15

Structural and Physiological Alterations in Susceptible Host Tissue

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fungus grows and sporulates. The changes in the host are assumed to benefit the fungus but usually in ways that are not entirely clear. Each rust uredium is the site of intense metabolic activities of both host and parasite. Changes in the host tend to be obscured by activities or substances in the fungus. Nevertheless, through diverse experimental approaches, many changes in the host have been described, and some are beginning to be understood.

The cereal host seems to go through two distinct responses to rust infection: (1) an initial juvenile, antisenescence response in which host cells are kept physiologically young and (2) an autolytic response in which cytoplasmic organelles slowly disappear and cells become highly senescent. This chapter will describe the principal structural and physiological changes associated with these responses.

II. Structural Changes in Rusted Host Tissues

A. THE JUVENILE HOST RESPONSE

For the first 4–6 days after inoculation, colonized host tissues at each infection site are maintained in a juvenile condition in which normal leaf senescence is retarded. This is directly visible if senescence is accelerated by detaching or shading infected leaves. As noncolonized tissues become yellow, the colonized tissue remains green, producing a "green island" at each infection site (Bushnell, 1967; Dekhuijzen, 1976; Durbin, Chapter 16, this volume; Ruttle and Frazer, 1927). As the fungus starts to sporulate, the central tissues, in contrast, may become chlorotic, leaving a green ring. The green island sometimes associated with infection type 2 in stem rust (Allen, 1926; Stakman *et al.*, 1962) is a special case in which the island becomes visible because peripheral yellowing is promoted by incompatibility.

Host cell cytoplasm in the juvenile response generally resembles cytoplasm of young healthy leaves. This is most evident in the vicinity of haustoria within infected cells. An extensive network of endoplasmic reticulum (ER) develops (Ehrlich and Ehrlich, 1971b), which sometimes touches the extrahaustorial membrane, and unique tubular complexes, thought to be synthetic or secretory structures related to requirements of the fungus, may be present in host cytoplasm (Harder and Chong, Chapter 14, this volume). Golgi bodies increase in number (Ehrlich and Ehrlich, 1971a; Shaw and Manocha, 1965b; Mares, 1979; Reiter *et al.*, 1976; Van Dyke and Hooker, 1969), and the volume of cytoplasm seems to increase (Ehrlich and Ehrlich, 1971a; Mares, 1979;

Shaw and Manocha, 1965b). These changes may be related to migration of cytoplasm and organelles to the haustorium instead of synthesis of new structures. J. L. Gay (personal communication) noted that the abundant ER and organelles near haustoria may be normal for cytoplasm near nuclei where haustoria are located.

The rust haustorium is universally found in contact with the host nucleus in cereal or grass hosts (Allen, 1923, 1926; Hilu, 1965; Mares, 1979; Ruttle and Frazer, 1927; Van Dyke and Hooker, 1969). Sometimes the nucleus and haustorium are partially enfolded with one another. Why the nucleus migrates to the haustorium and remains there is not known.

Rust infection of cereal cells generally stimulates a marked increase in volume of the host nucleus (Allen, 1923; Bhattacharya and Shaw, 1967; Hilu, 1965; Ruttle and Frazer, 1927; Whitney *et al.*, 1962). The increase begins in the juvenile host response stage, but extends into the beginnings of the autolytic, chlorotic stage. Thus Allen (1923) showed that host nuclear volume doubled with wheat stem rust by 7 to 10 days after inoculation. The increase in volume is greatest at the colony center, tapering to little change at the margin of the colony (Whitney *et al.*, 1962). The increase in nuclear volume is accompanied by an increase in volume of the host nucleolus with wheat stem rust (Bhattacharya *et al.*, 1968; Whitney *et al.*, 1962) and by a shift from several nucleoli per nucleus to a single nucleolus in a corn rust (Hilu, 1965). For wheat leaf rust, Allen (1926) noted that host nuclei did not increase in volume but that they elongated, producing a narrow, tapering lobe that frequently came in contact with the haustorium.

The period in which the host nucleus and nucleolus usually enlarge coincides with increased synthesis of nucleolar and extranucleolar RNA, and of cytoplasmic protein in host cells (see Section IV,B and C). Apparently, the enlarged nucleoli and nuclei produce increased amounts of ribosomes and messenger RNA (mRNA) that are used for protein synthesis in the cytoplasm. As noted, these changes are not strictly associated with the juvenile host response and instead may provide enzymes active in the autolytic stage to follow.

B. THE AUTOLYTIC HOST RESPONSE

By 10 days after infection, the cytoplasmic organelles of host cells at pustule centers begin to degenerate and eventually disappear in what seems to be a slow autolytic digestion process. The ER becomes less abundant, breaking into vesicles, and mitochondria lose their inner membranes (cristae), swell, and become vesiculate (Shaw and Man-

ocha, 1965b). Mitochondria may gain small electron-dense inclusions (Reiter *et al.*, 1976). With wheat stem rust, the volume of host chloroplasts is usually reduced (Allen, 1923; Whitney *et al.*, 1962), although chloroplasts sometimes appear swollen (Ehrlich and Ehrlich, 1971a). Volume of chloroplast stroma can increase (Reiter *et al.*, 1976). The chloroplast lamellae may become less compacted as grana structure becomes disorganized. The outer chloroplast membrane can become vesiculate (Shaw and Manocha, 1965b) and eventually rupture (Ehrlich and Ehrlich, 1971a). Leaf rusts of wheat and barley cause a less detrimental response in which chloroplasts may not shrink (Allen, 1926) or show signs of structural degeneration other than loss of chlorophyll (Calonge, 1967). Electron-dense materials have been seen in chloroplasts with wheat leaf rust (Reiter *et al.*, 1976) and stripe rust (Mares, 1979).

As part of the autolytic stage, the host nucleus shrinks rapidly. With wheat stem rust, Allen (1923) showed a decrease in nuclear width and volume by 14 days after inoculation. This occurred about 4 days after the chloroplasts began to shrink. With wheat leaf rust, Reiter *et al.* (1976) found a reduction in host cell nuclear volume as chromatin within the nucleus condensed.

As autolysis progresses, most host cells remain alive and turgid for as long as 3 to 4 weeks after inoculation. Vacuoles can form that contain residues of degenerated organelles, membranes, and electron-dense bodies (Reiter *et al.*, 1976). In advanced stages of autolysis, virtually the only host cell contents remaining are the host nucleus and the fungal haustorium (Hilu, 1965), as the cytoplasmic layer lining the cell wall becomes highly attenuated (Mares, 1979).

The autolytic host response resembles normal senescence of cereals and other plants (Shaw and Manocha, 1965b; Stoddart, 1981). The sporulating fungus seemingly accelerates the senescence of the host tissues; the result is digestion of host constituents, which are then probably utilized largely by the sporulating fungus.

C. THE PHYSICAL PRESENCE OF THE FUNGUS

Rust mycelium develops so abundantly in the intercellular spaces of the cereal leaf that its physical presence could be a factor in changes induced within host cells. As much as half the volume of spongy mesophyll can be intercellular space. Allen (1923) described a zone perhaps 1 mm in diameter at the center of young wheat stem rust pustules in which ". . . each chink and cranny of intercellular space becomes filled with the fungus, which forms little masses of pseudoparenchyma

conforming closely to the shape of the irregular passages they occupy." In older pustules, "some of the host cells are crowded out of shape and almost obliterated, but in many cases are still living." Similar fungal development has been described for a corn rust (Hilu, 1965) and oat crown rust (Ruttle and Frazer, 1927). Host cells are especially likely to be deformed or crushed in the layer of mesophyll immediately below the epidermis.

The engulfed host cells are thus subject to damage by the physical presence of the growing fungus and also by any metabolites that might leak or be secreted from the fungus. The gaseous environment around host cells is undoubtedly changed. The shift from the juvenile to the autolytic host response occurs at the onset of sporulation when hyphae begin to rupture the epidermis, separating it from the underlying fungal–host complex. Still, experimental evidence is lacking to show how the physical forces exerted by the fungus affect host cells.

D. CHANGES IN HOST MEMBRANES

During the juvenile response, host cells probably retain a full capacity for active uptake of metabolites. Later, as disease progresses into the autolytic stage, host tissues become leaky and readily lose ions, sugars, amino acids, and probably other substances if the tissues are immersed in water (Hoppe and Heitefuss, 1974a). Such leakiness is a characteristic of uninfected, naturally senescing tissues as enzymes located on membranes lose activity (Stoddart, 1981). Thatcher (1942) showed for wheat stem rust that the host cell plasmalemma has increased permeability to nonelectrolytes, which are now thought to enter cells through the phospholipid portions of the membrane. Some evidence for changes in phospholipid components of host membranes in bean rust was obtained by Hoppe and Heitefuss (1974b). Elnaghy and Heitefuss (1976) implicated the germination self-inhibitor of bean rust urediospores (methyl-3,4-dimethoxycinnamate) as a possible cause of the change in host membranes. The importance of membrane alterations to the movement of nutrients from host to parasite is discussed further by Durbin (Chapter 16, this volume).

E. DIFFERENCES IN HOST RESPONSE AMONG CEREAL RUSTS

The sequence of qualitative changes in host tissue is similar among the cereal rusts, but stem rusts induce more pronounced host changes than do leaf rusts. Allen (1923) provided a clear example in wheat. The

host cell in which the first haustorium was produced at each infection site was killed with stem rust, not leaf rust. Also, the changes in host nuclei and chloroplasts were greater with stem rust than leaf rust. Furthermore, the stem rust fungus produced a massive concentration of cells at pustule centers with vigorous runner hyphae at colony borders, whereas the leaf rust fungus grew less and lacked runners. Thus physiological changes may be correspondingly greater in stem rusts than in leaf rusts.

III. Hormonal Changes in Rusted Host Tissues

A. Overview of Growth Hormones in Rusts

Plant growth hormones generally increase greatly in rusted tissues. These hormones could be produced by the host, the fungus, or both. Most types of hormones have been found in rust spores or mycelium, and the quantities of hormones tend to parallel growth of the fungus, often peaking at the time of sporulation. This suggests that the hormones in rusted tissues are produced by the fungus and that they may have a role in fungal growth and sporulation, although the hormones could be produced by the host and transferred to the fungus in some cases. In addition to effects on the fungus, growth hormones may induce some or all of the host responses described in Section II. The somewhat sparse data suggest that rust fungi modify infected tissues either by producing the hormones themselves or by changing the local concentrations of host-produced hormones.

B. Cytokinins

Cytokinins are implicated as inducers of the juvenile host response to rust infection. If a cytokinin solution is applied as a drop to the surface of a detached leaf, tissues near the site of application remain green as the rest of the leaf rapidly senesces as a result of detachment. The green zones are metabolically active and closely resemble the green islands produced at rust pustules on detached leaves as described in Section II,A (Bushnell, 1967). The cytokinin-induced green zone acts as a sink for substances translocated from the yellow, senescing parts of the detached leaf as do rust-induced green islands (Durbin, Chapter 16, this volume). Furthermore, Shaw and Manocha (1965a,b) concluded from ultrastructural comparisons that cytokinin-treated tissues close-

ly resemble tissues infected with *P. graminis* f. sp. *tritici*. Cytokinins seem to delay leaf senescence by maintaining protein synthesis (Stoddart, 1981) through mechanisms that are not understood but that possibly act through effects on cell membranes (Durbin, Chapter 16, this volume).

Extracts from rusted leaves of bean (Dekhuijzen and Staples, 1968; Király *et al.*, 1967) and wheat (Sziráki *et al.*, 1976) have had more activity in cytokinin bioassays than have extracts from healthy leaves. Increases were principally due to a single chromatographic fraction in each case. In bean, this fraction chromatographically resembled cytokinin from the host and was unlike cytokinin from rust mycelium, implicating the host as the source of the increased cytokinin. The increases in amount of cytokinins, and the similarities in effect of rust and cytokinin, provide considerable evidence that cytokinins contribute to maintenance of the juvenile state in rusted tissues.

Nevertheless, the evidence that cytokinins control the juvenile state is incomplete, especially in cereal rusts. Extracts from spores of *P. graminis* f. sp. *tritici* and *avenae*, *P. coronata*, and *P. recondita* have all produced green zones in detached cereal leaves (Bushnell, 1967; Johnson *et al.*, 1966), but the green zones were not sinks for translocation of ^{32}P as expected. Several substances other than cytokinins can delay leaf senescence (Bushnell, 1967; Dekhuijzen, 1976; Durbin, Chapter 16, this volume; Stoddart, 1981); one or more of these could have a role in rust-induced senescence delay.

C. AUXINS

Auxins generally increase in rusted tissues, particularly in the rusts that produce galls and overgrowths of host tissue (Pegg, 1976a). In rusted cereal tissues, auxin apparently also increases, especially at sporulation, but the pattern of increase is incompletely documented. The frequently cited data of Shaw and Hawkins (1958) include only one value for a compatible host–parasite combination, showing a 20-fold increase in free indoleacetic acid (IAA) 10 days after inoculation. Shaw (1963) cited data of B.I.S. Srivastava showing 10- to 50-fold increases in free IAA with wheat stem rust at sporulation and smaller increases in bound IAA. Artemeko *et al.* (1980) reported a 2.5-fold increase in free IAA with wheat stem rust from 6 to 144 hr after inoculation, whereas esterified IAA did not increase. The surprising initial increase in free IAA was attributed to IAA in the spores used to inoculate the plants.

The increase in IAA in rusted cereal leaves may possibly be a conse-

quence of decreased IAA oxidation by oxidases (decarboxylases) or peroxidases (Daly and Knoche, 1976; Pegg, 1976a). Shaw (1963) listed other possibilities, including synthesis of IAA by the rust fungus. Indoleacetic acid has been found in urediospores of *Puccinia graminis* (Umnov *et al.*, 1978).

What is the role of IAA in rusted tissues? It may cause a small part of the respiratory increase thought to occur in host tissues (Section IV,F). It probably acts in concert with other growth hormones, especially cytokinin, to control the metabolic state of the host cell in the juvenile stage of host response. In addition, auxin may have a direct function in growth and sporulation of the rust fungus.

D. ETHYLENE

Emission of ethylene from wheat leaves can increase 10-fold or more as a consequence of rust (Daly *et al.*, 1971), with the highest rates at the start of sporulation. The large increases in emitted ethylene in rusted tissues are difficult to evaluate because the amounts within leaves can be large compared to amounts released (Pegg, 1976b). Chigrin *et al.* (1978) found increases in emitted ethylene with wheat stem rust to be small compared to the large, fluctuating amounts within leaves.

Ethylene is commonly emitted from injured or diseased plant tissues in which cells are killed (Williamson, 1950). Ethylene is sometimes emitted in bursts before or during expression of hypersensitive cell death in rusts (Chigrin *et al.*, 1978; Montalbini and Elstner, 1977). In compatible hosts, ethylene emission might relate to cell injury when the epidermis is ruptured at the start of sporulation. However, the ethylene is unaccompanied by ethane, a usual sign of injury (Montalbini and Elstner, 1977).

If wheat leaves are exposed to ethylene, peroxidase activity of the leaves increases. Nevertheless, the peroxidase content of rusted, susceptible leaves is low, despite high rates of ethylene emission (Daly *et al.*, 1971).

Could ethylene have a controlling role in the autolytic response of susceptible hosts? Premature senescence and yellowing are among the many physiological effects of ethylene (Archer and Hislop, 1975), and ethylene is emitted rapidly during the initial stages of chlorophyll loss in senescing leaves (Stoddart, 1981). The largest increases in ethylene in rusted leaves occur as the autolytic stage begins at pustule centers. Heath (1974) cautioned that the changes in chloroplasts apparently

triggered by ethylene in cowpea rust are more like changes in ripening fruit than in senescent leaves. Stoddart (1981) attributed some ethylene-induced changes to injury instead of senescence. In any case, the role of ethylene deserves further investigation.

E. ABSCISIC ACID

Abscisic acid has been shown to increase in wheat stem rust (Chigrin *et al.*, 1981). Abscisic acid has been postulated to have an indirect role in promoting senescence in healthy leaves and can promote premature yellowing of detached leaves (Stoddart, 1981). Along with ethylene, abscisic acid deserves investigation as a possible cause of the yellowing and autolysis of rusted tissues.

IV. Metabolic Changes in Rusted Host Tissues

A. THE HETEROGENEOUS RUSTED LEAF

The typical rusted leaf sampled for physiological purposes contains many uredia that may coalesce as they enlarge. With time, an increasing proportion of the leaf comes under the influence of the rust. Additional heterogeneity results from changes within each uredium. Tissues at uredial centers are usually at a more advanced stage of response than tissues at the edges. At certain times after infection, host tissues may be briefly homogeneous as zones of influence coalesce, but this homogeneity is difficult to achieve reproducibly. [Patterns of starch deposition can be useful for this purpose (Bushnell, 1967).] Because of this heterogeneity, results obtained at different infection densities are often inconsistent.

The growing fungus contributes to heterogeneity of the rusted leaf. Unfortunately, no entirely satisfactory way has been found to measure the amount of rust fungus present in infected leaves (Rohringer and Heitefuss, Chapter 7, and Rowell, Chapter 10, this volume). This is one reason estimates are poor for the relative contributions of host and parasite to an activity or substance common to both.

Changes in weight present another obstacle to interpreting results from rusted leaves. Dry weight per unit leaf area increases 20–100% in cereal rusts (Johnson *et al.*, 1968; Owera *et al.*, 1981; Quick and Shaw, 1964; Shaw and Colotelo, 1961; Samborski and Shaw, 1956). Fresh weight can decrease abruptly in advanced stages of infection. Conse-

quently, data reported on a weight basis are difficult to interpret if weight per unit area is not given.

Finally, the natural senescence of cereal leaves complicates interpretation of changes reported for rusts. As soon as the leaves are fully grown they begin to senesce, slowly losing protein and many metabolic components. Because of this, rusted tissues may have more of a substance than do nonrusted tissues at a given sampling time, because the loss was retarded and not because of an actual gain. To distinguish between the two possibilities, samples must be taken near the time of inoculation and periodically thereafter.

B. NUCLEIC ACIDS

How nucleic acids (and proteins) in rusted tissues relate to whether host and parasite will be compatible or incompatible is treated by Rohringer and Heitefuss (Chapter 7, this volume). Here we focus on compatible host–parasite combinations and the series of changes that occur within the host as disease progresses.

1. RNA

As part of the juvenile host response before the fungus sporulates as postulated in Section II,A, host cells should maintain or possibly increase their ability to synthesize protein. They should maintain the machinery needed for DNA-dependent transcription of mRNA and production of ribosomes. Protein synthesis occurs in cytoplasm, synthesis of ribosomes occurs in nucleoli, and synthesis of mRNA occurs in the extranucleolar portion of nuclei.

Enhanced metabolic activity in both the nucleolar and extranucleolar portions of host nuclei has been demonstrated cytologically for wheat stem rust by M. Shaw and co-workers. As the volumes of host cell nuclei and nucleoli increase (see Section II,A), the amount of both nucleolar and extranucleolar RNA doubles (Bhattacharya *et al.*, 1965; Whitney *et al.*, 1962). Incorporation of radioactively labeled uridine and cytidine (precursors of RNA) into nuclei was doubled (Bhattacharya and Shaw, 1967), as was incorporation of leucine into nuclear protein (Bhattacharya and Shaw, 1967). Heitefuss (1970) showed that actinomycin D inhibited the incorporation of labeled uridine, indicating that incorporation depended on transcription. Furthermore, the diffuse interchromatin network of the nucleus, where transcription occurs, increased in electron density (Manocha and Shaw, 1966). Finally, the amount of histone within the nucleus decreased, and apparently also the incorporation of amino acids into histone (Bhattacharya *et al.*,

1965, 1968). Histones are thought to repress transcription nonspecifically (Rohringer and Heitefuss, Chapter 7, this volume). Together, these cytological studies indicate that transcriptional activities increase in host nuclei as a consequence of rust.

The incorporation of ^{32}P into total RNA of rusted leaves increased two- to five-fold 3 to 6 days after inoculation in wheat stem rust (Dmitrieva and Zhukov, 1971; Rohringer and Heitefuss, 1961) and oat crown rust (Tani *et al.*, 1970), probably reflecting synthesis of RNA by both host and parasite. However, the total amount of RNA either increases modestly or not at all, even though the fungus is growing and probably synthesizing RNA. This suggests that the total amount of RNA in the host declines.

Trends in total RNA from the time of inoculation indicate that the loss of RNA that normally occurs in uninfected leaves is retarded by disease, resulting in 20 to 40% higher amounts in rusted than nonrusted tissues (Heitefuss, 1964; Johnson *et al.*, 1967; Quick and Shaw, 1964; Tani *et al.*, 1970). Fractions of RNA such as rRNA also tend to show retarded loss instead of actual gains, whereas chloroplast rRNA clearly declined with oat crown rust (Tani *et al.*, 1973a). Because amounts of RNA decline in the host while rates of RNA synthesis are enhanced (as described earlier), it follows that the rate of RNA degradation is increased.

2. *RNase*

In line with the probable enhancement of RNA degradation, the activity of RNase increases in rusted tissues (Rohringer and Heitefuss, Chapter 7, this volume). With wheat stem rust, RNase activity doubles at 1 to 4 days after inoculation [which could be an artifact of handling at inoculation (Nielsen and Rohringer, 1963)] and later peaks again at about 6 days at levels two to five times those of uninfected leaves (Chakravorty *et al.*, 1974; Sachse *et al.*, 1971). Apparently, the new RNase is the type found in uninfected leaves and not a fungal type (Rohringer and Heitefuss, Chapter 7, this volume). Furthermore, the RNase in rusted flax was of the type that degrades RNA and not of the type involved in posttranscriptional processing of RNA (Sutton and Shaw, 1982). The large amount of RNase activity in cereal rusts is probably involved in rapid RNA turnover.

3. *DNA*

Because cereal host cells do not enlarge or divide in rusted tissues, no increase in the amount of host DNA is expected; indeed, the amount of DNA in host nuclei as measured microspectrophotometrically does

not change with wheat stem rust until 9 days after inoculation when a slow decline begins (Bhattacharya *et al.*, 1965, 1968). Total DNA of host and parasite combined tends to remain constant (Heitefuss and Wolf, 1976; Quick and Shaw, 1964; Tani *et al.*, 1970), because it has usually been expressed on a dry-weight basis, and because the amount of nuclear host DNA eventually declines as new fungal DNA is produced. In wheat stem rust, the rate of ^{32}P incorporation into a DNA fraction increased (Heitefuss, 1965, 1966), probably a result of fungal DNA synthesis. Measurable DNase activity also increased (Heitefuss and Wolf, 1976), in line with DNA degradation in the host, at least late in pustule development.

C. PROTEINS

The total amount of protein in rusted cereal tissues sometimes increases 20–50% on a fresh-weight basis, paralleling increases in dry weight, at least in the first few days after inoculation (Quick and Shaw, 1964; Shaw and Colotelo, 1961). More frequently, the total protein of host and parasite either remains fairly constant (Johnson *et al.*, 1968; Samborski *et al.*, 1961) or declines (Gassner and Franke, 1938). Much of the total protein can be assumed to be in the developing fungus, especially at sporulation and thereafter. Probably little protein is left in the highly autolyzed host cell described in Section II,B.

Although total host protein declines, the evidence for accelerated RNA metabolism suggests that synthesis of some host proteins might be enhanced by rust infection, especially before sporulation. Surprisingly, there is little evidence showing what preexisting kinds of proteins have increased rates of synthesis or if new kinds are synthesized. With wheat stem rust, Fric and Heitefuss (1970) could not detect new kinds of host protein 5 days after inoculation using immunochemical and electrophoretic methods. New proteins were judged to be of fungal origin. With flax rust, von Broembsen and Hadwiger (1972) could find no change or only slight decreases in incorporation of radioactively labeled leucine into soluble protein in two compatible host–parasite combinations 6–18 hr after inoculation. In contrast, incorporation into several protein fractions was increased in incompatible combinations. Similar results were obtained by Tani and Yamamoto (1979) with oat crown rust 10–24 hr after inoculation. Blasticidin S, an inhibitor of protein synthesis, had no effect on crown rust development in a compatible combination, evidence that protein synthesis in the host was not required for the early stages of fungus growth. In samples taken after sporulation in wheat stem rust, Wrigley and Webster (1966) found reduced amounts of two protein peaks as detected on polyacrylamide

gels, one of which was thought to be largely ribulose-1,5-bisphosphate carboxylase, an important enzyme of photosynthesis and photorespiration (Section IV,E). Using similar methods, Staples and Stahmann (1964) found a decrease in an unidentified host protein in bean rust.

As rust develops in host tissues, several new isozymes can be detected on polyacrylamide gels. In most cases, these appear to be of fungal origin (Johnson *et al.*, 1968; Staples, 1965; Staples and Stahmann, 1964). The amount of a host isozyme may change as with acid phosphatase in bean rust (Williams and Staples, 1964; Staples and Stahmann, 1964), but most of the work with isozymes has been qualititative and does not clearly indicate quantitative changes. However, Sadler and Shaw (1979a) showed a change in a host glutamate dehydrogenase in flax rust at 1 and 7 days after inoculation. Although its molecular weight was apparently unchanged, the new form of the enzyme was distinct in degree of inhibition by ATP or pyridoxal phosphate, suggesting to Sadler and Shaw that the protein molecule had changed conformation or that subunits of the enzyme had been rearranged. Whether the enzyme had been modified during or after synthesis was not established.

Protein synthesized in cell-free translation systems using template from mRNA, chromatin, or polysomes from leaves have differed in kind and amount as a result of infection with oat crown or wheat stem rust (Chakravorty, 1982; Pure *et al.*, 1979; Tani *et al.*, 1973b). Such experiments are described by Rohringer and Heitefuss (Chapter 7, this volume). The results suggest that changes preceding translation, either before or after mRNA is produced by transcription, lead to changes in the proteins that are synthesized by the host. Although cell-free translation techniques have great potential, the results with rusts are still of a preliminary nature, and the new proteins are yet to be identified.

It seems that the juvenile host response is not accompanied by large qualitative or quantitative changes in host proteins. We know at least that host proteins are changed less in compatible than incompatible host–parasite combinations the first day after inoculation. How host proteins are changed at the beginning of the autolytic stage is unclear. As indicated in Section II,A, perhaps increased synthesis of host mRNA leads to synthesis of enzymes involved in the autolytic degeneration of cytoplasmic components of the host.

D. AMINO ACIDS AND AMIDES

Changes in rusted tissues seem to assure that generous amounts of amino acids and amides are available for nutrition of the fungus. During the juvenile host responses, these substances are probably synthe-

sized locally from photosynthates and ammonia, and also translocated from tissues distant from the infection site (Durbin, Chapter 16, this volume). Later in the autolytic stage, significant amounts of amino acids and amides probably also come from local degradation of protein.

Soluble nitrogen compounds (mostly amino acids and amides) can increase threefold in rusted tissues with wheat stem rust, especially in the first 3–6 days after inoculation (Shaw and Colotelo, 1961; Samborski *et al.*, 1961). Gassner and Franke (1938) found little or no increase, but they showed that the decline in soluble nitrogen as leaves aged was not as rapid in rusted as in nonrusted leaves. Glutamine generally increases in rusted tissues, and at least two investigators have reported increases for each of the following amino acids or amides: asparagine, arginine, phenylalanine, leucine or isoleucine, and valine (Farkas and Király, 1961; Rohringer, 1957; Shaw and Colotelo, 1961; Siebert, 1961). Tryptophan increased four- to fivefold when measured by procedures to conserve it during extraction (Kim and Rohringer, 1969). Ammonia also has accumulated in significant amounts (Farkas and Király, 1961; Siebert, 1961). Several amino acids have been reported to increase as early as 2 days after inoculation.

What amino acids or amides does the rust fungus require? In axenic culture, *Puccinia graminis* requires nitrogen in a reduced form as its principal source of nitrogen. Ammonia, aspartic acid, or glutamine can meet this need (Maclean, 1982; Mendgen, 1981). In addition, sulfur must be supplied in reduced form as cysteine, cystine, glutathione, or—with well-established cultures—methionine (Maclean, 1982). Rust fungi apparently do not have absolute amino acid requirements beyond these. They can synthesize several amino acids from glucose, either on artificial media (Maclean, 1982) or when growing as parasites (Mitchell and Shaw, 1968; Pfeiffer *et al.*, 1969; Reisener *et al.*, 1970). Despite those indications of minimal amino acid requirements, other evidence suggests that rust fungi take up and utilize many diverse amino acids from their hosts. Reisener and co-workers (Jäger and Reisener, 1969; Reisener and Ziegler, 1970) showed that *P. graminis* takes up arginine, glutamic acid, lysine, and tyrosine from wheat leaf tissue. Furthermore, rust fungi grow best on artificial media containing rich mixtures of amino acids, for example, certain peptones, casein hydrolysates, or a mixture resembling the amino acids of wheat leaves (Maclean, 1982; Mendgen, 1981). With the reservation that nutritional requirements may differ between artificial culture and leaf culture, rust fungi probably grow and sporulate at maximum rates in host tissues when amino acids and amides of many kinds are present in abundant supply.

Glutamine is probably the most important of the amino acids or

amides utilized by rust fungi in host tissues. As noted earlier, it accumulates consistently in rusted tissues. It is readily translocated from place to place within plants and, along with ammonia and asparagine, can be a major product of proteolysis (Lea and Miflin, 1980). Furthermore, glutamine can be the favored source of bulk nitrogen for axenic cultures of *P. graminis* (Maclean, 1982). Glutamine is a precursor for the synthesis of fungal chitin (Farkas and Király, 1961; Raggi, 1974).

Synthesis of several amino acids in higher plants is linked to photosynthesis and photorespiration. For example, glycine and serine are produced directly in the pathway for photorespiration (Tolbert, 1980). Raggi (1975) concluded from $^{14}CO_2$ incorporation into amino acids in rusted bean that decreases in amounts of glycine and serine probably relate to a decrease in photorespiration. Likewise, a decline in amount of alanine was linked to a decline in photosynthesis. In flax rust, Sadler and Shaw (1979b) showed that ammonia is assimilated via the glutamate synthase cycle, which requires reduced ferredoxin supplied by photosynthesis. These examples show that the amounts of some amino acids can relate to activities of chloroplasts, which, in turn, generally decline as part of the progressive autolysis of rusted hosts (Section IV,E). This does not seem to be detrimental to the fungus, which apparently is not highly dependent on the amino acids derived from chloroplast activities.

E. PHOTOSYNTHESIS AND PHOTORESPIRATION

The rate of net photosynthesis in heavily rusted leaves by 8 to 12 days after inoculation is generally reduced to rates one-third to two-thirds those of corresponding uninfected leaves (Doodson *et al.*, 1965; Livne, 1964; Mitchell, 1979; Owera *et al.*, 1981). To determine the actual (gross) rates of photosynthesis, the rates of both dark and photorespiration must be added to the net rate. Although rust is known to increase dark respiration (Section IV,F), its effect on photorespiration is less certain. Both photorespiration and photosynthesis use many of the same enzymes, most notably ribulose-1,5-bisphosphate carboxylase, which catalyzes the first step in CO_2 fixation in photosynthesis. Thus photorespiration usually declines concomitantly with photosynthesis (Kosuge, 1978), as it did with bean rust at high infection densities (Raggi, 1978), and with powdery mildews of beet and oak (Gordon and Duniway, 1982b; Hewitt and Ayers, 1975). However, this pattern has not been found with cereal rusts. Instead, photorespiration increased 1.5-fold with barley leaf rust (Owera *et al.*, 1981) and remained vir-

tually unchanged with wheat stem rust (Mitchell, 1979), whereas photosynthesis declined in both cases.

Although we cannot safely generalize about rates of photorespiration in cereal rusts, declines in net photosynthesis are always greater than increases in dark and photorespiration combined, so that the rate of gross photosynthesis declines. For example, despite an apparently large increase in dark and photorespiration with barley leaf rust, gross rates of photosynthesis were calculated to be 81–89% of rates in healthy leaves (Owera *et al.*, 1981). Gross rates are probably reduced more than this in most cases.

Net photosynthesis was temporarily stimulated in wheat by stripe rust in the first few days after inoculation (Doodson *et al.*, 1965) at 0.5% CO_2. Similar stimulation occurred with powdery mildew of barley at 0.5% CO_2, but not at 0.04% (Edwards, 1970). The increased photosynthesis at high CO_2 concentration was attributed to impairment of glycolic acid oxidase. However, chloroplasts in rusted tissues may temporarily have an enhanced ability to synthesize proteins used in photosynthesis. The normal decline in rRNA within chloroplasts was temporarily retarded with oat crown rust (Tani *et al.*, 1973a). In addition, a temporary decrease in photorespiration could contribute temporary increases in net photosynthesis, as indicated for powdery mildews of oak and pea (Ayres, 1976; Hewitt and Ayres, 1975).

As disease develops into the stage of definite decline in gross photosynthesis, cytological evidence indicates that chloroplasts become degenerate, especially with wheat stem rust (Section II,B). However, rates of photosynthesis start to decline before structural changes are conspicuous. To learn what limits the process in the early stages of the decline, several aspects of photosynthesis have been investigated.

1. *Resistance to diffusion of CO_2 into the leaf.* Diffusion of CO_2 into the leaf was not an important limiting factor for photosynthesis in barley leaf rust (Owera *et al.*, 1981). Resistance to diffusion decreased as pustules broke the leaf surface.

2. *Amounts of chlorophyll.* The amount of chlorophyll declines in rusted cereal tissues (Calonge, 1967; Doodson *et al.*, 1965; Mitchell, 1979), but the amounts of chlorophyll do not correlate closely with

492 W. R. Bushnell

tually unchanged with wheat stem rust (Mitchell, 1979), whereas photosynthesis declined in both cases.

Although we cannot safely generalize about rates of photorespiration in cereal rusts, declines in net photosynthesis are always greater than increases in dark and photorespiration combined, so that the rate of

in gross photosynthesis (Mitchell, 1979). The amount of chlorophyll also declined, suggesting that both factors contribute to the loss in photosynthetic capacity. In contrast, Owera *et al.* (1981) calculated the rate of photosynthesis per unit of chlorophyll to increase substantially because rates of photorespiration were estimated to be high. This illustrates the importance of quantitating photorespiration in interpreting photosynthesis in rusted leaves.

4. *Amounts of ribulose-1,5-bisphosphate carboxylase.* A protein in rusted wheat judged by Wrigley and Webster (1966) to be ribulose-1,5-bisphosphate carboxylase decreased (as noted in Section IV,C). Loss of this enzyme has also been implicated in the decrease in photosynthesis in powdery mildew of sugar beet (Gordon and Duniway, 1982a).

5. *Photophosphorylation.* Although Wynn (1963) detected no change in photophosphorylation with oat crown rust, Buchanan and coworkers found rates of noncyclic photophosphorylation reduced in host tissues with both broad bean rust and powdery mildew of sugar beets (Magyarosy *et al.*, 1976; Montalbini and Buchanan, 1974). Activity was reduced to 70% of that in uninfected leaves. For powdery mildew, the reduced activity was attributed to a reduction in amount of the cytochromes used for electron transport in noncyclic photophosphorylation (Magyarosy and Malkin, 1978). More work on photophosphorylation in rusted cereals is needed, including comparisons with uninfected senescing leaves.

In summation, the reduction in photosynthetic activity in rusted leaves seems to be due to loss of chlorophyll and to key proteins such as ribulose-1,5-bisphosphate carboxylase or possibly cytochromes. Ribosomal RNA within chloroplasts was reduced in amount with oat crown rust (Tani *et al.*, 1973a), suggesting that synthesis of chloroplast proteins may be generally depressed. Increased protein degradation may also contribute to the loss of protein, especially in the late autolytic stage of disease when decompartmentation probably allows hydrolytic enzymes to reach the chloroplast.

F. RESPIRATION

Respiration as measured in the dark can increase severalfold in rusted cereal leaf tissues. This phenomenon was described thoroughly in the 1950s and 1960s when manometric methods for measuring gas exchange were popular and when respiratory pathways in healthy higher plants were being intensively investigated. For reviews of this era see Allen (1959, 1966), Daly (1976), and Shaw (1963).

1. *Combined Respiration of Host and Parasite*

Respiratory rates of heavily rusted leaf tissues are usually two to three times the rates of uninfected tissues with wheat stem rust (Antonelli and Daly, 1966; Heitefuss, 1965; Mitchell, 1979; Shaw and Samborski, 1957), wheat leaf rust (Staples, 1957), barley leaf rust (Owera *et al.*, 1981), or wheat stripe rust (MacDonald and Strobel, 1970). Respiratory increases are first detected about 5 days after inoculation. Tissues excised from pustule centers can have rates 10–15 times those of uninfected tissues (Bushnell, 1970; Samborski and Shaw, 1956), reflecting the intense respiratory activity of the compacted, sporulating fungus. The respiratory quotient (ratio of volume of CO_2 released to volume of O_2 used) of rusted tissues is near 1.0, indicating that lipids are not the principal substrate for respiration (Daly, 1976). The ratio of carbons in respired CO_2 contributed by C_6 and C_1 from hexose substrates (the C_6:C_1 ratio) declines from about 0.5 in healthy tissue to 0.3 at sporulation in rusted tissue (Antonelli and Daly, 1966; Shaw and Samborski, 1957), suggesting that a part of the enhanced respiration occurs by the oxidative pentose phosphate (PP) pathway instead of via glycolysis and the tricarboxylic acid (TCA) cycle. Confirming this, the activities of two key enzymes of the PP pathway, glucose-6 phosphate (G6P) dehydrogenase and 6-phosphogluconate (6PG) dehydrogenase, were found to increase with wheat stem rust (Lunderstädt, 1964; Lunderstädt *et al.*, 1962).

2. *Respiration of the Rust Fungus*

Much, and perhaps most, of the increased respiratory activity in rusted tissues is contributed by the fungus. The PP pathway is known to be important in both rust mycelium (Williams and Shaw, 1968) and urediospores (Staples and Wynn, 1965). The pathway reduces $NADP^+$ to NADPH, which is thought to be used in synthesis of fungal lipids as well as in synthesis of mannitol and arabitol, two of the principal carbohydrates found in rust fungi (Section IV,G). Because the fungus cannot be separated from the host in rusted tissues, the actual rates of fungal respiration cannot be estimated accurately, nor can the proportion of total fungal respiration by way of the PP pathway be determined.

3. *Respiration of the Host*

Before the rust fungus sporulates, host tissues probably undergo respiratory increases of 20 to 30%. Rates are increased 20 to 60% at 5 to 6

days after inoculation (Antonelli and Daly, 1966; Daly *et al.*, 1961), when the amount of fungal mass is small relative to that of the host, and the fungus is therefore not likely to contribute significantly to total respiratory activity. Daly (1976) has emphasized that the C_6:C_1 ratio remains unchanged prior to sporulation, instead of decreasing as would be expected if the fungus with its predominant PP pathway contributed significantly to respiratory activity.

The small, putative respiratory increase in the host prior to sporulation occurs as part of the juvenile host response when senescence is delayed (Section II,A). Auxins and cytokinins, both tentatively implicated in disease-induced senescence delay (Section III,B and C), may induce part of the respiratory increase. Each has increased respiratory rates of uninfected wheat or barley leaves by 20 to 30% (Bushnell, 1967; Daly *et al.*, 1962). Increased concentrations of carbohydrates (Section IV,G) may also cause small increases in respiration.

As the rust fungus sporulates and total respiratory activity of host and parasite increases two- to threefold, respiratory rates in host tissues are suspected to increase. Much of the respiration in the rusted host is probably by way of the TCA cycle coupled to cytochrome electron transport. Evidence for this has been previously summarized (Shaw, 1963; Daly, 1976). In addition to continued activity of the TCA cycle, activity of the oxidative PP pathway is postulated to increase in the host. This has been shown to be the case in powdery mildew of barley, in which most of the fungus can be removed so that host respiratory activities can be measured without major interference by the fungus. The respiratory rates of such host tissues are two to three times those of uninfected tissue (Bushnell and Allen, 1962; Scott, 1965). Activities of G6P and 6PG dehydrogenases increase in the mildewed host (Scott, 1965), indicating that the enhanced respiration is by way of the PP pathway. Furthermore, the respiratory alterations seem to be coupled to changes in chloroplasts. Respiratory increase coincided with onset of chlorosis and decline in photosynthesis in the host (Scott and Smillie, 1966). No increase in respiration or activities of G6P or 6PG dehydrogenases occurred in tissues lacking chloroplasts, even though powdery mildew developed abundantly if the leaves were supplied White's culture medium with sucrose. Postulating that $NADP^+$ lost from chloroplasts stimulated the PP pathway in cytosol, Ryrie and Scott (1968) obtained evidence that $NADP^+$ moved from chloroplasts to cytosol in mildewed tissues, although the separation of chloroplasts from cytosol was incomplete in their preparations.

Several lines of evidence suggest that respiratory activities in rusted hosts are the same as those in mildewed hosts:

1. Activities of G6P and 6PG dehydrogenases were shown cytologically and by enzyme assay to increase in host tissues at the borders of bean rust pustules (Tschen, 1974; Tschen and Fuchs, 1968), an indication that PP-pathway activity had increased in the host. Indirect evidence for increased activity of the pathway in the host was obtained from patterns of enzyme activity in rusted plants with and without potassium deficiency (Lunderstädt and Fuchs, 1968).
2. Respiratory increase coincided with decrease in photosynthesis with wheat stem rust (Mitchell, 1979), as with powdery mildew of barley. To be meaningful, such correlations must be general over a wide range of infection densities, environmental conditions, and cultivars. Indeed this requirement is yet to be met for powdery mildews.
3. Rusts, like powdery mildews, develop abundantly in the absence of photosynthesis if leaves are supplied sugars and other nutrients (Section IV,G). Whether fungus development occurs under such conditions without respiratory increase in the host as reported for powdery mildew is unknown.
4. The amount of NAD increases in diseased tissue with rust (Rohringer, 1964) and with powdery mildew (Ryrie and Scott, 1968). Part of the increase was judged to be in host tissue in both diseases. Ryrie and Scott (1968) suggested that NAD has a role in the breakdown of chloroplasts that was thought to lead to release of $NADP^+$ from the chloroplast.

These findings indicate that the PP pathway is enhanced in rusted tissues and that its activity might be linked to degradation processes in chloroplasts as postulated for powdery mildew of barley.

How would the fungus benefit by increased respiration via the PP pathway? There is no evidence that NADPH or other intermediates of the pathway in the host are utilized directly by the fungus. As noted earlier, the fungus respires via the PP pathway and apparently uses it to supply NADPH for synthesis of lipids, mannitol, and arabitol. These products are unlikely to be synthesized in the host and transferred to the fungus; in fact, mannitol and arabitol do not support rapid rust fungus growth when supplied to rusted leaves in the dark (Silverman, 1960; Samborski and Forsyth, 1960).

Alternatively, enhanced operation of the PP pathway may not be of direct benefit to the fungus, but instead may be only an early manifestation of decompartmentation that eventually leads to host cell autolysis. Some enzymes of the PP pathway increase in activity during senescence of uninfected, detached wheat leaves (Farkas *et al.*, 1964).

Respiratory increase associated with senescence of uninfected tissues excised from barley leaves can depend on light (Allen, 1966), suggesting that senescence-induced respiratory increase is related to photosynthesis, as Scott and co-workers have found for powdery mildew-induced respiratory increase.

For powdery mildew, Scott (1982) postulated that $NADP^+$ enhances the PP pathway either in the cytosol (as noted earlier) or possibly in the chloroplast itself. In addition, activity of the PP pathway can be controlled by amounts of G6P and 6PG dehydrogenases (Turner and Turner, 1980), which could be synthesized as part of a general activation of protein synthesis in rusted host cells.

G. CARBOHYDRATES

"A plentiful supply of carbohydrates to the host is a *sine qua non* for the development of obligate parasites on a genetically congenial host plant or leaf." So wrote P. J. Allen (1954), a statement that still applies accurately to rusts and powdery mildews.

1. *Carbohydrate Requirements of the Rust Fungus*

Rust mycelia in artificial culture can grow on any of several carbohydrates including glucose, fructose, mannose, sucrose, raffinose, cellobiose, and soluble starch (Maclean, 1974). Several indirect lines of evidence indicate that the needs of rust fungi growing as leaf parasites are met by one or more of these carbohydrates, most likely glucose, fructose, and sucrose. Glucose fed to rusted wheat leaves was utilized by the fungus without rearrangement of carbons 1 and 6, indicating that the intact glucose molecule was taken up (Pfeiffer *et al.*, 1969). Glucose, fructose, or sucrose have given the most abundant rust development when supplied to rusted corn or wheat leaves in darkness (Dickson *et al.*, 1959; Silverman, 1960), to rusted albino corn leaves (Dickson *et al.*, 1959), or to rusted wheat leaves in which photosynthesis is inhibited (Mashaal *et al.*, 1981). Finally, Lewis (1976) implicated sucrose as a principal carbohydrate source for *Puccinia poarum* on leaves of *Poa pratensis*, by showing that sucrose infiltrated into rusted leaves specifically inhibited movement of ^{14}C-labeled sucrose from host to parasite.

Whether sucrose is taken up directly by the rust fungus or first hydrolyzed to glucose and fructose is not clear. The amount of invertase in rusted cereal leaves increases severalfold, concomitantly with fungal growth (Lunderstädt, 1966; Mitchell, 1982; Mitchell *et al.*,

1978). Urediospores apparently do not have invertase (Lunderstädt, 1966), but the wheat stem rust fungus grown in artificial culture is thought to hydrolyze sucrose before uptake (Maclean, 1982). Higher plants produce invertase, especially in young leaves or in response to wounding (Long *et al.*, 1975; Lewis, 1976), so that invertase in rusted leaves could be of both fungal and host origin.

Rust fungi do not accumulate host carbohydrates as such but, instead, convert them mainly into arabitol, mannitol, trehalose, and glycogen, none of which are common host constituents (Daly, 1967; Lewis, 1976). Glucitol and ribitol were found in *P. graminis* grown on artificial media (Maclean, 1982). Conveniently, this means that most of the glucose, fructose, sucrose, and starch found in rusted tissues can be assumed to be from the host.

2. *Carbohydrates in the Host*

Sucrose, glucose, and fructose often increase severalfold in rusted cereal leaves as part of the juvenile host response prior to fungus sporulation, and then decline rapidly thereafter (Lunderstädt, 1966; Mitchell *et al.*, 1978; Syamananda and Staples, 1963). Similar patterns occur with bean rust (Inman, 1962) and rust of *Poa pratensis* (Lewis, 1976). The tissues tend to retain photosynthate and to favor import of sugars from distant tissues. Reasons for these changes in sugar translocation patterns in rusted tissues are discussed by Durbin (Chapter 16, this volume). The sugars are later depleted during the autolytic host response as the sporulating fungus uses increasing amounts of carbohydrate.

Starch tends to accumulate along with sugars, so that host tissues within and near pustules stain with iodine (Bushnell, 1970). The starch is found cytologically to be in chloroplasts. However, the amount of starch can show large, puzzling day-to-day fluctuations with cereal rusts (MacDonald and Strobel, 1970; Mirocha and Zaki, 1966). Host tissues seem to shift rapidly between starch synthesis and degradation.

Inorganic phosphate inhibits starch synthesis and favors starch degradation in higher plants (Preiss and Levi, 1980). Supporting this, MacDonald and Strobel (1970) showed a negative but incomplete correlation between fluctuating levels of starch and inorganic phosphate with stripe rust. Inorganic phosphate can be sequestered as polyphosphate by rust fungi, which could favor starch synthesis in host cells (Lewis, 1976; Scott, 1982). Starch synthesis also could be favored by the accumulation of sugars in host cells, which would favor movement of triose phosphate into chloroplasts, which in turn would promote

starch synthesis allosterically (Preiss and Levi, 1980). An unidentified activator of host β-amylase was found in bean rust urediospores, which possibly related to a temporary disappearance of starch soon after bean plants were inoculated with the spores (Schipper and Mirocha, 1969). Similar activators were found in urediospores of *Puccinia graminis*, *P. coronata*, and *P. recondita*.

Uninfected cereal leaves usually do not contain starch. By inducing the host to store starch early in pustule development when carbohydrate supplies are abundant, the rust fungus presumably adds to the total carbohydrate available when supplies are eventually depleted. More work is needed on mechanisms controlling starch synthesis and degradation within host chloroplasts.

V. Concluding Statement

Rust physiologists working in the 1950s and early 1960s were motivated in part by the mysteries of obligate parasitism. How did the dependence of the rust fungus on living hosts relate to metabolic processes in the host and to changes induced therein by the fungus? Were host and parasite intimately linked with respect to metabolic pathways and intermediates? Since then, the culture of rust fungi on relatively simple culture media (Williams, Chapter 13, this volume) has suggested that such complex metabolic interactions may not exist. Instead, the changes induced in the host seem only to ensure a generous supply of simple substrates, principally sugars and amino acids. The diverse investigations of rusted leaves reviewed here have not revealed intimate metabolic interdependencies between host and parasite. Furthermore, the changes in the cereal host are not unique to biotrophic disease as was once suspected. Host responses are well within the normal range of plant capabilities. Events in the host may be delayed, hastened, or amplified, but not changed qualitatively.

A reduction in efforts devoted to understanding the physiology of interactions between rust fungi and compatible cereal hosts has occurred in the last 10 years as emphasis has shifted to specificity and recognition phenomena (Rohringer and Heitefuss, Chapter 7, this volume). Because of the many recent advances in understanding the physiology of healthy plants, a return now to responses in susceptible hosts would appear productive. In special need of investigation are (1) identification of host enzymes that are possibly synthesized in response to infection, (2) changes in photosynthesis in relation to changes in pho-

torespiration and dark respiration, and (3) the role of growth hormones and other substances in controlling alteration within rusted hosts. A better understanding of these phenomena in rusted cereals will ultimately help us manipulate host–parasite interaction to minimize parasite development and yield loss.

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This chapter is dedicated to the memory of Paul J. Allen, who set high intellectual standards for the study of host–parasite interaction and who did so with sensitivity and good cheer.

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