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PLANT–FUNGAL INTERACTIONS: A PLANT PHYSIOLOGIST'S VIEWPOINT

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Key Word Index—Plant–fungal interaction; plants response to infection; recognition; mycorrhiza; lichens; parasitic plants; transport; toxins, phytoalexins; elicitors.

Abstract—The various levels of interaction between plants and fungi from mutualism to obligate parasitism are described briefly. Some of the relations, nutrient transport and respiration are analysed. The possible plant defence mechanisms such as elicitor—mediated phytoalexin formation, secondary metabolites and barrier formation and their energy cost are discussed. It is pointed out that many plant responses to fungi also occur in healthy plants under certain conditions. The requirement for fungal invasion from the point of view of the metabolic mechanisms developed by the invading fungus and the ways in which fungi overcome host defences are outlined. The importance of mutual recognition between fungi and plants is stressed and ways in which these might be related to recognition mechanisms existing within each group of organisms are suggested. Attention is drawn to possible analogies in the relationship between the seed embryo and endosperm, between parasitic plants and their plant hosts and the relation between a fungal parasite and its plant host.

INTRODUCTION

The purpose of this article is to consider some of the features of the interaction between plants and fungi from the point of view of plant physiology, rather than as a problem in phytopathology. It is not intended as a detailed review, but rather as an attempt to approach the problem from a slightly different angle. The purpose is to look at what is common to all the interactions, what is the physiological basis of plant responses and what might be learned from them.

Over evolutionary time a close interrelationship between plants and fungi has developed which encompasses all possible levels of interaction. These range from the fascinating symbiosis or commensualism between algae and fungi in lichens [1], via what is probably a symbiotic state in the prothalli of *Psilotum, lycopodium* and some of the Filicales [2], through the state of coexistence of fungi with plants in roots–the mycorrhizas [3, 4] which is frequently termed mutualism and the unusual endophytic fungi of grasses [5] and onto parasitic fungi, (Table 1). It is difficult to establish any definite sequence in which these interactions evolved or indeed to state with any certainty that they did evolve in a definite sequence. It is by no means clear whether parasitism preceded or followed symbiotic relations or if they occurred more or less simultaneously.

Among the parasitic fungi it is possible to distinguish obligate and facultative parasitism; between fungi which will only attack the living host cells and invade them actively, the biotrophic parasities and fungi which are essentially necrotrophic (necrophytic). The latter are able to and do invade and damage a living tissue, when provided access due to localized damage to the tissue as a result of wounding or damage.

Table 1. Types of plant fungal interaction

Symbiosis or Mutalism		Lichens
		Fern Prothalli
		Mycorrhiza
		Grass endophytes
Parasitism	-Obligate	Necrotrophic
		Biotrophic
	-Facultative	

The fungi involved in the interaction with plants show an amazing heterogeneity in structure, life cycle and metabolic activity. The systematic location of the fungi in the evolutionary scheme is still open to discussion. At one time the fungi were placed together in the same kingdom as plants as is still done in some Botany texts [6], but it is more usual to place them in a separate kingdom [7]. It has been suggested that the fungi do not really constitute a single group which has evolved, but rather are made up of two distinct groups: Oomycetes and Chrytridiomycetes on the one hand and Ascomycetes and Basidiomycetes on the other, which may differ in origin. Classification remains a subject open to discussion [8].

There is evidence that fungi attack and parasitize algae [9, 10] and many marine fungi are associated with algae [11]. Fungi parasitic to all other plant forms are well known, starting with fungi which parasitise bryophytes. There can be no doubt that the fungi are very catholic in their selection of host plants, and that they are able to tackle all forms of plant life effectively (Table 2).

Table 2. Organisms attacked by fungi

Algae	Fungi	Bryophytes
Pteridophytes	Gymnosperms	Angiosperms
	Most Animals	

Although few if any plants are totally resistant to fungi, plants are nevertheless rarely, if ever, completely at the mercy of an invading fungus. Over the evolutionary period of coexistence of fungi and plants, mechanisms have evolved which limit, to a greater or lesser extent, the damage caused to the invaded plant by the fungal pathogen.

Probably one should regard the mycorrhizas as examples of a very successful limitation of the advance of the fungus through the host tissue, which has eventually led to some kind of benefit accruing to the host due to the presence of the fungus. The orchid mycorrhiza illustrate this as the infecting fungal genus, *Rhizoctonia*, can be pathogenic in other plants [3, 4].

The interaction between fungus and plant has many aspects and may be considered from a number of points of view. I will not deal with the genetics of host–pathogen interaction nor with the gene-for-gene theories of virulence, which are outside the scope of the present approach. The use of the techniques of molecular biology and recombinant DNA technology [12–14] will facilitate the study of the host genes responsible for virulence or avirulence [14] and the problems of the gene interactions. More information about the molecular mechanisms responsible for resistance, virulence, and recognition between pathogen and host will be revealed by these approaches [15, 16].

The interaction can be considered in various ways. One way is to look at it from the point of view of the plant responses, another from the point of view of the invading fungus. Both will be discussed and some elements are common to both mentioned.

The cellular structure and the basic metabolism of all plants show a great deal of similarity. Fungi therefore did not have to invent basically new mechanisms or strategies during evolution of the interaction between fungi and plants. Rather they had to modify existing metabolic mechanisms and add on variations to existing pathways. The plant products which the fungi use as the basic elements for their nutrition are the same in all plants, e.g. carbohydrates, amino acids, lipids. The way in which they are synthesized does not seem to make any difference to the fungus, e.g. whether photosynthesis proceeds via the normal Calvin cycle or whether a C4 pathway also exists. The plant on the other hand has to contend with exposure to quite a varied armoury of attacking devices.

A basic element common to all the interactions is the recognition signals between pathogen and host [17]. The existence and importance of such recognition signals is now generally realised and being studied intensively (see later). The entire plant–fungal interaction involves a complex set of developmental steps in which genes in both partners are switched on or off by specific signals. These signals are often effective only at a defined developmental or physiological stage and given environmental conditions and inactive at others.

The host plant will normally only form phytoalexins in response to elicitors, although their formation can be induced by various other means. Elicitors are formed either as a result of the action of fungal degradative enzymes, primarily on the cell walls of the host, or by the action of plant enzymes on fungal wall constituents [18–20]. The host response may be determined by its developments stage and its physiological status. The fungal degradative enzymes are formed at a certain developmental stage of the fungus. The plant degradative enzymes may be activated or their synthesis stimulted by the invading fungus. Elegant immunochemical and other techniques provide evidence for the extreme rapidity with which some of these changes can be invoked [21–23]. Mycorrhizal invasion of many hosts is restricted to the zone just above the tip of young roots, older roots being invaded far less readily. In orchids it is the protocorm which is particularly receptive to invasion, while tubers are not normally invaded [3]. Adhesion of pathogen to the host may be in response to morphological and chemical signals orginating in the host plant. Adhesion proteins have been described in lichens [24] and in bean rust fungi [25].

In considering the response of plants to fungi it is important to distinguish between overall plant response, such as a general stress, on the one hand and limited local responses caused by activation of host defence mechanism, evoked by the invading fungus, on the other hand.

THE PLANT PERSPECTIVE OF THE INTERACTION

As a plant physiologist, I want first to consider what changes can be observed in the fungus-infected plant, a discussion chiefly dealing with higher plants [26–28], since very little work has been done on other groups or divisions of plants, with the exception of lichens, i.e. fungal–algal interactions [1]. Some of the responses are summarized in Table 3.

Photosynthesis and respiration

The photosynthesis of the infected plant is frequently reduced by infection, possibly due to interference in electron transport systems [29]. At the same time respiration generally rises or rather there is increased oxygen uptake. Differentiation between the respiration and oxygen uptake has not always been done carefully. A number of causes could lead to this rise in oxygen uptake; increased requirement for maintenance respiration or

Table 3. Plant perspective

Process	Result of fungal invasion
Plant structure	Localized changes in anatomy and morphology
Photosynthesis	Normally reduction
Respiration (O_2 uptake?)	Localized increase Possible changes in pathways
Translocation	Changed (often chanelled to site of invasion)
Water relations	Changed (either increased or decreased transpiration)
Hormone pattern	Changed (both endogeous, host and fungal synthesis are involved)

increased respiration to initiate and carry out repair processes or non-specific oxygen uptake due to injury, e.g. polyphenol oxidase, or peroxidase-mediated oxygen uptake. Stress is known to alter the ratio between different oxidative pathways, e.g. glycolysis vs pentose phosphate pathway and it may be assumed that fungal invasion causes the same kind of changes.

Translocation and water relations

The presence of a fungus frequently alters the pattern of transport of metabolities in the host [30]. The changes can take different forms. In some cases the presence of the pathogen simply results in the formation of a localized sink to which nutrients are channelled, while at the same time export from the infected tissue is decreased. Necrotrophic and biotrophic fungi differ markedly in this respect. Other changes may be due to changes in cell permeability due to the presence of the fungus.

The pattern of hormone distribution may be changed, either by altered synthesis or transport, which in turn leads to changed nutrient translocation. The pathogen may locally deplete supplies of phosphate ion which can result in very marked, but localized, changes in cellular metabolism. A frequent response observed after fungal infection is increased invertase activity, with decreased sucrose at the site of infection, which can lead to further changes, such as phloem loading followed by altered long distance transport. Many of these long distance transport problems are as yet poorly understood. But the localized changes in transport from host plant cell to invading haustorium are equally important.

Transport in the fungal invaded plants has some special features. In the case of disease causing fungi, nutrients are translocated from the host to the pathogen [31], but in the symbiotic state of equilibrium, e.g. in mycorrhiza, two directional transport exists, from host to fungus for some compounds and in the reverse directon for others [3]. Probably the contact between the fungal haustorium and the host cell is different in these two situations and perhaps the ATPases near the membranes of host and fungus which are in proximity, differ in their function in the two states [32–34]. The interface between fungus and plant cell shows great structural complexity as revealed by microscopic and electron microscopic studies [35]. Transport clearly must also be a problem in the lichens, since here the fungal hyphae do not normally penetrate the algal cells, but envelop them, yet sugars and nitrogenous compounds are transferred [36–38], perhaps by leakage or secretion from one partner followed by uptake by the other.

An interesting comparison might be made between this situation, i.e. fungal haustorium and host and the scutellum and endosperm of the germinating cereal seed [39–41]. Here, too, transport is a central problem, and here, too, the structure and permeability of the cell membrane is a central issue. The papillar cells of the scutellum penetrate the endosperm, secrete enzymes and absorb breakdown products from the endosperm. The whole process is under strict regulation, probably hormonal in nature. Parasitic plants living on their plant host show similar properties to those of fungal parasites and their host. In some parasitic plants, the parasite haustoria invade the cells of the host plant and absorb nutrients from it, e.g. in the case of *Striga*. In the case of *Orobanche*, the parasite grows into the host till it actually reaches the vascular system [42–43].

Water relations and nutrient transport in infected plant [31, 44] and the normal responses of plants in special situations show marked similarities. For example, blockage of vascular systems such as the phloem by callose or xylem elements by tyloses are common phenomena. The former is believed to occur normally at certain periods as well as in response to wounding [45]. The reaction of the host to fungal invasion by blockage of vascular elements is therefore not an unusual response, but it may also be related to resistance to infection [46]. An analogous occurrence is that the xylem is blocked by gummosis in response to infection by *Verticillium* or *Fusarium* [47]. In the normal healthy plant water relations and especially stomatal movement is regulated at least in part by hormones such as ABA. One fungal toxin – fusicoccin is known to reduce stomatal resistance [48] by changing the ionic balance in the guard cells, which leads to increased transpiration, but instances of reduction of transpiration due to infection are also known. Other ways in which the water balance may be altered is by changes in root permeability to water or root death due to the pathogen.

Such effects constitute rather drastic interference in the overall physiology of the host whose precise results are difficult to predict. They are, however, essentially responses of the host to all kinds of traumas, not specifically evoked by a fungal invader.

Structure and hormal relations

Invasion of a plant by fungi is frequently associated with local structural, anatomical or even morphological changes. The first line of defence of the host are the existing barriers; cuticle, bark, cell wall etc. In addition localized restriction of the zone of entry by lignification, thought to be a frequent and general response [49–53] or by the hypersensitive reaction, occurs. The latter, which involves localized cell death, constitutes the sacrifice of some tissue, in order to protect the remaining tissues.

Basic changes in the hormonal balance of the plant are frequently induced by fungal attack. Fungal invasion can induce ethylene production, leading to secondary damage due to the effects of ethylene. Ethylene formation by the fungal pathogen is also known to occur [54]. Similarly IAA production may be enhanced in the plant and fungal IAA production may take place.

The involvement of cytokinins in crown gall, although this is bacterially induced, is well known, but fungal cytokinins have also been described and fungal GA production is perhaps the classical example of modification of the host hormonal pattern.

Too little is known about ABA at present, but fungal production of ABA has been demonstrated, e.g. by *Cercospora* [55] and it is likely that ABA levels too will be modified by fungal pathogens.

Molecular and specialized responses

Some of the observed effects of fungi on their plant hosts can be ascribed to the action fungal toxins, e.g. fusicoccin, helminthosporol and victorin, the ophiolin group of toxins or the controversial wilting toxins [56, 57]. The chemical structure of these toxins and the molecular level at which they act still remains to be elucidated in many cases. The chemical structure of the

very active victorin has been described [58] as have those of the ophiolins and of tentoxin to name a few [59]. The action of fusicoccin to change membrane potential is one of the few definite mechanisms ascribed to toxins. This lack of knowledge is perhaps not surprising since the molecular action of plant hormones, despite knowledge of their chemistry, is still not elucidated either.

The production by the invading fungus of enzymes is another reason for localized changes. In the simplest case such enzymes merely dissolve or damage the cell walls and membranes thereby permitting utilization of the host nutrients by the pathogen simply due to leakage from cells or due to cell death. But in the case of the biotrophic invasion one may regard the effect of the pathogen on the host almost as a local anaesthetic, which permits utilization of compounds in the host without causing too much damage and certainly without causing death of the host. Localized permeability changes must occur, either by changes in the membrane properties or in the pumping mechanisms located in them.

In addition to the very general metabolic changes already discussed, which are part of the overall plant response, many other reactions to the presence of an 'alien' cell are known. Firstly, the plant cell frequently contains ready-made protectants—phenolics, alkaloids, cyanogenic compounds, glucosinolates and others [27, 50, 60]. Most of these are regarded as protectants against predators, but it is not unlikely that they also affect fungi, by slowing down or even poisoning the invader. This is particularly likely because the compounds are generally toxic and a good many basic metabolic paths are common to all living organisms. The inactivation of fungal metabolism can therefore also results from the action of these rather general toxic compounds, provided they are liberated during fungal invasion, from some precursor form or released from the cell compartment in which they may be sequestered.

A second group of compounds are the phytoalexins, which are produced in response to invasion or damage, not necessarily fungal [20, 61, 62]. Normally it is asumed that the phytoalexins are absent, or present at very low levels in unifected plant, although some putative phytoalexins may be present in the uninfected plant. The plant responds to the invader, due to some specific signal, by switching on the metabolic pathways needed for phytoalexin formation. Thirdly plants can and do restrict progress of pathogens physically, as already discussed, by barrier formation of some kind.

We have as yet no evidence for mechanisms such as the formation of the protein inhibitor inducing factor, PIIF [64], evoked in some plants, e.g. potato and tomato, by insect attack, but it would not be surprising if similar responses to fungi also exist.

A special place has always been afforded to phenolic compounds in the protective mechanism, perhaps because no clear alternative function has ever been ascribed to these compounds [50, 52]. The plant also contains an armoury of hydrolytic enzymes which can attack the invader, such as cellulases, chitinases, proteinases and others [65]. The presence of some of these enzymes, e.g. chitinase was at one time uncertain, but their formation by plants is no longer in doubt [66].

Table 4. Fungal perspective

Germination	On or near host
	Utilisation of host
	Metabolites as germination stimulators
	Appresorium formation
	Zoospore attachment
Invasion	Dissolution of plant protective coating
	Neutralization of localized anti-fungal compounds
Growth	Utilization of host metabolites by fungal enzymatic machinery
	Penetration of cell wall ability to alter direction of nurient flow

Energy costs

The host response to pathogens is costly in terms of energy. The formation of ready made, always present, protectants, if they are not needed, constitutes an investment in resources which may never be utilised. The induced production of phytoalexins is also energy requiring. For various reasons it is not really possible to draw up a balance sheet. The changes in metabolism which lead to the formation of secondary metabolities effectively divert metabolites from other processes, while cell death reduces the actvity metabolising mechaniery and therefore less energy is available for plant growth. It must of course be shown that there is an energy deficit and there are no reserves for additonal energy production and growth. This is by no means certain. It is also not clear whether a numerical value can be assigned to the energy requirement of the formation of a given metabolite. Sometimes more than one pathway exists for its biosynthesis. Furthermore some pathways may have a common precursor, which may be diverted from one pathway to an other. Moreover, the energy balance which is significant is that which determines how much energy is left for production of a new generation, i.e. for survival of the species.

If, as a result of expenditure of energy, parts of the plant fail to develop, but flowers and an adequate number of healthy seeds can be produced, then the energy was not really wasted. Intuitively one must conclude that the energy expenditure is worthwhile, since plants under natural conditions are not eradicated by fungal pathogens and since plants and fungi usually coexist without catastrophic results to the plant partner. Rarely are there epidemics, which can wipe out a local population of some species, although such catastrophes can easily occur in cultivated crops

From preceding brief survey it is clear that the basic metabolism of the host plant can be radically modified by interaction with fungal invaders, whether they are pathogen or not. Many of the changes are similar to those occurring in the normal plant as a result of stress conditions, such as water, salt or heat stress. While in some cases the mechanism underlying the change is rather obvious, e.g. fungal hormonal production, this is not so in others.

THE FUNGAL PERSPECTIVE OF THE INTERACTION

The plant–fungal interactions can be considered from a different aspect: how does the fungus cope with the coexistence (Table 4)? Two central problems seem to exist: (i) The actual invasion of the host, be it through wounds or by actual penetration through cell walls and

(ii) the ability to utilize the plant components for its nutrition. Probably the latter presents no major difficulty, provided the cell wall, reserve materials or the proteins are broken down to low M_r compounds, which are the building blocks for fungal metabolism.

Spore germination and its stimulation and inhibition

Generally invasion of the host is preceded by germination of the fungal spores on or near it. In some cases, e.g. zoospores, attachement to the host surface is a critical step. Frequently appressorium formation precedes infection, and is highly organized and regulated [67].

Conditions for germination of the spore and development of the germling are generally well defined and show specific requirements. A moist environment is, of course, one of the conditions required for spore germination, no matter whether other requirements exist. Local topography may be another signal, which starts off germination or at least directs the outgrowth from the germinating spore for example towards stomata and signals the location of the stomata [67, 68].

In the case of fungi interacting with a host it is likely that some kind of signal originating from the host sets off germination although often the only requirement for the germination of fungal spores is the presence of water. Indeed a variety of stimulatory substances has been described [69].

At the same time one expects and indeed finds that certain substances originating in the host can inhibit or prevent spore germination and germling development [70–72]. These substances must be present at or near the surface of the host, whether they are stimulators or inhibitors. In either case, they might be compounds which leak out, or are secreted or originate from damaged cells. Osmotic factors and pH also play a role in determining whether germination or outgrowth will occur [73]. Such factors are part of the signalling system which leads to mutual recognition. The germination stimulating compounds are probably quite specific, while the inhibitors of fungal spore germination probably have very little specificity.

An interesting parallel is observed in the germination of seeds of parasitic plants and haustorium formation by their roots and the signals which regulate fungal spore germination and haustorium formation. The seeds of the parasitic plant *Striga* require stimulants for germination. Following germination, the radicle of the seedlings will form a haustorium near the host root only in response to chemical signals. This chemical signal arises apparently from an enzyme produced by the parasite which attacks cell walls of the host [43, 74].

Penetration

The actual invasion process presents a real problem to the invading fungus. The solution is generally that the fungus excretes a great variety of enzymes, celluloytic, pectolytic, proteolytic and amylolytic as well as the more unusual cutinases [75, 76].

This combined armoury of enzymes is able to degrade or dissolve plant cell walls and the contents of the cells. The full range of enzymes which fungi secrete probably is still not known. These enzymes, after secretion, remain stable and active outside the fungal cell and they are able to resist changes in their immediate, non-cellular environment. This is indicated for examples by stability of many extracellular enzymes in the culture medium, in which the fungi are grown. Such enzymes must be secreted at the right time and in the correct amount. Obviously, since their production is an energy requiring process untimely or excessive production would be wasteful.

Many fungi in addition producing and secreting enzymes also secrete compounds of a mucilaginous nature, sometimes as a sheath, but often as an extracellular compound. Frequently these are polyglucans of some kind. It is therefore possible that such polyglucans create an environment favourable to the activity of the extracellular enzymes, perhaps by maintaining a fixed humidity, a fixed pH and perhaps even a favourable ionic composition in the environment. They also might perhaps absorb, and thereby concentrate, some of the enzymes.

Recognition

The last problem of fungal plant interactions is that of mutual recognition [28, 71, 72] (Table 5). In both plants and fungi recognition is a basic problem during sexual reproduction. In the fungus recognition of mating types, or + and − forms of gametes is well established, although the chemistry is still not fully characterized in all cases. Another example of fungal recognition is provided by the anastomosis groups existing for example in the genus *Rhizoctonia solani* [79]. In plants, recognition between pollen and stigma is the most researched and also the most sophisticated [80]. This recognition is especially interesting because it involves the recognition and rejection of self in the incompatibility reaction, and the acceptance of non-self in the same species. In addition, in plants recognition at the level of gametes also exists, in this case involving the more common rejection of non-self. Interspecific rejection is also known.

Therefore, in both partners of the plant–fungal interaction, recognition of signals in or on the cell walls must exist. It is not surprising therefore that both have also developed means for recognizing each other.

Both plants and fungi are capable of synthesizing a very varied set of secondary metabolites [81–83]. There can be little doubt that these compounds confer some of the specificity in the interactions. The presence or absence

Table 5. Recognition

Plant signals	Elicitors	Fungal signals
	Receptor sites (at cell surface or inside the cell)	
Host response		Fungal response
Phytoalexin formation		Production of lytic enzymes
Liberation of anti-fungal compounds		Lysis of host cell
		Enzymes detoxicating plant defences
Barrier formation		
Hypersensitivity		

of some compounds in the plant harmful to the fungus may mean success or failure of plant defence mechanisms.

The ways in which this mutual recognition is achieved has attracted a great deal of attention. The chemical basis of the recognition appears to involve molecules from both the plant and the fungus. The elicitors of phytoalexin formation are an example, but may constitute a rather special case. Since in many recognition processes glycoproteins or proteoglycans are involved, as expected, in the plant–fungal interaction they also play a part. Certain, well defined polysaccharide wall fragments, the elicitors already mentioned, act as recognition signals.

In addition to the question of the nature of the molecules which serve as the signals, the problem of the location and nature of the receptor sites must be considered, although we have no evidence that specific receptor sites exist in plants cells to which elicitors bind.

No matter where the signals originate from, they must eventually react at the surface membrane of the recognising cell or penetrate it in order to entrain the necessary reaction which follow recognition. A good example, illustrating this is laccase formation in *Botrytis cinerea*. In *Botrytis*, formation and secretion of laccase is induced by the presence of a phenolic compound and induction is amplified by pectins added to the culture medium. Both primary and secondary inducers have maximal effectiveness if added to the growth medium at zero time [84]. Nevertheless enzyme formation and secretion is delayed by several days. During the time which elapses both inducers in the medium will have undergone considerable metabolic change. It may be reasonably assumed that the inducers or products formed from them have entered the fungal hyphae well before enzyme synthesis has begun. The two compounds, or compounds formed from them, therefore, appear to act as developmental inducers, probably in the cell and not at its surface. When *Botrytis* infects a host, these inducers, or compounders similar to them are produced due to breakdown of host cells. In this case it is therefore possible that the inducers signalling for enzyme production are transferred from the plant into the fungal cell. We should not therefore be too dogmatic about searching for receptor sites only at the outer membrane level.

PERSPECTIVE

Where will the study of plant–fungal interactions go in the future? Molecular biology and genetics will solve some of the problems and provide some answers. The techniques of molecular biology will increasingly provide the tools for answering specific questions about the interaction, particularly those concerned with local events, evoked by the interaction at the molecular, cellular and tissue level. But this must be accompanied by a much more fundamental understanding of some of the basic physiology and biochemistry of plants and fungi, including a much better understanding of the interaction at the ultrastructural level, using all the modern techniques of immunocytology. Our knowledge of how plants and fungi work is still very incomplete. Without fully understanding the basic features of both partners in the interaction at the physiological and biochemical level, including the functioning of the intact organism, we cannot understand the interaction itself, whether it is symbiotic, pathogenic or commensual.

REFERENCES

1. Ahmadjian, V. and Hale, M. E. (eds) (1983) *The Lichens*. Academic Press, New York.
2. Smith, G. M. (1955) Cryptogamic Botany Vol. 2. McGraw–Hill, New York.
3. Harley, J. L. and Smith S. E. (1983) *Mycorrhizal Symbiosis*. Academic Press, London.
4. Harley, J. L. (1984) in *Encyclopedia of Plant Physiology* Vol. 17, p. 148, (Linskens, H. F. and Heslop-Harrison, J., eds). Springer, Berlin.
5. Siegel, M. R. Latch, G. C. M. and Johnson, M. C. (1987) *Annu. Rev. Phytopathol.* **25**, 293.
6. Weier, T. E., Stocking, C. R., Barbour, M. G. and Rost, T. L. (1982) *Botany*, 6th Edn. John Wiley, New York.
7. Alexopolous, C. J. and Mims, C. V. (1979) *Introductory Mycology*, 3rd Edn. John Wiley, New York.
8. Webster, J. (1980) *Introduction to Fungi*. Cambridge. University Press, Cambridge.
9. Round, F. E. (1981) *The Ecology of Algae*. Cambridge University Press, Cambridge.
10. Richmond, A. (1986) in *CRC Handbook of Microalagal Mass Culture*, (Richmond, A., ed.), p. 287. CRC Press, Boca Raton.
11. Gareth-Jones, E. B. (ed.) (1976) *Recent Advances in Aquatic Mycology*. John Wiley, New York.
12. Gilchrist, D. G. and Yoder, O. C. (1984) in *Plant–Microbe Interactions*, (Kosuge, T. and Nester, E. W., eds) Vol. 1, p. 69. Macmillan, New York.
13. Kerr, A. (1987) *Annu. Rev. Phytopathol.* **25**, 87.
14. Ralton, J. E. Howlett, B. J. and Clarke A. E. (1986) in *Hormones, Receptors and Cellular Interactions in Plants*, (Chadwick, C. M. and Garrod, D. R., eds), p. 281. Cambridge University Press, Cambridge.
15. Michelmore, R. W. and Hulber, S. M. *Annu. Rev. Phytopathol.* **25**, 382.
16. Halk, E. L. and De Boer, S. H. (1985) *Annu. Rev. Phytopathol.* **23**, 321.
17. Callow, J. A. (1984) in *Encyclopedia of Plant Physiology*, N. S. Vol. 17, (Linskens, H. F. and Heslop-Harrison, J. eds), p. 212. Springer, Berlin.
18. Sharp, J. K., McNeil, M. and Albersheim, P. (1984) *J. Biol. Chem.* **259**, 11321.
19. Davis, K. B., Darvill, A. G., Albersheim, P and Dell, A. (1986) *Plant Physiol.* **80**, 568.
20. Dixon, R. A. (1986) *Biol. Rev.* **61**, 239.
21. Hahlbrock, K., Chappell, J. and Kuhn, D. N. (1984) *Annu. Proc. Phytochem. Soc. Europe* **23**, 71.
22. Hahn, M. S., Bonhoff, A. and Grisebach H. (1985) *Plant Physiol* **77**, 591.
23. Fritzemeier, K-H., Cretin, C., Kombrink, E., Rohwer, F. Taylor, J., Scheel, D and Hahlbrock, K. (1987) *Plant Physiol.* **85**, 34.
24. Bubrick, P., Frensdorff, A. and Galun, M. (1985) *Symbiosis* **1** 85.
25. Epstein, L., Lacetti, L. B., Staples, R. C. and Hoch, H. C. (1987) *Physiol. Mol. Plant Pathol.* **30**, 373.
26. Ayres, P. G. (ed.) (1981) *Effect of Disease on the Physiology of the Growing Plant*. Cambridge University Press, Cambridge.
27. Creasy, L. L. (1985) in *Recent Advances in Phytochemistry* **19**, 47.
28. Daly, J. M. (1984) *Annu. Rev. Phytopathology* **22**, 273.
29. Buchanan, B. B. Hutcheson, S. W., Magyarosy, A. C. and Montalbini, P. (1981) in *Effects of Disease in the Physiology of the Plant* (Ayres, P. G., ed.), p. 13. Cambridge University Press, Cambridge.
30. Whipps, J. M. and Lewis, D. H. (1981) in *Effects of Disease in*

the Physiology of the Plant (Ayres, P. G. ed.), p. 47. Cambridge University Press,
31. Gay, J. L. and Manners, J. M. (1981) in *The Effect of Disease on the Physiology of the Growing Plant* (Ayres, P. ed.), p. 85. Cambridge University Press, Cambridge.
32. Smith, F. A. S. and Smith, S. E. (1986) in *Mycorrhiza, Physiology and Biochemistry*, 1st ESM (Gianiazzi-Pearson, V. and Gianiazzi, S., eds) p. 75. INRA, Raris.
33. Lei, J. and Dexheimer, J. (1988) *New Phytol.* 108, 329.
34. Spencer-Phillips, P. T. N. and Gay, J. L. (1981) *New Phytol.* **89**, 393.
35. Bonfanti-Fasoli, P. (1987) *Symbiosis* **1**, 249.
36. Richardson, D. H. S. (1973) in *The Lichens* (Ahmadjian, V. and Hale, M. E., eds.), p. 249. Academic Press, New York.
37. Millbank, J. W. and Kershaw, K. A. (1973) in *The Lichens* (Ahmadjian, V. and Hale, M. E. eds.) p. 284. Academic Press, New York.
38. Chambers, S., Morris, M. and Smith, D. C. (1976) *New Phytol.* **76**, 484.
39. Negbi, M. and Sargent, J. A. (1986) *Bot. J. Linnean Soc.* **93**, 247.
40. Negbi, M. (1984) *Bot. J. Linnean Soc.* **88**, 205.
41. Akazawa, T. and Hara-Nishimura, I (1985) *Annu. Rev. Plant Physiol* **36**, 441.
42. Riopel, J. L. and Bairs, W. M. (1986) in *Biology and Control of Orobanche*, Proc. Workshop in Wageningen (ter Borg, S. J., ed.), p. 94.
43. Musselman, L. J. (Ed). (1987) *Parasitic Weeds in Agriculture* Vol. 1, *Striga*. CRC Press, Boca Raton.
44. Ayres, P. G. (1984) in *Effect of Disease in the Physiology of the Plant* (Ayres, P. G. ed.), p. 131. Cambridge University Press, Cambridge.
45. Dale, J. and Sutcliffe, J. E. (1986) in *Plant Physiology* Vol. IX. (Stewart, F. C. ed.), p. 455, Academic press.
46. Vander Molen, G. E., Beckman, C. H. and Rodehorst, E. (1987) *Physiol. Mol. Plant Pathol.* **31**, 185.
47. Catesson, A. M. and Moreau, M. (1985) *Is. J. Botany* **34**, 157.
48. Marre, E. (1979) *Annu. Rev. Plant Physiol.* **30**, 273.
49. Ride, I. P. (1975) *Physiol. Plant Pathol.* **5**, 125.
50. Friend, J. (1985) *Annu. Proc. Phytochem Soc. Europe.* **25**, 367.
51. Friend, J. (1981) in *Effects of Disease in the Physiology of the Plant*, p. 179 (Ayres, P. G., ed.), Cambridge University Press, Cambridge.
52. Rhodes, M. J. C. (1985) *Annu. Proc. Phytochem. Soc. Europe* **25**, 99.
53. Hammerschmidt, R., Lamport, D. T. A. and Muldoon, E. P. (1984) *Physiol. Plant Pathol.* **24**, 43.
54. Van-Loon, L. C. (1984) in *Ethylene* (Fuchs, Y. and Chalutz, E. eds, p. 171. Nyhoff- Junk, The Hague.
55. Walton, D. C. (1987) in *Plant Hormones and their Role in Plant Growth* and *Development* (Davies, P. J., ed.). p. 113. Nyhoff, Dordrecht.
56. Durbin, R. D. (ed.) (1981) *Toxins in Plant Disease*, Academic Press, New York.
57. Yoder, O. C. (1980) *Annu. Rev. Phytopathol* **18**, 103.
58. Macko, V., Wolpert, T. J., Acklin, W. and Arigoni, D. (1987) in *Current Topics in Plant Biochemistry and Physiology* Vol. 6, (in press). University of Missouri, Columbia.
59. Strobel, G. A. (1982) *Annu. Rev. Biochem.* **51**, 309.
60. Harborne, J. B. and Ingham, J. L. (1978) in *Biochemical Aspects of Plant and Animal Coevolution*, (Harborne, J. B., ed.), p. 343, Academic Press, London.
61. Bailey, J. A and Mansfield, J. W. (1982) *Phytoalexins*. Blackie, Glasgow.
62. Darvill, A. G. and Albersheim, P. (1984) *Annu. Rev. Plant Physiol.* **35** 273.
63. Knogge, W., Kombrink, E., Schmelzer, E. and Hahlbrock, K. (1987) *Planta* **171**, 279.
64. Ryan, A. C. (1980) in *Curr. Topics Cellular Regulations* **17** 1.
65. Boller, T. (1987) in *Plant–Microbe Interactions* Vol. 2 (Kosuge, T. and Nester, E. W., eds), p. 385. Macmillan, New York.
66. Kurosaki, F., Tashiro, N. and Nishi, A. (1987) *Phyciol. Mol. Plant Pathol.* **31**, 201.
67. Hoch, H. C., Staples, R. C., Whitehead, B., Comeau, J. and Wolf, E. D. (1987) *Science* **235**, 1659.
68. Hoch, H. C. and Staples, R. C. (1987) *Annu. Rev. Phytopathology* **25**, 231.
69. French, R. C. (1985) *Annu. Rev. Phytopathol.* **23**, 173.
70. Sussman, A. S. (1966) in *The Fungi* (Ainsworth, G. C. and Sussman, A. S., eds), Vol. 2, p. 733, Academic Press, New York.
71. Fries, N. (1966) in *The Fungus Spore* (Madelin, E. F., ed.), p. 189. Butterworths, London.
72. Leppik, R. A., Holloman, D. W. and Bottomley, W. (1972) *Phytochemistry* **11**, 2055.
73. Gottlieb, D. (1978 *The Germination of the Fungus Spore*, Meadowfield Press, C. Durham, England.
74. Chang, M. and Lynn, D. G. (1986) *J. Chem. Ecol.* **12**, 561.
75. Kolattukudy, P. E. (1985) *Annu. Rev. Phytopathol.* **23**, 223.
76. Collmer, A. and Keen, N. T. (1986) *Annu. Rev. Phytopathol.* **25**, 383.
77. Ayres, A. R., Goodell, J. D. and De Angelis. P. L. (1985) in *Recent Advances in Phytochemistry* Vol. 19 (Copper-Driver, G. A., Swain, T. and Conn, E. E., eds). Plenum Press, New York.
78. Ralton, J. E., Smart M. G. and Clarke, A. E. (1987) in *Plant and Microbe Interactions* Vol. 2 (Kosuge, T. and Nester, E. W., eds), p. 217 Macmillan, New York.
79. Ogoshi, A. (1987) *Annu. Rev. Phytopathol.* **25**, 125.
80 Heslop-Harrison, J. (1983) *Proc. R. Soc. Lond. B.* **218**, 371.
81. Copper-Driver, G. A., Swain, T. and Conn, E. E. (eds) (1985) *Recent Advances in Phytochemistry* Vol. 19, Plenum Press, New York.
82. Griffin, D. H. (1981) *Fungal Physiology*. Wiley, New York.
83. Callow, J. A. (1983) *Biochemical Plant Physiology*. John Wiley, New York.
84. Marbach, I. Harel, E. and Mayer, A. M. (1985) *Phytochemistry* **24**, 2559.