *Corynebacterium michiganense* were inoculated with a highly virulent culture isolated from *Hyoscyamus niger*, virulence changed very slowly (McNew, 1938). Subcultures transferred serially in very susceptible hosts such as *H. niger*, *Nicotiana glutinosa*, and *Lycopersicum esculentum* maintained a high degree of virulence without developing physiological specialization. The same culture maintained in very resistant *Phaseolus vulgaris* gained

only slightly in virulence for this host while losing some of its virulence for the other hosts. Thus the principle laid down for *B. stewartii* in inbred lines of maize does not apply to *C. michiganense* in different species of plants. This is in general agreement with Wellhausen's observation (1937) that *B. stewartii* does not gain in virulence for maize when cultured in resistant grasses.

The data on *B. stewartii* are probably indicative of what goes on in nature over a long period of time. As deduced by Zinsser and Wilson (1932), the rise and fall of epidemics may be determined by dissociation of the causative agent to give less virulent strains. There is no evidence that virulence increases indefinitely among animal hosts and eventually the less virulent variants will dilute the inoculum potential of a culture. There are good statistical reasons why these weaker forms eventually would reduce the severity of an epidemic.

There are two divergent influences in the epidemic of a plant parasite that could make its course differ from that in animals. There is very little evidence that antibodies develop in plants to give a true immune reaction as in animals. The nearest approach to this is the immune reaction or recovery of certain plants such as tobacco from ring spot virus as discussed in the preceding section.

The second major difference between animal and plant diseases is that the health of the individual plant is of less concern than the fate of the entire population. Population genetics change more rapidly in plants so the natural laws of selection and adjustment to epidemics are more obvious as described below.

V. The Law of Host-Parasite Balance in Pathogenesis

The host population in most species of plants has a diversity of genetic material. In the natural state this provides plants that are likely to differ in their reaction to parasites. In the course of a severe epidemic, the more susceptible members are destroyed or so seriously handicapped that they do not reproduce. This automatically increases the resistance of the entire population for that particular disease.

The end result is that the host may become adjusted to each change in virulence of the parasite. This means that the host and parasite establish a balance or coexistence at a higher level of parasitic tolerance for each change in the parasite unless there is a mechanism for attenuating the parasite as described above for *Bacterium stewartii*. Undoubtedly if there is a loss in virulence, more susceptible types of host would survive and the host-parasite balance would be established at a lower level of parasitism. This process of readjusting resistance and virulence to a balanced state might be designated as the natural law of survival in pathogenesis.

A. *The Races of Wheat Rust*

The existence of this law can be demonstrated from studying some of the things that man has done to change the host-parasite balance. Much has been learned about what can be done—and what should not be done—in changing the natural balance that parasites and hosts have been able to achieve over the centuries. An excellent model for demonstrating the law may be seen in the breeding of wheat for resistance to *Puccinia graminis* in the United States.

The stem rust susceptible varieties of 1910 such as Bluestem and Fife were replaced by the rust-escaping Marquis. The rust epidemic of 1916 drove farmers to replace Marquis with the durum wheats. These in turn were susceptible to several races of rust of which No. 11 was outstanding. A cross of the varieties Marquis by Kota produced the variety Ceres which was immune to the races of black stem rust prevalent from 1926 to 1934. However, a new race designated as No. 56 which would attack Ceres was identified as early as 1928 and became prevalent by 1934. It reached severe proportions in 1935 and 1937 and sealed the fate of the variety Ceres. Losses of 160,000,000 bushels of wheat occurred in the United States during 1935.

Ceres was replaced by the variety Thatcher which had withstood the epidemic of race 56 in 1935. Thatcher, Hope, and hybrids of them were widely used in subsequent years. These varieties contained a combination of genes for resistance derived from varieties of durum, emmer, and flour wheats which was adequate to control the prevalent races of stem rust (17, 19, 38, and 56). Race 15B was discovered in 1939 and gradually increased in the next few years. Its biotype 15B2 swept over much of North America in 1950 since it could attack resistant durums. By 1953 and 1954 the macaroni wheat varieties had been virtually annihilated and the plant breeders had to turn their attention to use of resistance obtained from varieties in Kenya.

In looking back over the past 50 years, one is impressed that the germ plasms of *Puccinia* and *Triticum* have been playing tag with each other. As soon as a new set of genes for resistance was introduced into the crop, the parasite developed a new race for virulence. In only 20 of the 50 years between the great epidemics of 1904 and 1954 was wheat adequately protected from *Puccinia graminis* on the great plains of North America according to Stakman and Harrar (1957, p. 507).

At each step along the way, man speeded up the processes of evolu-

tion by seeking out the sources of resistance around the world and introducing the genes into the commercial crop. Very shortly a new physiological race of the parasite would come into prominence and restore the parasitic balance in favor of the pathogen. Then the plant breeder reacted, and so on, *ad infinitum*. Unfortunately for the crop, its custodian runs a poor second in this race because rust operates on a 14-day cycle and the breeder on a 7- to 10-year cycle for propagation of a new variety.

Plant parasites that get out of control and violate the law of natural balance are the exception rather than the rule. Usually this has occurred in modern history (the past 200 years) only when man has been primarily at fault. Unfortunately men are careless and often prone to make mistakes. There are four common blunders against the law of natural balance in pathogenesis which set the stage for devastation by plant parasites.

B. *The Role of Introduced Parasites*

The most serious offense is the introduction of a virulent parasite into an unconditioned population of hosts. If the population has never had an opportunity to participate in the evolution of pathogenesis it is completely at the mercy of the parasite. We need to cite only a few examples. The introduction of downy mildew of grape caused by *Plasmopara viticola* into the vineyards of Medoc, France, from the United States was a national catastrophe. The importation of chestnut blight caused by *Endothia parasitica* into the United States, presumably from China, deprived an entire nation of one of its most valued forest trees. Never has a pathogen created such havoc on a defenseless population over such an acreage of land. About 50,000,000 acres of chestnut forest were eradicated because resistance of the host had not evolved to the same level as the parasitism as its enemy.

The citrus industry of this country escaped a comparable ravage from citrus canker caused by *Xanthomonas citri* only by taking drastic action when it was introduced from the Orient. Groves extending over thousands of acres had to be burned in order to eradicate the parasite. The downy mildew of hops caused by *Peronospora humuli* was introduced into England from Japan in 1917. Within 5 years, it became the limiting factor in hop culture in western Europe.

C. *The Attack on Introduced Plants*

A second mistake that man has made repeatedly in upsetting the disease balance in crops is to introduce a new crop into an established population of parasites. Although the results are not so disastrous economically as the reverse situation, the results are no less sensational. The American colonists soon found that their favorite table and wine grapes from Europe would not grow in the New World. The logical assumption was that they were not adapted to the climate. Today we know that they were wiped out spontaneously by the downy mildew disease which went to them from the native wild grape.

A similar situation was encountered when the superior varieties of *Hevea* rubber trees were introduced into South America from the Orient. They were quickly eradicated by the leaf blight fungus, *Dothidella ulei*, that was indigenous to this area. In the 75 years that *Hevea* had been cultivated and bred for better yield in the Orient, it had lost all resistance to this pathogen. The wild types in Brazil, from which the new varieties had been derived, had maintained their resistance because they had been exposed continuously to infection pressure and natural selection.

When the American colonists settled along the Atlantic seaboard in the 17th century they found that the wild apples, crabapples, and hawthorns were infected with fire blight caused by *Erwinia amylovora*. According to the accounts by Cox, the trees on the banks of the Hudson had mild infection that caused the loss of an occasional branch but the disease seemed to fluctuate around a mild endemic level. However, when the horticulturally superior pears of France and Western Europe were introduced, they were literally eradicated. As men began to assemble select varieties of apples into orchards where the disease could spread easily, fire blight became a chronic problem. Evolution of the parasite continued while man held the evolution of the host static under conditions ideal for infection.

Apparently as long as *Malus* spp. and other rosaceous hosts were distributed in a general plant society, *Erwinia amylovora* was restricted in its spread. Some trees were undoubtedly destroyed but the more tolerant ones escaped destruction and reproduced their kind of resistant progeny. If a virulent new strain of the bacterium arose, its spread was hampered sufficiently by mixed plantings to prevent eradication of the hosts before a new type of resistance could be evolved. Thus the host and parasite had come into a normal balance before man upset the scheme of things by introducing new hosts that had never been exposed to natural selection by infection and trees were crowded into orchards where conditions favored the parasite.

D. *The Aftermath of Inadequate Plant Breeding*

Man also upsets the balance of resistance-pathogenicity by efforts to breed superior crops. For many years the unscientific horticulturist selected new varieties for yield, quality of produce, and ecological

adaptability. If pathogens were not present so that varieties would be chosen automatically for disease resistance, the breeder could, and often did, lose all innate disease resistance just as the breeders of rubber trees did in the Eastern Hemisphere.

There was enough heterozygosity left, however, in the open-pollinated field crops to keep the new varieties from being exposed to the full force of epidemics. Any infection on a susceptible plant might be checked or handicapped by the more resistant members in the mixed population. Often this was not true in the vegetatively propagated fruit crops; so diseases of orchards, rubber, banana, and fruit plantations became uniformly destructive and demanded special disease control measures. The breeders of field and vegetable crops were somewhat slower in reducing their crops to completely uniform, susceptible populations. They had to learn how to develop pure lines by inbreeding and subsequent hybridization in order to make every plant in a field uniformly susceptible or uniformly resistant.

In the period 1890–1910, Orton and others demonstrated that varieties could be bred for resistance by selection and hybridization. This is one of the greater gifts to the welfare of mankind. After the force of hybrid vigor had been exploited in the era 1930–1940, it was obvious that the greatest remaining improvement which could be made in many crop varieties was to improve their resistance to disease.

This has not proved to be as easy as it sounds. As indicated by the story on breeding wheat for rust resistance, the plant parasites are versatile adversaries, and much more skill is required than simple hybridizing and backcrossing. In some instances serious problems have arisen from breeding for disease resistance because uniform resistance to one parasite may mean uniform susceptibility to another one.

E. *The Victoria Oat*

This is well illustrated among the otherwise remarkable achievements during the past 30 years in breeding oats for resistance to crown rust caused by *Puccinia coronata*. By hybridizing and selection, the old unimproved varieties were given stem rust resistance from the varieties White Tartar and Richland and smut resistance from Black Mesdag. In order to cope with the changing population of crown rust, the resistance of Victoria was bred into this synthetic variety. The superior varieties obtained from this cross swept into popularity and dominated oat culture in the United States during the period 1942–1948.

The genes from Victoria induced a hypersensitive reaction to invasion by all biotypes of *P. coronata*. As soon as the leaf tissue was invaded, the adjacent cells died and the infection was destroyed before

spores could be formed. Host injury was restricted to a small fleck. This reaction which is equivalent to immunity apparently set the stage for the development of the seedling blight caused by *Helminthosporium victoriae*. This fungus was a minor parasite on grasses and it had never attacked enough oats to attract attention. However, it is a facultative saprophyte that thrives on dead tissue, so, apparently, the hypersensitive reaction of Victoria-like varieties promoted its parasitism. The *Helminthosporium* blight gained momentum and within 3 years after the fungus was named it had become a major factor in oat production. Yields were being reduced by 25 to 50% wherever weather conditions were favorable to fungous development.

Fortunately, other forms of resistance to crown rust were available in the variety Bond. New varieties were developed to replace the Victoria type within 5 years but, as predicted by H. C. Murphy, the varieties were soon attacked by a new race of rust that came into prominence as soon as the Bond varieties were used extensively. These in turn were soon replaced by new varieties that had derived their resistance from the varieties Sante Fe and Landhaufer.

Nowhere in the annals of plant pathology is there a better illustration of the evolution of pathogenicity toward a nominal host-parasite balance than in this story of man's struggle with the fungi for the oat crop of North America. We have witnessed in 3 decades a process by which man speeded up evolution many hundredfold.

Thousands of years might have elapsed while germ plasm of host was matched against germ plasm of the parasites had not man intervened. He brought an assortment of oat germ plasm from Australia, South America, Russia, etc., into an arena where the two types of parasites could operate freely. As soon as one gained ascendancy over the host, the variety was removed before it was eradicated. New germ plasm was introduced just as would have been done inevitably, but much more slowly, under natural conditions.

Man's role was to create ideal conditions for epidemics by planting millions of acres of oats tightly packed with uniformly resistant—and uniformly susceptible—plant material. The pathogens were uniformly handicapped at some times, and uniformly successful at others. New germ plasm was fed into the arena as it was needed. In short, man was forced to speed up the processes of evolution to maintain his food supply in this generation. At present he has everything under control but no one knows how long it will be before the next stage of evolution will present itself.

The lesson to be derived from this experience is that the art of breeding for disease resistance depends more on an understanding of patho-

genesis and the nature of parasitism than it does on genetic manipulation. There is nothing so profound in the processes of inbreeding, hybridizing, and backcrossing that any technician could not master them in a few months. The real problem is to estimate what course evolution of the parasite is likely to follow and what hazards of pathogenicity are likely to be encountered after every genetic change in the host.

This brief account of the evolutionary processes by which parasites come into balance with host resistance and the disasters to which man has exposed his favorite crops by meddling, makes one wonder whether it is safe to breed forest trees for resistance. Until much more is known about the nature of their diseases and the genetic potentials for evolution of their parasites, extreme caution must be observed.

What might happen, for example, if every white pine tree in the United States was uniformly homozygous for resistance to *Cronartium ribicola*? If a new race of white pine blister rust arose spontaneously, every tree might be uniformly susceptible. The disease would sweep across young forest plantations like wildfire wherever there are wild currants. At least 75 to 125 years would have to elapse before a new resistant line could be developed and put into commercial production. The disaster to an annual crop such as oats described above would be nearly fifty times as difficult to correct technically and probably would be even more serious economically for forest trees.

The least that should be done is to forbid widespread planting of any one line of forest trees. Blocks or mixed plantings of trees with different hereditary attributes should be used. Furthermore, there is great need in breeding trees to find the best possible means of transferring one or two genes for resistance without reducing genetic heterogeneity for other attributes in any way.

VI. The Effect of Environment on Different Classes of Parasitism

By classifying plant diseases into the six physiological classes according to their effect on the host, an elderly arrangement of plant diseases has been obtained. It is obvious from the discussion in the preceding section that certain forces operate to cause an upgrading of parasitic ability. There are very substantial data to prove how the parasitic way of life can be developed from saprophytism to give representatives of the three lower classes of pathogens. There is also appreciable circumstantial evidence of evolution in the higher classes in which the forces of parasitism and host resistance can be kept in balance even as virulence increases or decreases.

The physiological basis of classifying diseases should be valuable in understanding how environmental changes would influence the balance between host and parasite. The changes in soil and air would be expected to affect the different classes of parasites differently because their basic relationships to their respective hosts are different. Those parasites that specialize in a saprophytic existence apart from their hosts would be more directly affected by the change while those that are highly dependent or obligately dependent upon the host might be affected more indirectly through the host's response.

Those parasites that prosper on very actively growing, metabolically active cells should be most severe on the well-nourished vigorous plants. On the other hand, the facultative saprophyte that attacks only the injured or weak plant might be expected to be most destructive on plants grown under adverse conditions, particularly if those conditions were also conducive to its own growth and multiplication on or near the host. A brief review of the effect of weather and soil fertility will serve to substantiate these ideas.

A. *The Effects of Weather*

The facultative saprophytes that cause the first three classes of disease (the storage rots, the seedling diseases, and the root rots) are almost entirely dependent on the environment. A modest change in the ventilation or temperature of an apple or sweet potato storage will prevent *Penicillium* rot and black rot infection, respectively. Only a slight increase in soil moisture content is necessary to cause pea seed decay by *Pythium ultimum*. If there is free water in and on the soil for a day or two, the emergence of seedlings may be reduced 60% or more, but if the moisture content remains at about 30 to 50% of water-holding capacity, a perfect stand will be obtained. Lima bean seed, on the other hand, are destroyed much more readily by this same fungus if development of the seedling is handicapped by cold soils for a few days. A cool weather crop such as the pea is rendered more susceptible by water than by a drop in temperature while the reverse is true in the warm weather crop such as the lima bean.

The severity of *Aphanomyces* root rot of peas can be accentuated appreciably by reduction in soil temperature while the *Fusarium* root rot of the same crop can be very severe in warm seasons. Minor changes in the hydrogen ion concentration of soil will alter the survival and parasitism of such soil inhabitants as *Pseudomonas solanacearum* on potatoes and *Thielaviopsis basicola* on tobacco. The addition of nitrogenous materials to soil will reduce the survival of *Ophiobolus graminis*.

Many soil saprophytes are destroyed or inhibited by microbial antagonists that can be stimulated by addition of organic matter in the form of animal manure, green manure, or even sugar.

These various facts are well recognized and are commonly used in controlling the poorly specialized parasites that live in the soil. Every effort is made to reduce their incidence or to handicap their development by various means. Disease-escaping practices such as sanitation, crop rotation, planting on selected dates, and choice of fields that are unfavorable to the pathogen are widely recommended.

Analogous procedures are also used for some of the leaf blight parasites that are poorly specialized although less use is made of environmental control except to insert toxic spray materials into the scheme.

Comparable precautions are rarely emphasized in controlling the more specialized parasites of the leaf. The genetics and growth of the host determine the aggressiveness of the parasite. The viral diseases and rusts are exceptionally destructive on succulent, rapidly growing plants.

B. *The Effects of Mineral Nutrition*

The mineral nutrition of plants may have a marked effect on the severity of their diseases. Although few diseases are completely controlled by varying the soil fertility, the damage from the pathogen may be ameliorated appreciably by adjustment of the balance and total quantity of nutrient elements in the soil.

If diseases are considered *in toto* there seems to be very little logic to the effects of various nutrients when the data available in some 700 reports in the literature of this subject are brought together. For example, the reports on nitrogen supply show that a deficiency of this element decreases the severity of 19 diseases and increases it in 11. On the other hand, a generous supply of nitrogen decreases the injury in 32 diseases but increases it in 58 others. About the only conclusion that can be formulated is that excess nitrogen probably should be avoided but every disease has to be considered as a special case. The same confusion exists when the responses of various diseases to potassium or phosphorus are listed.

If, however, mineral nutrition is considered in terms of the six physiological classes of diseases, a reasonable degree of order can be established. There are certain effects from mineral nutrition because disease production by the parasite and recovery of the host are physiological processes dependent upon nutrition.

Usually there are certain balances in nutrient elements that dominate the disease response of the plant. For example, an increase in phosphorus supply in soils reduces the severity of the take-all disease of wheat caused by *Ophiobolus graminis* in Kansas and Australia while an increase of nitrogen often has the same effect in Canada and England (see review in McNew, 1953). The two elements must be balanced in order to promote quick recovery of infected plants. The type of nutritional balance required varies most remarkably with each class of pathogens as briefly summarized below.

1. *Destruction of Food Reserves*

The storage rots and decay diseases that destroy the food reserves, are caused by the facultative saprophytes, are only moderately affected by the type of soil fertility prevailing at the time the crop produced the fruit, seed, or other storage organs. However, plants supplied with an excess of nitrogen do produce a crop more susceptible to decay. Careful study on the storage rots of apples in Northern Ireland and New Zealand have shown that fruit from heavily manured trees are more seriously injured by fungal pathogens than from those grown on untreated trees. The fruits from nitrogen-fed trees are more nourishing to *Cytosporina ludibunda* as can be demonstrated by removing sections aseptically and inoculating them in culture dishes.

Moderate applications of potassium around apple trees produce a firmer fruit less susceptible to mechanical injury and consequently to decay but an excess of potassium promotes physiological breakdown of the tissues. The general conclusion seems to be that an excess of nitrogen renders the fruit more nutritious to the pathogens. The effect of nitrogen is strictly on the growth rate of the pathogen in the presence of an unlimited supply of carbohydrates since there is no evidence that the host enters into a counter-reaction or growth response of any sort. In this type of disease, nitrogen or nitrogen-potassium balance is the predominant factor in determining host invasion.

2 and 3. *Prevention of Seedling Metabolism and Procurement*

The seedling diseases and root rots are definitely affected by soil fertility. An excess of nitrogen increases susceptibility to invasion of the plant tissues by the soil inhabitants with low grade to average pathogenicity traits. Apparently an excess of nitrogen either injures the seed and seedling or prolongs the juvenile condition of susceptible tissues that normally become more resistant with maturity.

The balance between nitrogen and phosphorus is very definitely important in such diseases as take-all, foot rot, and *Pythium* root rot of wheat. The proper balance assures rapid regrowth of roots from the crown of infected plants (Garrett, 1948) so as to assure a quick recovery from loss of diseased roots. The phosphate fertilizers are no less important

in reducing the infection of sugar beet seedlings by *Aphanomyces cochlioides* (Afanasiev and Morris, 1949). By speeding up growth so the seedlings can rapidly pass into the resistant stage, losses in stand are reduced and yields are increased threefold. A comparable effect of nutrient balance has been demonstrated for Texas root rot. One may conclude that nitrogen-phosphorus balance is important in seedling diseases and root rots because it permits maximum growth of roots and rapid recovery from these low grade but very destructive pathogens.

4. *Interference with Upward Transport*

The wilt diseases, that affect movement of water and nutrients on the other hand, are much more affected by the balance of nitrogen and potassium than by nitrogen and phosphorus. An excess of nitrogen stimulates the severity of wilting and a deficiency reduces wilting apparently because of the attendant succulence of the tissue and because a tracheal sap rich in nitrogen is a more acceptable culture medium for the pathogen (McNew and Spencer, 1939) than a sap poor in nitrogen. This situation holds for the fusarial wilts of cotton and tomato, the bacterial wilt of maize, and comparable diseases.

If the plant is provided optimum supplies of nitrogen, its susceptibility is decreased by additional potassium supplies. The most logical explanation is that adequate supplies of potassium must be available to promote the utilization of available nitrogen in the processes of host growth (Shear and Wingard, 1944). This prevents accumulation of extra nitrogen that could be quite useful in promoting the growth of the parasite in the tracheal tube. Phosphorus increases the severity of infection only slightly and essentially has no effect on wilting until it is provided in excessive supply as was shown for bacterial wilt of maize (Spencer and McNew, 1938).

5. *Destruction of Food Manufacture*

a. *The Leaf Blights, Cankers, and Rots.* The leaf blights and cankers are rendered more destructive by nitrogen fertilization except for a few examples such as the bacterial shot hole of peaches caused by *Xanthomonas pruni* where nitrogen retards abscission formation and helps prevent defoliation of infected leaves. There is also some skimpy evidence that nitrogen fertilization may prevent extreme severity of early blight on tomatoes infected with *Alternaria solani.*

The more common experience, however, is for nitrogen fertilizers to increase susceptibility to invasion by parasites such as *Erwinia amylovora* on branches, foliage, and flowers of pear and apple, or *Pseudomonas tabaci* in tobacco wildfire. The use of moderate supplies

of nitrogen and adequate supplies of potassium leads to firmer tissue in leaves, stems, and fruits so these organs are less severely invaded. The outstanding example is the use of potassium to prevent or reduce the susceptibility of tobacco leaves to water-logging of tissues, a major prerequisite to extensive invasion by the wildfire bacterium.

c. *The Powdery Mildews.* Powdery mildew infection is handicapped by cells that have thick, tough cell walls. Because of this, plants are rendered susceptible by heavy fertilization with nitrogen (Stuch, 1926). As a general rule, plants receiving generous allotments of potassium and phosphorus are much less severely infected. Potassium is particularly important and a few investigators have reported that a combination of potassium and silicates speeds up the hardening of leaf cells so the mildew fungi are less invasive.

d. *The Rusts.* The rusts of cereal crops are more severe on plants supplied with nitrogen. Moderately resistant varieties of wheat, rye, and oats may be rendered susceptible by depriving them of potassium while their resistance may be increased by adding potassium to the nutrient medium according to German investigators (Gassner and Franke, 1934; Gassner and Hassebrauk, 1933). Gassner and Hassebrauk (1931, 1934) explain these effects by the changes in amount of albumen in the leaf tissues since this is increased either by addition of nitrogen or removal of potassium from the nutrient supply. However, this is probably only one facet of the problem, since wheat plants provided with additional nitrogen will produce a toxin for *Puccinia glumarum* at a more rapid rate than normal.

6. *Diversion of Foodstuff to Abnormal Uses*

b. *The Galls.* The galls are affected most conspicuously by the balance between potassium and calcium. There must be adequate supplies of nitrogen and phosphorus to support cell division in the tumor just as in normal tissue so a deficit of these materials will suppress the severity of club root of crucifers, crown gall of tomato, bunt of wheat, and common scab of potatoes. However, if these two nutrients are available in adequate supply for host growth, additional amounts have only moderate effects compared to potassium and calcium.

There has been much confusion about adding lime to soils for club root control and use of acid-forming materials for potato scab because attention was focused on soil reaction rather than the change in nutrient balance in the host. The hydrogen ion concentration of the soil solution is not the sole factor operating because severe club root of cabbage may occur in soils at pH 7.0 to 8.2.

The amount of calcium ions available and, even more, its balance

with potassium determine the severity of the growth response to invasion. Contrary to what was observed in the wilt and rot diseases, an increase in supply of potassium promotes gall growth. As a matter of fact, a large club on the main taproot of a cabbage or turnip plant will accumulate so much potassium that the remainder of the plant may be deprived of supplies adequate for normal growth. This situation is readily understood when it is realized that the potassium is indispensable for conversion of inorganic nitrogen into amino acids. The calcium is necessary for spindle formation and other processes involved in cell division and in the formation of new cell walls. Again there is a very sound reason why this particular nutrient balance has proved so consistently important when the nature of the pathogenesis is given full consideration.

c. *The Viral Diseases.* In the viral diseases that have been studied so far there is a direct correlation between the supplies of nitrogen made available to the host, the severity of symptoms, interference with growth, and multiplication of the virus. There are considerable conflicting data on how the heavyweight virus protein is formed and acquires the ribonucleic acid that gives it virus properties. The plant apparently does not reallocate nitrogenous materials from its tissues to the virus when inorganic nitrogen is in short supply. The nitrogen from the soil is incorporated directly in the precursors of the nucleoprotein so a shortage of nitrogen hampers virus multiplication (Spencer, 1941; Bawden and Kassanis, 1950).

In addition to nitrogen, the supply of phosphorus affects the severity of virus reaction in plants. The tumor tissue of sorrel incited by the clover wound tumor virus has a phosphorus requirement several times as large as that of normal tissues. These effects appear logical since phosphorus is essential for the heavier energy demands of the cell and the formation of nucleotides used in the synthesis of nucleic acids.

This very brief condensation of over 700 original reports may be an oversimplification of the effect of soil fertility. However, it does show that there is a logic to the results being obtained on the effects of host nutrition on plant diseases. When the diseases are grouped according to the physiological basis of their parasitism, the basic effects become apparent.

VII. Host Genetics in Relation to Type of Pathogenicity

The employment of disease-resistant varieties has come to be accepted as one of the cheaper and more effective methods of avoiding the ravages of plant pathogens. The method has become standard for controlling many of the highly specialized parasites such as the rust and powdery mildew fungi. It has been possible to synthesize varieties

of cereals, for example, that are highly resistant to specific races of the obligate parasites.

With such records of success before them, enthusiastic novices in plant breeding often assume that all plant diseases should be controlled by use of resistant varieties. They soon discover that resistance to a fruit rot, for example, is harder to achieve than resistance to a rust, and, once resistance is found, it is much more difficult to transfer into a commercial variety by hybridization and backcrossing. Are there any principles that could guide the plant pathologist in making a decision as to whether breeding should be preferred over sanitation, crop escaping, or preventive sprays for disease control? There appear to be such principles because the inheritance of resistance by the host seems to vary with the six degrees of specialization involved in the parasitic processes. This interesting relationship appears worthy of discussion.

1. *Destruction of Food Reserves*

The storage organ rots and decays of the food reserves have rarely been controlled by use of resistant varieties. As emphasized above, these diseases are essentially digestive processes of saprophytes whereby a tremendous reserve of readily available carbohydrates is attacked as soon as the protective covering is injured. About the only chance of securing a more resistant variety is to find one with either a thicker skin or firmer flesh so that it is not wounded so easily. An alternative possibility is to find a variety with a toxin in the tissue that can inhibit the pathogen.

If such sorts of resistance for storage and transit rots are found, it is very likely to impart such undesirable quality to the produce that it will destroy its horticultural value. These attributes, if they did occur and were acceptable, are the product of general growth phenomena that undoubtedly are regulated by scores of genes. The problem of transferring such genes in mass and eliminating all genes that contribute toward susceptibility is a tremendous undertaking. In this type of disease, the advantages rest with the unspecialized parasite that is versatile in its food acceptance and has no special system of establishing itself on an actively growing cell.

2 and 3. *Prevention of Seedling Metabolism and Procurement*

The root rots and seedling diseases are comparable to the storage organ rots in the general omnivorous nature of the parasites, but they differ in that they do attack and destroy living cells. They usually invade through wounds, but they may also form mycelial masses on the plant surfaces and breach the epidermis by pressure or digestive enzymes such as is shown by *Thielaviopsis basicola* on tobacco.

Resistance can be found in mechanical strength of the tissues or the presence of toxic materials such as the phenolic and alkaloid materials in certain crops resistant to Texas root rot (Greathouse and Rigler, 1940). In addition, the plant, even though invaded, may escape destruction by replacing injured tissue. Many maize seedlings have the epicotyl completely destroyed by *Diplodia zeae,* operating from either the seed or the soil; but the timely establishment of a permanent crown of roots from the lower nodes of the stem compensates for the loss of water and nutrients normally provided by the primary root system. "Resistance" of some strains of tobacco to black root rot is almost entirely achieved by prompt development of roots to replace those destroyed by the fungus during periods of weather favorable to the parasitic establishment.

These types of "growth" resistance are regulated by many genes and resistance of this sort is rarely transferred readily from resistant to susceptible lines. There may be two or three major genes for resistance but invariably their effects are modified by a battery of secondary genes, the number of which is so complex that they are rarely determined by the breeder.

4. *Interference with Upward Transport*

The situation changes drastically when one progresses from these relatively nonspecialized soil inhabitors to the more specialized parasites of the transport system. These pathogens have fairly well-defined nutritional requirements which are met best by the environment of the tracheal system. However, in choosing this more selective, specialized environment they automatically have sacrificed some of their freedom of action, i.e., ability to accept wide deviations in the qualitative and quantitative condition of the nutrients in the host. Their fate can be determined by a relatively small change in the nature of the host cell and host metabolism.

It is not surprising, therefore, that most crops attacked by vascular parasites have different degrees of resistance ranging from complete susceptibility through tolerance to near immunity in different varieties. The number of wilt diseases controlled by use of these resistant varieties is legion: sweet corn wilt caused by *Bacterium stewartii;* the wilts of cotton, tomato, peas, bean, cantaloupe, watermelon, pepper, cabbage, etc. caused by *Fusarium* spp.; and even *Graphium ulmi* on elm.

The breeding of this resistance into commercially desirable lines presents no insurmountable problem as a general rule. Usually resistance of this sort is inherited through a relatively few genes (two to six). By reasonable diligence, the resistant hybrid can be backcrossed to the susceptible commercial parent to fix desirable agronomic and horticultural traits, while retaining the major genes for resistance. One advantage is that the wilt parasites apparently are not so prone to develop a multitude of parasitic races as the obligate parasites because they do have an existence apart from their hosts during part of their life cycle.

5. *Destruction of Food Manufacture*

a. *The Leaf Blights, Cankers, and Rots.* The situation in *the leaf blights, cankers, and rots* is highly confusing because of the diversity of the fungi and bacteria involved. Some of them are omnivorous, facultative saprophytes much like the root rot fungi. They attack injured tissues, have no special aptitude either in initiating an attack or in establishing suitable liaison with the functional cells of the host. Among these are many species of *Alternaria, Septoria, Stemphylium, Pseudomonas,* and *Xanthomonas.* Others such as species of *Cercospora, Colletotrichum,* and *Venturia* have well-developed parasitic patterns including the production of parasitic races with very specific varietal preference. Breeding for resistance in this group is successful almost directly in proportion to the degree of parasitic specialization exhibited. Breeding is quite rewarding in the latter group but relatively unimportant in the less specialized types.

b. *The Downy Mildews.* The downy mildew fungi, in general, have very well-developed parasitic capabilities ranging from facultative to obligate parasitism. Resistance is relatively easy to develop and transmit by breeding. The outstanding example is the development of varieties of potatoes resistant to *Phytophthora infestans* by transfer of four or possibly five genes from *Solanum demissum* to *S. tuberosum.* The most recently developed resistant varieties constitute about the fifth time that the late blight problem has been "completely solved" by breeding in North America and in Europe. Resistance has been quite ephemeral merely because the fungus has great ability to produce new parasitic races. Currently there is evidence that resistance being developed in Mexico may be more enduring (Niederhauser, 1956). Resistance to this general class of parasites seems to be regulated by a relatively few major genes, since only one to three are required for resistance to a single biotype of the fungus.

c. and d. *The Powdery Mildews and Rusts. The obligate parasites* of the Erysiphaceae and Uredinales have highly specialized parasitic races. The physiological relationship of host and parasite is so delicately balanced that a small change in the host protoplast may be detrimental to the parasite. Although very little is known about the physiology of the obligate parasite or the specific requirements made upon the host, it is obvious that in establishing a partially commensal state with the

host, these fungi have sacrificed most of their independence of action. The obligate parasites are not versatile enough to accept the biochemical changes induced in the host by two or three new genes. Resistance to a single biotype usually is regulated by a single gene or pair of genes. Because of the prevalence of monohybric and dihybric inheritance of resistance, breeding of new varieties is a relatively simple matter. It is equally simple for a new race of the parasite to circumvent such highly specialized forms of resistance by reorganization of its own genes.

Because of this interplay of genes of the host for resistance and those of the parasite for invasiveness, Flor (1946) has suggested that there are matching genes in *Melampsora lini* and its host. This intriguing concept must be true at certain levels but there are undoubtedly many genes that operate independently because a rust and its host do have many functions that are independent of each other. Only time and the acquisition of knowledge on the biochemistry of the host-parasite relationship in obligate parasitism will reveal to what extent there are matching of genes for resistance in the host and for parasitism in the pathogen.

6. *Diversion of Foodstuff to Abnormal Uses*

The Galls. It should be pointed out that certain types of fungi, still classified as obligate parasites, have rather wide host ranges and are not readily controlled by breeding for resistance. Among these are certain members of the lower fungi such as *Plasmodiophora brassicae,* the cause of clubroot. Many efforts to develop resistance in its various cruciferous hosts met with uniform failure until within the past 20 years (Walker, 1936). This experience shows that parasites which are not readily cultured in artificial media do not necessarily have to be highly specialized in their host preference. Their major nutritional requirements may be met by some common function of photosynthesis or protein metabolism but apparently they are not so highly specialized that they may be destroyed by relatively minor changes in the host.

In general, there have been variable degrees of success in breeding for resistance to those pathogens that operate as growth regulators. Probably this is attributable to the fact that some of them are relatively poor parasites. Some are unspecialized facultative saprophytes that have an unusual ability to produce extracellular chemicals that have plant growth stimulating ability while others are obligate parasites.

Many of the pathogens such as viruses that regulate cell functions from within the tissues without causing necrosis are highly specialized, and almost without exception can be controlled by certain forms of resistance. For example, the parasitism of cereal smuts which converts

ovary tissue into an overexpanded mass of mycelium and host cells apparently is controlled by two or three genes in many varieties. Likewise, there is ample evidence that the susceptibility to viruses may be changed by replacing one to three genes.

VIII. Search for Effective Disease Control by Application of Logical Principles

Plant pathology has one ulterior purpose—the prevention of plant disease. Therefore, much of the knowledge and skill of the profession must be directed toward *control of the forces of pathogenesis.* Any idea for controlling pathogenesis should take into account that disease is just as much the product of host reaction as it is the result of a parasitic invasion.

A. *Methods of Regulating Pathogenesis*

Much more is involved in this concept of disease control than the ordinary idea of destroying or avoiding pathogens. It is based upon the obvious truth that a parasite may be tolerated if means can be developed to regulate or circumvent its ability to induce disease. As mentioned in the opening paragraphs and as illustrated by several examples in subsequent discussions, parasitism may play a minor role in many diseases.

This idea of regulating the forces of pathogenesis as well as exterminating the pathogen is entirely sound because the records show that man has never learned "to control" any of the major pathogens. About all that has been achieved is to find methods of living with them—but not at their mercy. How many have ever been eliminated (i.e., eradicated entirely) by breeding, application of protective chemicals, sanitation, disinfection of the environment, etc.? Not one! The nearest approaches have been in the breeding of the Washington varieties of asparagus for resistance to *Puccinia asparagi,* an autoecious rust, and in eradicating citrus canker (*Xanthomonas citri*) in Florida by burning all infected groves. But the rust can still be collected today on wild asparagus and citrus canker does flare up at times so these are horticulturally successful measures rather than absolute victories over the pathogen.

A tolerable situation such as this can be established by: (a) use of disease-escaping tactics, (b) suppression of the pathogens to permissible levels, or (c) use of disease-resistant or tolerant varieties of crops to establish a favorable host-parasite balance. Many prevalent diseases can be escaped by use of disease-free seed, selection of sites unfavorable to the pathogen, choice of favorable planting dates, avoiding wounds and bruises, use of quarantines, etc. The prevalence of the pathogen can

be suppressed by seed treatment, soil disinfection, protective foliage sprays, space fumigation of soil and storage areas, stimulation of antibiosis, or rotation of crops. The disease response of plants to infection can be ameliorated by antidoting toxins, proper fertilization of soil, encouragement of new root growth, avoiding extreme fluctuations in soil moisture and available nutrients, etc. Resistant crops can be developed by selection, hybridization, and grafting on resistant root stocks.

Which one of these various methods is likely to be most successful against a specific pathogen? Does the physiological basis of organizing the knowledge on plant disease offer any help in developing some sound principles? If the principle is fundamentally sound, it should be useful in directing research activities into fruitful channels. In order to draw the facts together in an intelligible manner it is well to study the interrelationship of various factors in an epidemic.

B. *The Factors in an Epidemic*

Any disease outbreak depends upon three elements: the inherent susceptibility of the host, the inoculum potential of the parasite, and the impact of environment on parasitism and pathogenesis. Each of these has two components as indicated schematically by the triangle of epiphytosis in Fig. 1. The height of the apex (severity of the epidemic) is determined by the relation of these six components.

The base for any epidemic is the susceptibility of the host. This susceptibility is composed of two factors: its seasonal development which exposes it to the pathogen, and its inherent resistance to invasion or tolerance of the forces of pathogenesis. If either of these elements is shortened as indicated in Fig. 1 from D-10 to D-2 on one hand or from I-10 to I-2 on the other, the host will either escape the infection periods or resist entering into a pathogenic process. Even if all other elements remain the same, damage to the crop could be reduced from 100 units to 20 or less as the base of an epidemic is reduced. If either side of the base is reduced to 0, obviously there can be no crop damage.

Hinged to the seasonal development of the crop is the influence of environment. If the host develops at the proper season, it may encounter conditions that will be favorable to the pathogen and to its own disease reaction. If the various climatic and edaphic factors of temperature, moisture, acidity, etc., are properly balanced, the environment becomes more conducive to disease development. These could be indicated by the length of the left-hand side of the triangle. However, the full impact of environment depends on the frequency and duration of these periods which are usually designated as infection periods. If the periods are reduced to zero, there can be no disease so this force operates as the

angle on the left-hand side of the triangle to determine whether maximum damage will be 100, 50, or 0 units, for example.

The virulence and, to a large measure, the prevalence of the parasite will be determined by the inherent susceptibility of the host as described in the section on host-parasite balance. The virulence of the pathogen,

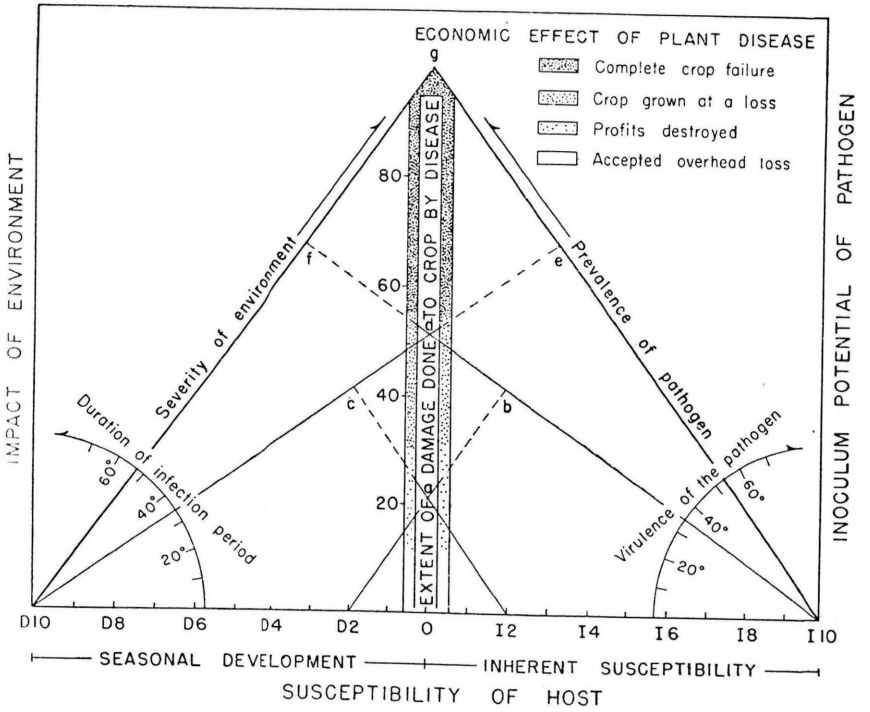

FIG. 1. The triangle of factors that limit an epidemic. The full impact of a pathogen may be avoided either by reducing host susceptibility, inoculum potential of the parasite, or environmental conditions favorable to pathogenesis. The peak may be reduced from a maximum point of "g" to some lower level such as a, b, c, e, or f by restricting one or more of the six factors in an epidemic, as indicated by the internal lines. This would reduce crop losses from a maximum of 100 to some lower figure such as 50, 20, or even 0. All figures are assigned empirically and cannot be given precise values at this stage of knowledge. The preferred treatment for a disease shifts progressively clockwise from the left corner to the lower right side of the base as the specialization in pathogenesis increases from class 1 to class 6 of disease.

unfortunately, has never been fully divorced from the innate properties of the host so no one has been able to reduce inherent virulence for a given host in a practical way. Even if methods for causing dissociation of avirulent strains were developed, the forces of selective evolution would prevent survival of such strains under field conditions.

Only one basic rule seems to emerge. As the inherent susceptibility of the host is reduced by breeding or selection, the angle of virulence increases so the parasite tends to maintain a constant inoculum potential. Disease control must depend on reducing the prevalence of the parasite (shortening the right side of the triangle) or disrupting the degrees of virulence by frequently changing the genetics of the host. Schematically, the severity of an epidemic could be held at 50 units either by reducing the prevalence of the pathogen to "e" or the angle of virulence to 35 degrees, as shown in Fig. 1.

C. *Relation of Class of Parasitism to Disease Control*

Disease control, therefore, may be achieved by disrupting any one of six factors in the epidemic triangle. Which one of these factors is most likely to be successful? It depends strictly upon the type of pathogenesis involved. For example, if all the disease prevention methods currently used were enumerated for diseases representative of each class of pathogens, they could be classified somewhat as indicated in Table I.

The more poorly specialized parasites of the storage organs (class 1), which are nothing much more than opportunistic facultative saprophytes, are controlled by use of disease-escaping tactics—avoid wounds, harvest fruit when mature, dry immediately, prevent sweating in storage, etc. In short, disease controls are applied at the left-hand base and angle of the epidemic triangle. The second alternative is to regulate the environment as much as possible, and the third measure is to reduce the population of parasites. Scarcely a thought is given to inherent resistance. In other words, there is diminishing likelihood of success in developing preventive methods as one progresses clockwise around the triangle from the left angle.

The seedling diseases (class 2) which are so analogous to the rots in many respects have only slightly less possibility of control by disease-escaping measures; but there is much more emphasis on changing the environment. Seed are sown in well-prepared seed beds of proper soil texture and are treated with chemicals or the soil is treated. In other words, the emphasis in disease control shifts one more step clockwise on the triangle but does not reach over to inherent resistance.

Control of the root rots (class 3), which are one stage more highly specialized as parasitic pathogens, depends very heavily on proper choice of environment but, by far, the major emphasis is on reducing the prevalence of the parasite by various soil treatments. A form of disease resistance, albeit very imperfect, may be developed for a few of the root rots. Usually, this is a genetically controlled growth response in which the host recovers from infection although this is not a positive

TABLE I
SUMMARY OF DIFFERENT TYPES OF CONTROL MEASURES FOUND TO BE EFFECTIVE FOR DIFFERENT CLASSES OF DISEASE

Class number and type of disease	Effective class of control measures		
	Preferred method	Second choice	Third choice
(1) Rots of storage organs	Avoid wounds	Improve storage	Chemical treatment
(2) Seedling diseases	Chemical treatments	Better culture	Choice of sites
(3) Root rot diseases	Crop rotation	Stimulate antibiosis	Treat soil, resistance
(4) Vascular diseases			
(4a) Soil	Resistant varieties	Choice of site	Crop rotation
(4b) Insect	Resistant varieties	Insect control	Crop rotation
(5) Leaf diseases			
(5a) Leaf blights and cankers	Chemical protection	Resistant varieties	Sanitation
(5b) Downy mildews	Chemical protection	Resistant varieties	Sanitation
(5c) Powdery mildews	Resistant varieties	Chemical protection	Sanitation
(5d) Rusts (autoecious)	Resistant varieties	Crop rotation	Chemical protection
(5e) Rusts (heteroecious)	Resistant varieties	Eradicate Alt. host	Chemical protection
(6) Utilization diseases			
(6a) Floral smut	Resistant varieties	Seed treatment	Sanitation
(6b) Root galls	Crop rotation, soil treatment	Sanitation	Avoid wounds, resistance
(6c) Viruses (mosaic)	Resistant varieties	Seed certification	Sanitation
(6c) Viruses (yellows)	Resistant varieties	Sanitation	Vector control

form of immunity. In other words, the root rots, most of which are facultative parasites, are controlled by changing the factors one more step clockwise on the chart than for the seedling diseases.

While discussing the soil microorganisms, it might be well to consider the more highly specialized gall forming (class 6) parasites that are capable of invading roots and regulating their growth (6b). Nearly all of these have an existence apart from the host so special effort is made to reduce their population in soil. However, there may be definite forms of resistance in varieties, so breeding may be more valuable than for any of the other diseases discussed above. Control measures rotate clockwise to the right-hand side of the triangle.

The wilt diseases (class 4) further demonstrate that type of control measure is largely determined by the physiological specialization in the parasite. Disease resistance becomes very important with secondary consideration to methods of avoiding the pathogen or reducing its prevalence. Crop rotation is important and avoidance of vectors is a major consideration.

The same series of changes may be observed in disease-prevention measures for the pathogens of aerial organs (class 5). In the least specialized leaf blights, efforts are made to avoid the pathogen by choice of sites and planting dates, but most of the control revolves around use of chemicals to reduce the prevalence of the parasite. A great many of the leaf blights and downy mildews are sufficiently specialized so they can be avoided by use of disease-resistant or tolerant varieties. The emphasis upon disease-resistant or even immune varieties increases as obligate parasites are examined. The more highly specialized pathogens of the utilization system of the plant (class 6) such as the smuts, the powdery mildews, rusts, and viruses are very susceptible to hereditary changes in the physiology of the host.

Although there are minor exceptions to the sweeping generalities made here, it is interesting that a classification of disease based on the physiology of pathogenesis leads to such an orderly understanding of disease-control measures. This is not a surprising revelation because the system aligns the parasites in a more or less orderly fashion according to the specialization in their parasitic abilities.

The pathogen that is divorced from its susceptible host most of the time is very susceptible to changes in the environment. Those that destroy the host most readily when it is handicapped by unfavorable environment are less destructive when conditions are made favorable to the host's growth. The parasite that is exposed on a leaf, even for a brief period in moving from leaf or twig to new susceptible tissue, can be suppressed by chemicals; but if it is so highly specialized that it

has no existence apart from its host, the metabolism of the host cell is the dominant feature in determining how successful its parasitism will be. Such parasites are most readily suppressed by use of resistant varieties.

IX. Discussion and Summary

When the efficiency of a plant is so reduced that it cannot make maximum use of the factors of its environment for growth and reproduction, that plant must be considered as diseased. Efficiency may be impaired by disrupting any one or more of the several basic physiological processes involved in the synthesis and utilization of foodstuffs. Therefore, plant disease is essentially a physiological process of the plant regardless of whether it is incited by physiogenic or pathogenic agents in the environment.

Most plant diseases are caused by pathogens, so pathogenesis is of major concern. Pathogenesis is the result, in many but not all diseases, of a parasitic establishment. The direct effects of parasitism, however, may be rather mild even though the loss of food materials does constitute a drain on the host's efficiency. However, the further exacerbation of disease condition is more often due to the foreign chemicals produced either by the pathogen or the invaded tissues. Extracellular materials such as the enzymes that dissolve cell walls, the toxins that poison metabolic processes of the cell, or the growth regulants that influence the rate of cell multiplication and differentiation are vitally important in determining pathogenesis in plants.

The postulate is advanced that parasites are in a constant process of evolution in which they progress from nonparasitic associative relations with the host through facultative saprophytism and parasitism to obligate parasitism. Simultaneously, there is a reduction in the tendency to destroy host cells by secreting digestive enzymes and the pathogen tends to become associated with the host cell in a compatible arrangement. The habitat of the parasite in the host changes from a general cell invasion to a more specific intercellular establishment. The ultimate in parasitism is found as an intercellular invasion with haustoria in the cell or even an external habit with haustoria in the host cell as in the Erysiphaceae.

Concurrently with these changes, the host becomes more responsive or even actively reactive to invasion so that in the more advanced types of parasitism there is an essentially commensal state existing between host and parasite. The parasite curbs its destructiveness (pathogenicity) and seeks out a compatible intracellular association where it can procure food or regulate cell activities from a point of contact near to the host

nucleus. In the most advanced stages of evolution the parasite regulates activities of the host cell for its own gain, and may even be commensal with it.

There is substantial evidence in support of this hypothesis that parasitism evolves toward commensalism by increased specialization and skillful change in the type of disease-inducing chemicals released. The host and parasite come into a balanced state either by physiological adjustment on the part of the parasite or by selective hereditary changes in the host. An understanding of this process of evolution in parasitism seems to be a prerequisite to formulation of programs for disease control.

The specialization of the parasite apparently depends upon the intricacy of the host function that it affects. The destruction of a food reserve or digestion of a dead cellulose cell in wood or fiber can be achieved by any saprophyte with the proper enzymes provided it has tolerance for the cell environment. The general destruction of nutritious cortical cells in the seedling tissue or in roots, so as to prevent establishment of water-procuring organs, is only one stage more specialized. However, selective invasion of special cells such as the tracheal tubes and use of the nutrients of the tracheal sap necessitate much more specialization. The parasite sacrifices a measure of its independence and becomes more dependent upon the host's physiology. With only minor variations this specialization becomes intensified as the pathogens become more adept at operating on or in the cells that are actively photosynthesizing foodstuffs where they cause various forms of leaf blight. Any interference in the transport and assimilation of foods into new cells and tissues without outright destruction of the tissues constitutes a very delicate adjustment, particularly when the metabolism of the cell is altered or growth processes are changed as occurs in various regulatory diseases such as viruses.

When diseases are classified into six groups according to this scheme, without concern for the taxonomic classification of the parasite or the particular hosts involved, the orderly arrangement proves very valuable in understanding the nature of disease. Examples were offered to show how this order can be used to explain much of the influence of environmental conditions on disease, why host nutrition affects various diseases differently, how host genetics influence parasitism, and what sort of disease-control practices are preferred for different diseases.

It is only natural that this system of organizing the knowledge on plant diseases would cast new light on such considerations because the system is based on physiological considerations that are basic to parasitism and pathogenesis. Because this approach reaches beyond superficial trivialities and provides basic principles, it should be valuable in organizing research and in teaching the principles of plant pathology. It is easier to remember details and judge circumstances when they are coordinated in a logical scheme rather than as isolated events.

Only the most primitive effort can be made today to classify diseases as physiological functions of parasitism because so little is known about the biochemistry of disease processes (pathogenesis). This is to be regretted very deeply because it prevents maximum progress in solving disease problems in plants. Probably an equally great disservice is being performed for other professions because knowledge of the basis of pathogenicity could cast much light upon physiology of plants in general.

Indicative of the great contributions that plant pathologists could provide the world by intensive research on the physiology of pathogenesis are such achievements of the past as the studies on the pectinase enzymes in vegetable soft rots by Jones (1909), the development of new classes of plant growth regulants in the gibberellins isolated from the bakane disease of rice by Kurosawa (Stowe and Yamaki, 1957), the new concept of an autocatalytic growth incitant in crown gall by Braun and Mandle (1948), and the discovery of a new amino acid (tabtoxinine) in the toxin from wildfire of tobacco by Woolley *et al.* (1952b).

Such knowledge has significance far beyond the boundaries of plant pathology. Hundreds and possibly thousands of such secrets are hidden in the galls, the blights, the rots, the pigment changes, the abscission of leaves, and many other symptoms. These mechanisms of pathogenesis could be the key to an understanding of cancer or could cast new light on the forces that make plant cells grow and differentiate into new tissues.

Never was the time better for launching investigations of this sort. The chemists and physicists have placed at the disposal of biologists new techniques and analytical tools that are most expedient and reliable. There is no longer any sound excuse for not investigating the amino acids, the organic acids, the keto acids, the sugars, the growth regulants, the pigments, the toxins, or any other class of chemicals wherever the imagination would lead an investigator.

The tools of paper chromatography, column chromatography, electrophoresis, paper ionophoresis, ultraviolet and infrared spectrophotometry, and many others await full use in this field. They are the means of gaining a full understanding of the forces behind pathogenesis. With understanding, far better than that proposed above, will come progress in altering the metabolism of the host so as to incite acquired resistance

to parasites, in developing new chemicals to inhibit pathogens or alleviate their damage, and in improving the processes of breeding for inherited resistance.

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