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19

DEFENCE MECHANISMS OF PLANTS

BY

BRIAN J. DEVERALL

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Preface

This book is concerned with the dynamic mechanisms involved in the defence of plant cells against attack by parasitic bacteria and fungi. Thus I scarcely discuss those plant features such as bark and cuticle which play an obvious role in defence, but which are essentially static contributors. Circumvent these barriers and the ability of apparently undifferentiated parenchyma to defend itself is revealed. Furthermore, this ability is dependent upon particular genes in plant and parasite which interact after infection. My interest is with the processes by which plant cells perceive the approach of an intruder and occasionally permit, but commonly discourage, its further progress. How do the genes of host and parasite communicate to determine the outcome of attempted parasitism? Is there a universal defence mechanism in all plants, and, if so, what is it? What contribution does the much studied process of phytoalexin formation make to the defence of plants?

Research on the physiology of host–parasite relationships has been prolific in recent years and a number of multi-author treatises are being published on different aspects of this work. Hopefully, this monograph will make a useful contribution by presenting a shorter and personal view of those parts of this research which bear directly upon the processes of resistance in plants. My envisaged readership comprises research workers in the subject, and University teachers and their advanced students in plant pathology, botany and plant biochemistry.

I wish to thank Professor A. H. Ellingboe for his suggestions and comments on some of the chapters, Elizabeth Froggatt for typing the manuscript, Stella McLeod for assistance with the References and my family for their tolerance and encouragement.

University of Sydney
February 1976

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CHAPTER I

Introduction to the Host–Parasite Interaction

Plants at all stages of their life-cycles are exposed to many potentially parasitic micro-organisms. Seeds germinate in soils which contain numerous resting parasites awaiting the arrival of roots to stimulate them into activity. Aerial parts of plants are inoculated by fungal spores and bacterial cells carried in air currents and rain-splash droplets. Under favourable conditions of moisture and temperature, plant tissues are thus subjected to attempted infection on numerous occasions. However, these attempts often fail, and most plants remain healthy. Successful establishment of a parasite depends upon a special genetical and physiological relationship so that the cells of the host accept the parasite.

This book is concerned with the processes whereby plants succeed in remaining healthy despite their constant exposure to potential parasites. It leads to a consideration of one of the most interesting problems in biology and biochemistry, namely the molecular basis of the high degree of specialization which is often observed in relationships between parasites and hosts. As will become apparent, there are reasons to believe that the basis of much specialized parasitism rests in the ability of parasites to confound a recognition system linked to other reactions in host cells. Through this linked system, the host normally notices and then fails to accept the intrusion of an alien organism.

INFECTION TYPE, SUSCEPTIBILITY AND VIRULENCE

Before starting to analyse these processes, it is essential to re-emphasize that a disease is the product of the interaction of two organisms, host and parasite. This product can depend upon fine differences in the properties of the two organisms, as is well recognized in research on rust diseases. Thus under

TABLE 1. *The alternative attributes of parasite and host and the result of their interaction*

Attributes of parasites	Attributes of host plants	Resulting infection type
Virulence	Susceptibility	High
Lower virulence	Lower resistance	Intermediate
Avirulence	Resistance	Low

standard and ideal conditions for infection, an isolate of a wheat rust fungus will fail to develop on one cultivar of wheat *Triticum aestivum*, but will produce large uredia on another and an intermediate condition on a third cultivar, comprising, for example, small uredia surrounded by an area of necrosis or chlorosis in the host. These different products are termed *infection types*, and they are determined by the genetic constitutions of the rust isolate and wheat cultivar under particular environmental conditions. A common convention is to qualify infection type by *high* where there is substantial rust development and by *low* where there is little or no development (Loegering, 1966). A low infection type implies that the cultivar was *resistant* to the rust isolate and that the isolate was *avirulent* on that cultivar. High infection type implies the product of an interaction between a *susceptible* host and a *virulent* rust isolate. Thus, resistance or susceptibility as properties of a plant are defined with respect to the response of that plant to infection by a particular isolate of parasite. Similarly, virulence or avirulence as properties of a parasite are defined with respect to success or failure to colonize a particular host plant. Intermediate infection types result from interactions between parasites and hosts possessing lower degrees of virulence and resistance. The interaction between these alternative attributes of parasites and hosts is shown in Table 1.

The concept of infection type will be used in considering all host–parasite interactions in this book, and will not be restricted to the rust diseases.

Many virulent parasites such as most rust fungi grow in an apparently harmonious relationship with their susceptible hosts because they cause no visible adverse reaction in the host cells which they penetrate. It is generally assumed that these parasites derive their nutrients from the living host cells, and they are thus said to be *biotrophic* in their parasitism. By contrast, soft-rot parasites such as the bacterium *Erwinia* in tubers of the potato *Solanum tuberosum* or leaf-spot parasites such as *Botrytis fabae* in the leaves of the broad bean *Vicia faba* kill adjacent plant cells by chemical secretions. These highly successful parasites presumably derive their nutrients from cells which they have killed and thus are said to be *necrotrophic* in their parasitism.

The rust fungi and *B. fabae* are extremely different types of parasite in their relationships with host cells, but it is important to appreciate that some parasites, such as *Colletotrichum lindemuthianum* in bean *Phaseolus vulgaris*, are often biotrophic for part of their development and necrotrophic for the remaining part. Analyses of the features essential to the host–parasite interaction must consider these possibly changing relationships. Very special capacities of the parasite might be anticipated during its biotrophic phase when the integrity and function of living host cells are maintained despite the intrusion of hyphae or haustoria into protoplasts. During this phase, the relationships between the cells of host and parasite can be termed compatible. *Compatibility* will be used in this book to describe harmonious relationships between parasite and host, and *incompatibility* to describe interactions which cause deleterious changes in cells of host and/or parasite.

The use of the concept of compatibililty is thus more restricted here compared with its use by Day (1974) to refer to any host–parasite interaction which gives rise to a high infection type. This restriction might facilitate an analysis of the nature of the compatibility which enables cells of a biotroph and host to live together harmoniously. Quite a different sequence of physiological events might be envisaged to underlie the success, as a parasite, of the virulent necrotroph *B. fabae* in giving rise to a high infection type despite the incompatibility of broad bean cells to the fungus. The concept of compatibility in the host–parasite cellular relationships should therefore be kept distinct from the concept of final infection type.

GENETIC DETERMINATION OF THE HOST–PARASITE INTERACTION

No attempt is made here to give a detailed review of the genetics of the host–parasite interaction, and the reader is referred to the book bearing that title by Day (1974) and to reviews by Ellingboe (1976), Johnson (1976*a*, *b*) and Day (1976) for more complete discussions of this subject. However, as part of a general introduction to the nature of the host–parasite interaction, it is well to recall the widely held generalizations pertaining to the genetic control of the interaction. These arise from results of analyses done by Flor on the inheritance of resistance and virulence in flax rust disease. Different genes conferred resistance in cultivars of flax *Linum usitatissimum* to different races of the rust, and different genes conferred avirulence in rust races to different flax cultivars. The analyses were the basis for the gene-for-gene hypothesis of Flor (1956) which implies that infection type is determined by complementary single genes for resistance in the host and avirulence in the parasite.

TABLE 2. *Dependence of infection type upon genes in host and parasite*

		Parasite alleles for avirulence	
		A	*a*
Host alleles for resistance	*R*	Low	High
	r	High	High

The gene-for-gene hypothesis is considered to apply to many host–parasite interactions, and its basis is illustrated in the simplest form in Table 2 where infection type depends upon the alleles at single gene loci in parasite and host. A low infection type results from the genetic interaction between the dominant alleles for avirulence in the parasite and for resistance in the host. Absence of the dominant allele in either partner results in a high infection type where virulence and susceptibility are expressed. Thus expression of avirulence and resistance is a more particular phenomenon, requiring precise matching of genetic information in both partners.

TABLE 3. *Dependence of infection type upon complementary genes in host and parasite*

		Alleles at gene loci for avirulence in the parasite			
		A1 A2	*A1 a2*	*a1 A2*	*a1 a2*
Alleles at gene loci for resistance in the host	*R1 R2*	Low	Low	Low	High
	r1 R2	Low	High	Low	High
	R1 r2	Low	Low	High	High
	r1 r2	High	High	High	High

The complementary role of alleles at specific gene loci in control of infection type is illustrated in Table 3, where two gene loci are depicted in both host and parasite. Here it can be seen that low infection type results from the matching of specific complementary dominant alleles in host and parasite. Thus avirulence and resistance are expressed only when avirulence gene *A1* matches resistance gene *R1* or when *A2* matches *R2*. Virulence and susceptibility are expressed if avirulence gene *A1* is matched with recessive allele *r1* or dominant allele *R2* in the host. Thus Table 3 emphasizes more strongly than Table 2 that expression of avirulence and resistance is the result of a highly specific genetic interaction.

Many analyses show that resistance in crop plants is inherited as single dominant genes, but relatively few corresponding analyses of inheritance of virulence in parasites have been performed. Putative genes for avirulence are often assigned to races of parasites based on the gene-for-gene hypothesis and knowledge of corresponding genes for resistance in the host. However, exceptions to the major generalizations discussed above are known where susceptibility and virulence are the dominant characters in host and parasite; for example, the relationship between oats *Avena sativa* and the fungus *Helminthosporium victoriae* which will be considered in a later chapter. Polygenic control of resistance towards particular parasites has been claimed in a number of important crop plants, but this phenomenon requires critical genetic analysis under controlled conditions of environment and, in at least two cases, has not withstood this test (see Ellingboe, 1976; Johnson, 1976*b*).

The genetic control of parasitism in natural vegetation is not

well known, and we can only speculate about how resistance and avirulence evolved before man sought resistance genes for incorporation into his crop plants. The selective advantage of resistance to plants seems self-evident, but the role of avirulence in the development of populations of parasites is more difficult to conceive except as a means of separating evolving populations within species.

The most important implications for physiological and biochemical analyses of expression of resistance and avirulence to arise from genetic studies are that infection type is under control of genes in host and parasite, and that the expression of these interdependent properties is often determined by highly specific interactions between particular complementary genes in both partners. Ellingboe (1976) and Johnson (1976*b*) have pointed out that the most specific interactions are usually for expression of resistance and avirulence. The simplest mechanism mediating this expression would be based upon confrontation of primary products of the genes for resistance and avirulence following upon infection. Evidence bearing upon these hypothetical products will be discussed later, but firstly it is useful to consider what is known about stages in parasitism when resistance is expressed. This will be done by assessing the fate of potential parasites at the stages of attempted entry and then early growth into plants. Attempts to understand natural processes of defence in plants should accommodate reports that plants can be cross-protected against normally virulent parasites by previous infection by other organisms. The extent will be sought to which cross-protection is achieved by activation of a process of induced resistance in plants. Any analysis of the process of expression of resistance must assess the contribution of the many anti-microbial chemical compounds in plants, and especially of those which form after infection. Finally, however, it is essential to return to the initial question concerning the nature of recognition between parasites and plants, and to consider the molecular means by which genes in parasite and host interact to determine specificity.

CHAPTER 2

Discriminatory Events before and during Penetration into Plants

Many micro-organisms are dispersed in air currents or in splash droplets caused by rain, and thereby arrive on leaves and stems. Other fungi and bacteria move in soil water before encountering roots or persist as resting stages until roots grow into their vicinity. Plants can then influence micro-organisms around their surfaces by physical and chemical means, thus starting an interaction which must be followed by entry of the parasite into the plant by a specialized route, if parasitism is to have a chance of success. This chapter is concerned with the few attempts that have been made to assess quantitatively the contributions to the success or failure of parasitism of these primary interactions between potential parasites and hosts.

EFFECT OF ROOTS ON PARASITES IN THE SOIL

The principal effect of roots on organisms in the soil is a general stimulation of germination and growth, and this is particularly important for parasites most of which remain dormant until contacted by their living substrates. Fungal parasites lie dormant in soil as a number of different types of resting body such as sclerotia, oospores, chlamydospores, basidiospores and hyphal fragments. Their dormancy is considered to be of two types, constitutive or exogenous (Sussman, 1966). Constitutive dormancy is thought to be maintained by internal factors in the fungus, and is particularly important in basidiospores. Experimentally, constitutive dormancy can be broken, in at least a small proportion of spores, by temperature shocks, treatment with certain chemicals, and proximity to other micro-organisms and some plant roots in culture. Presumably environmental fluctuations in soils cause some of these spores to germinate in favourable seasons. It is also likely that emanations from roots induce

breaking of dormancy of basidiopsores of mycorrhizal fungi (Fries, 1966). Exogenous dormancy is conferred by external factors in the soil. Among the emanations from other soil inhabitants which prevent germination of fungi are ethylene (Smith, 1973; Smith & Cook, 1974) and possibly antibiotics, many of which were discovered as products of soil fungi and soil Actinomycetes in culture, although their role in natural soils remains uncertain. The effects of some of the fungistatic factors in soil are overcome by stimulatory substances which diffuse from plant roots. Many substances exude from the zone of elongation of roots in particular (Schroth & Hildebrand, 1964) and these include mineral salts, sugars, amino acids, organic acids, nucleotides and vitamins (Rovira, 1965).

Reports of stimulatory emanations from plant roots are plentiful, but toxic factors such as the cyanogenic glucoside linamarin in *Linum* (Trione, 1950), and compounds inhibitory to nematodes in *Asparagus* (Rohde & Jenkins, 1958) and *Tagetes* (Winoto Suatmadji, 1969) are also known to occur in roots. These factors or their anti-microbial products may diffuse in the soil. The role of inhibitors in the pre-penetration interactions between roots and parasitic micro-organisms is not well established, as discussed below, but Wallace (1973) considers that such factors may influence parasitic nematodes before they reach the roots of some plants, although he states that resistance to nematodes usually occurs during or after penetration.

There is as yet little evidence that susceptible roots are exceptionally stimulatory to parasitic fungi in the soil or that resistant roots are repressive. As Schroth & Hildebrand (1964) emphasize, the soil environment is complex, and stimulants and inhibitors diffusing from plant roots are likely to affect the activities of many soil inhabitants, including saprophytic antagonists, with unpredictable consequences for the success or failure of a parasitic organism. Some laboratory experiments, which precluded these complexities, indicated that susceptible roots stimulate the formation of infection structures and that resistant roots suppress the germination of spores in a selective way, but the specificity of these effects has been discounted in subsequent work. Thus Flentje (1957) and Kerr & Flentje (1957) showed that the formation of appressoria of *Pellicularia filamentosa* was stimulated by exudates from susceptible roots, but Flentje, Dodman & Kerr (1963) and de Silva & Wood (1964) concluded that there

was little difference between hosts and non-hosts in this phenomenon. Buxton (1957) found that washings from resistant cultivars of the pea *Pisum sativum* inhibited the germination of conidia of *Fusarium oxysporum* f. sp. *pisi*, and then revealed a similar phenomenon with washings from resistant cultivars of the banana *Musa* sp. and conidia of *F. oxysporum* f. sp. *cubense* (Buxton, 1962). However, specificity in the effects of exudates from pea roots on the germination of micro-conidia or chlamydospores of *F. oxysporum* f. sp. *pisi* was not found in the work of Kommedahl (1966). Furthermore, sterile exudates from resistant and susceptible cultivars were equally stimulatory to different physiologic races of the parasite *in vitro* and also when allowed to diffuse through porous blocks into soil containing chlamydospores (Whalley & Taylor, 1973). There is therefore little reason to believe that susceptible roots selectively encourage germination and chemotropic growth of virulent parasites.

The attraction of zoospores of several *Phytophthora* spp., *Aphanomyces euteiches*, *Olpidium* spp. and *Pythium aphanidermatum* to the zones of elongation of different roots has been established, as discussed by Hickman & Ho (1966). Zoospores swim towards, attach to and encyst on the roots. Water-soluble extracts of roots were attractive to zoospores of *P. aphanidermatum* (Royle & Hickman, 1964*a*, *b*) as also were many of the individual compounds known to exude from roots. The only substance in this work which caused chemotaxis, trapping and encystment of zoospores was glutamic acid in the presence of ammonium bases. However, glutamic acid was considered unlikely to act alone in attracting zoospores to roots. Troutman & Wills (1964) found that zoospores of *Phytophthora parasitica* were attracted to negative electrodes, and suggested that electrostatic adhesion might occur to the zone of elongation of roots. Contrary to most research on the response of zoospores to roots, Zentmyer (1961) saw that some zoospores were specifically attracted to host roots and not to non-host roots. *P. cinnamomi* moved towards roots of susceptible cultivars of the avocado *Persea americana* within a few minutes, and encysted and germinated within an hour. Zoospores of this fungus moved less readily to roots of resistant cultivars and were not attracted to roots of the tomato *Lycopersicon esculentum*, tobacco *Nicotiana tabacum* and *Citrus* sp. Similarly, zoospores of *P. citrophthora* were attracted to roots of citrus but not to those of avocado. In