Proc. Indian natn. Sci. Acad. **B46** No. 3 pp. 426-438 (1980)

Theory on the Specificity of Plant Parasites*

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(Received 1 July 1979)

The theory of host-specificity of parasitic micro-organisms states "Inhibitory substances—preformed or post-infectionally formed—in the plants govern the specificity of pathogenic micro-organisms; when the inhibitors are removed or their activity is overcome, the organisms become pathogenic to that particular plant." Parasites have evolved many mechanisms to detoxify/circumvent the biochemical shield—prohibitins and phytoalexins. Parasites overcome the prohibitin barriers by direct enzymatic cleavage, evolution of alternate metabolic pathways, creating unfavourable physiological conditions at which they are inactive, direct interference with the synthesis of prohibitins, lack of reactive components, and tolerance to prohibitins. Parasites have developed mechanisms to circumvent phytoalexins by delaying the synthesis of reducing the accumulation of, and inhibiting the formation of phytoalexins and degrading them enzymatically.

The essence of host specificity lies in the capacity of parasites to overcome its biochemical shield.

Key Words: Parasite, Host, Prohibitin, Phytoalexins, Specificity

Introduction

Much of the recent interest in disease resistance is due to the cooperative endeavours of biochemists, organic chemists and plant pathologists in the isolation and characterization of phytoalexins from infected plants. The interest on preformed inhibitors "prohibitins" (Mahadevan 1970) however, has dissipated in the last decade, although one is deeply impressed by the vast amount of data on the toxic constituents of plants (Mahadevan 1973, Overeem 1976, Schonbeck & Schlosser 1976).

Despite these fascinating developments, the most fundamental question in plant pathology is the basic mechanism that determines the way in which pathogenic

*Memoir No. 298 from the Centre of Advanced Study in Botany

microorganisms specifically infect certain plant species and localize in specific tissues continues to remain elusive.

Host specificity must be expressed through the ability of the organism to resist host defences, to multiply and to damage host tissues. Basically therefore we are concerned with factors that limit microbial growth and multiplication in tissues. Most of the earlier theories on the specificity of parasites were essentially based on the presence of nutrients in host tissues favouring the growth of parasites. These theories and their supporting data were discounted based upon the impressive and substantiating evidences against these theories (Mahadevan 1969, 1973).

But a theory was proposed by me based on the meagre evidences available in 1969 (Mahadevan 1969). Since then, interest on disease resistance has assumed an exponential proportion and the evidences have convincingly substantiated the postulates of the theory. In this paper, I have up-dated the theory.

The theory states "inhibitory substances preformed or post-infectionally formed in the plants govern the specificity of pathogenic micro-organisms; when the inhibitors are removed or their activity is overcome, the organisms become pathogenic to that particular plant" (Mahadevan 1969, 1970).

Three years later, Deverall (1972) without acknowledging the existence of the theory, formulated essentially a similar hypothesis.

Any theory on host-parasite interaction should obey the gene-for-gene interaction postulated by Oort (1944) and confirmed by Flor (1956). This theory clearly obeys the postulates including the operon theory of gene regulation (Jacob & Monod 1961).

In this paper, the various mechanisms evolved by plant parasites in circumventing and detoxifying the prohibitins and phytoalexins of plants are discussed.

Prohibitins Influencing the Specificity of Parasites

Prohibitins are "preformed inhibitory substances that confer some degree of protection to the host plants against microorganism" (Mahadevan 1970). The occurrence of prohibitins and their significance in disease resistance have been well documented (Skinner 1955, Overeem 1976, Schonbeck & Schlosser 1976). As a rule, disease develops in those plants which contain low concentrations of prohibitins which are not toxic to the invading parasites. Evidences in support of this were reviewed (Mahadevan 1978b).

Alternatively the invading parasites must have evolved mechanisms either to tolerate the prohibitins or to detoxify them. In the recent years, this area has gained much attention; admittedly the key for host-parasite specificity lies here.

Enzymatic detoxification of prohibitins: For successful parasitism, the parasite must continuously detoxify the prohibitin of plants, otherwise its very existence is threatened (Mahadevan 1973). The available evidences were summarized earlier (Mahadevan 1973, 1974, 1978b). It will be redundant to review them here, but can be authoritatively stated that during infection, pathogenic microorganisms detoxify the prohibitins of plants by producing an assortment of detoxifying enzymes. Non-pathogens, because they lack the necessary gene complement to produce the enzymes are sensitive to the prohibitins and therefore succumb to them.

Despite the voluminous data on the cell wall degrading enzymes produced by plant parasites, the interest shown by investigators on the enzymes of parasites in degrading the toxic prohibitins has been totally lacking. Fortunately during the last 3 to 4 years, interest in this direction has been certainly encouraging. I am optimistic that

in the morrow, enzymatic detoxification of prohibitins will form an important attraction to biochemical plant pathologists.

We will consider an interesting example: the detoxification of HCN by parasites. In sorghum, the active prohibitin is HCN which is metabolized to formamide by *Gloeocercospora sorghi* by releasing the enzyme formamide hydrolyase (Fry & Munch 1975). Parasites of cyanogenic plants such as *Colletotrichum graminicola, Fusarium moniliforme, G. sorghi, Helminthosporium maydis, H. sorghicola, Macrophomina phaseoli, Mycoleptodiscus terrestris. Periconia circinata Phoma* sp. and *Stemphylium loti* contain formamide hydrolyase (Fry & Evans 1977). Even addition of 10 nM of HCN generally caused at least 50 fold increase in formamide hydrolyase. This enzyme converts cyanide to nontoxic formamide which is partly responsible for the tolerance of the pathogen *S. loti* to high concentrations of cyanide *in vitro* (Rissler & Millar 1977). Furthermore, *S. loti* has developed a cyanide-insensitive constitutive alternate respiratory pathway. This is advantageous to the pathogen. We will agree with Rissler and Millar (1977): the cyanide released from the host, could block the cytochrome pathway in pathogen cells. Subsequently the alternate respiratory pathway could be induced or activated. Growth would not occur but the organism could produce formamide hydrolyase to detoxify the cyanide. Then the cytochrome pathway would function to provide for growth and for increases in activity of formamide hydrolyase to detoxify any cyanide subsequently released as the fungus invaded more host cells.

This is the first of the major research on a novel mechanism of detoxification. This study has ushered a new thinking in plant pathology that pathogens have evolved alternate metabolic pathways to circumvent the toxic action of prohibitins.

Other detoxifying enzymes that have been implicated in the detoxification of prohibitins are glycosidases, phenol oxidase, peroxidase and laccase (Mahadevan 1974). Their significance in pathogenesis has been mentioned and it is my optimistic hope that more number of investigators will enter this fascinating field.

Not only does the detoxifying enzyme remove the toxic barrier in advance of infection, the detoxified product favours the growth and development of the infecting parasite. Consider the example of *Botrytis tulipae. B. tulipae* releases hydroxylic acids from tuliposides (I), which in turn stimulated its growth (Schroder 1972).

Parasites have developed other ways to circumvent the toxic activity of prohibitins. Although these data have been ignored, I have reinterpreted the results and want to stress their significance in specificity.

1. Inactivation of prohibitins in unfavourable physiological state

Tomatin (II) a prohibitin present in solanaceous plants is toxic to microorganism at neutral and alkaline *p*H levels (McKee 1957) *B. cinerea, Corticium rolfsii* and *Monilia fructigena* were quite sensitive to tomatin under neutral or alkaline conditions (Schlosser 1975). These fungi successfully colonize tomato fruits because they lower the *p*H to acidity, at which tomatin is hardly toxic. *C. rolfsii* is a copious producer of oxalic acid (Bateman & Beer 1965). Obviously, *C. rolfsii* will be insensitive to tomatin.

2. Interference with the release of prohibitins

According to Schlosser (1973), a few parasites produce substances that interfere with the release of prohibitins from their parent molecules. For example, leaves of *Hedera helix* contain saponins up to 30% of the dry weight. In intact tissue the major saponins are hedersaponins B and C which

are biologically inactive. When the tissue is wounded, these saponins come in contact with host enzyme system which converts them into highly active A and B-hederin. The hederins are strong inhibitors of pathogenic fungi.

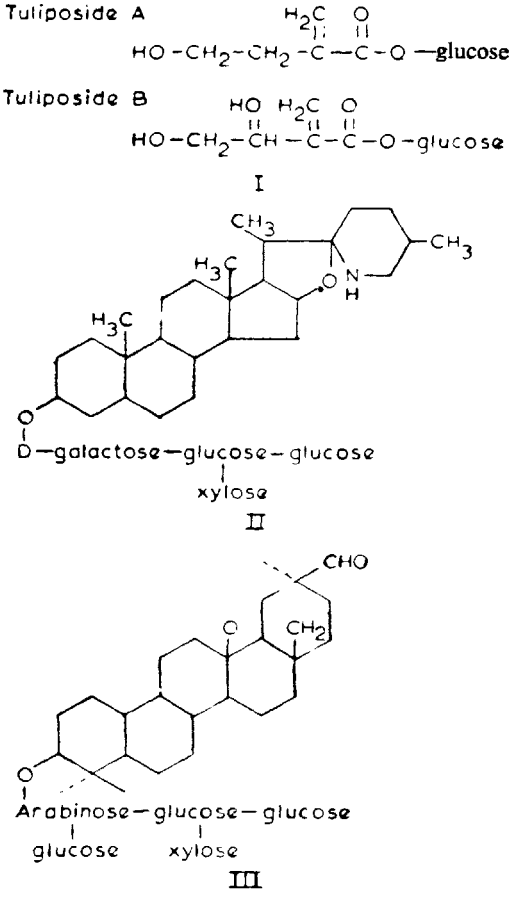
Despite their presence, *Phyllosticta concentrica* and *Pestalotia microspora* colonize ivy tissues. Not that these fungi release any extracellular enzymes that inactivate the saponin. But in tissues infected by these parasites, one or more substances inhibitory to the saponin activating enzyme of the host are present. Presumably, these fungi have the capacity to partially inhibit the enzymatic release of α-hederin from hederasaponin.

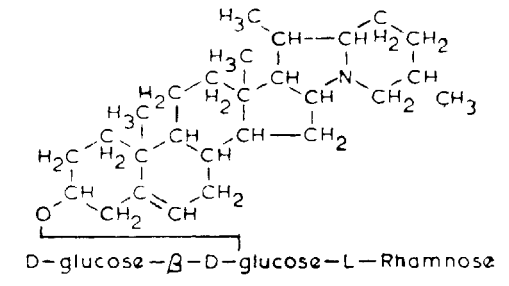
3. Direct interference with prohibitin synthesis

A careful perusal of the literature reveals that a few parasites have developed an indigenous mechanism to interfere with the synthesis of prohibitins. ***B. cinerea*** is pathogenic to *Cyclamen europaeum*. It is insensitive to cyclamin (III), even though it does not produce an extracellular β-glucosidase (Schlosser 1971).

The stem which has only traces of the saponin not sufficient to inhibit the fungus is readily invaded. But the development of ***B. cinerea*** in the stem presumably induces the cyclamin inactivating mechanism which may or may not move towards the leaf in advance of the fungal hyphae. By this, the parasite overcomes the saponin gradient which extends from the stem base to the leaf which is conducive to the colonization of ***B. cinerea*** on the leaf which had been weakened before by the partial or complete block of the vascular system due to the macerating enzyme of the parasite.

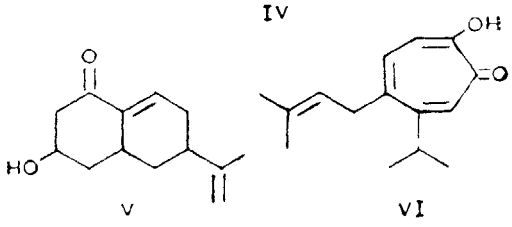
Another example was presented by Ishizaka and Tomiyama (1972). In potato infected by ***Phytophthora infestans*** race 'O' which causes incompatible reaction with the variety "Rishiri", large quantities of solanine (IV) accumulated. In contrast, in the compatible combination, only did traces accumulate. Clearly, infection with the compatible race interfered with the accumulation of solanine. In the fruits of *Capsicum frutescens*, capsidiol (V) increased significantly when inoculated with *Alternaria fumigatus*, *Cladosporium herbarum*, *F. oxysporum*, *Monilia fructicola*, *Penicillium frequentans*, and *Trichoderma viride* (Stoessl et al. 1972). But when inoculated with pathogens like ***B. cinerea*** and ***P. capsici*** there was hardly any increase.





D-glucose-β-D-glucose-L-Rhamnose

IV



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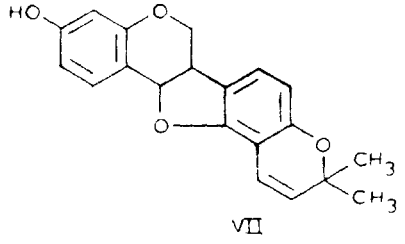

VII

4. Lack of reactive components

A few pathogenic microorganisms have evolved mechanisms by which they lack certain components in their system which make them less vulnerable to the toxic action of prohibitins. Fungi belonging to Pythiaceae family lack sterol in their membranes (Hendrix 1979). These are virtually insensitive to saponins and alkaloids, which primarily react with membrane sterols (McKee 1959). This unique specialization is to the advantage of the parasite.

5. Tolerance to prohibitins

Perhaps due to continued exposure and gradual adaptation to prohibitins, a few parasites have developed tolerance. Isolates of *Poria weirii* from *Thuja plicata* were more tolerant to the extractives than those from *Pseudotsuga menziesii* (Morrison 1969). Evidently the strains from *T. plicata* have adapted to the prohibitins compared with the strains from *P. menziesii*. Similarly, Smith and Czerjesi (1970) found that two "black strain" fungi were highly tolerant to nootkatin (VI). These degraded yellow cedar heartwood, despite the prohibitin.

Admittedly the mechanisms of detoxification of prohibitins are complex. Whether any single mechanism decides the localization of parasite in tissue or several mechanisms jointly restrict the development of parasites on tissues is not clear. Do parasites produce impedins (Mahadevan 1978a) which interfere with the activity of prohibitins? Of course one should keep in mind that no single experimental approach is going to provide definitive answers to the complex detoxification mechanisms.

Phytoalexins Inuflencing the Specificity of Parasites

The significance of phytoalexins in compatible-incompatible reactions was not clear at the time when I formulated the specificity theory, as the available evidences were fragmentary and lacked definitive authentication. Moreover, a few investigators were stubbornly sceptical on the role of phytoalexins in disease resistance. But the recent deluge of papers on the formation of phytoalexins in plants infected by parasitic microorganisms, and their significance in resistance have dispelled doubts and scepticism on the significance of phytoalexins in disease resistance.

A reinterpretation of the published results on phytoalexin formation in plants reveals that compatible combination leads to any one of the following events: (1) Delayed synthesis of phytoalexins; (2) Reduced accumulation of phytoalexins; (3) Inhibition of the formation of phytoalexins; and

Table 1 *Sensitivity of parasites to phytoalexins*

Plant	Phytoalexin	Parasite	Sensitivity	Pathogenicity	Reference
1	2	3	4	5	6
Arachis hypogea	Trans. resveratrol	*Phytophthora megasperma sojae*	+	−	Keen 1975
		Pyrenochaeta terrestria	+	−	
Cicer arietinum	Maackiain, Medicarpin	*Helminthosporium carbonum*	+	−	Ingham 1976
Citrus limon	Xanthoxylin	*Phy. citrophthora*	+	−	Hartmann & Nienhaus 1974
Coffee arabica	Not identified	*Hemileia vastatrix*	+	−	Rodrigues et al. 1975
	Lubimin, Hydroxy	*Monilia fructicola*	+	−	Ward et al. 1976
Datura stramonium	lubimin, Capsidiol				
	Hydroxyphaseollin	*Pseudomonas lachrymans* races 1.5	+	−	Keen & Kennedy 1974
Glycine max	Hydroxyphaseollin	*Col. gloeosporioides*	+	−	Nonaka & Matsuzaki 1976
		F. solani pisi	+	−	
		Xanthomonas phaseoli sojae	−	+	
	Ipomeamarone	*Ceratocystis fimbriata*	+	−	Kojima & Uritani 1976
Ipomea batatas	Dehydroipomeamarone	Non-pathogenic strains			
	Ipomeamaronol	Pathogenic strains	−	+	
Lotus corniculatus	Sativan, vestitol	*Helminthosporium turcicum*	+	−	Bonde et al. 1973
Lycopersicon esculentum	Rishitin	*F. oxysporum* f. *lycopersioi*	−	+	McCance & Drysdale 1975
		V. albo-atrum	−	+	Tjamos & Smith 1975
	Medicarpin	*Helminthosporium turcicum*	+	−	Higgins & Miller 1969, 1970
Medicago sativa		*Helminthosporium* spp.	−	+	
	Maackiain	*Stemphylium botryosum*			
		Verticillium albo-atrum	−	−	Khan & Milton 1975
		V. dahliae	+	−	
Orchis militaris	Orchinol	*Rhizoctonia repens*	+	−	Gaumann & Kern 1959 a, b
		Many fungi	−	+	
	Loroglossol	*Phy. infestans*	+	−	Ward et al. 1975
		M. fructicola	+	−	

1	2	3	4	5	6
Phaseolus vulgaris	Phaseollin	*Colletotrichum lindemuthianum*	−	+	Cruickshank & Perrin 1963
		Monilia fructicola	+	−	
		Rhizoctonia solani	−	+	Pierre & Bateman 1967
		Fusarium solani	−	+	
		B. cinerea	−	+	van den Heuvel &
		C. lindemuthianum	−	+	Glazener 1975
		F. solani f. *phaseoli*	−	+	
	Phaseollin, Phaseollidin, 2'-Methoxy-phaseolin, isoflavan, Phaseollin isoflavan, Kievitone	*F. solani* f. *phaseoli*	−	+	van Etten & Smith 1975
		F. solani f. *cucurbitae*	+	−	
	Phaseollin, Phaeollidin, Phaseollin isoflavan, Kievitone	*Colletotrichum lindemuthianum*	+	−	Bailey 1973
Pisum sativum	Pisatin	*M. fructicola*	−	−	Cruickshank 1965
		Many fungi and bacteria	+	+	
		Ascochyta pisi	−	+	Uehara 1964
		F. oxysporum	−	−	
		Alt. alternata	+	−	Pfleger & Harman 1975
		F. solani	−	+	Nonaka 1967
		F. oxysporum f. *pisi* race 1	−	+	De-Wit-Elshove 1969
		F. solani f. *pisi*	−	+	
		Mycosphaerella pinodes	−	+	
		A. pisi	−	+	
		F. solani	+	−	
		A. pisi (nonpathogenic strain)	+	−	
		Glomerella cingulata	+	−	
		Botrytis fabae	+	−	
		C. lindemuthianum	+	−	
		Cladosporium cucumerinum	+	−	
		Aspergillus fumigatus	+	−	
		Erysiphe pisi	+	−	van Ettan 1973
		E. graminis hordei			
		F. solani	+	−	Christensen &
		M. fructicola	+	−	Hadwiger 1973
		Phytophthora infestans strain 'O'	+	−	Metlitskii et al. 1974
		strains 1,2,4	−	+	Dorozhkin et al. 1975

Solanum tuberosum	Rishitin	*Erwinia carotovora*	+	−	Lyon et al. 1975
	Lubimin	*E. carotovora* var. *atroseptica*	+	−	
	Rishitin	*E. carotovora* var. *carotovora*	+	−	
	Phytoberin	*H. carbonum*	+	−	Duczek & Higgins 1976
Trifolium pratense	Medicarpin, Maackiain	*M. fructicola*	+	−	Cruickshank et al. 1974
T. repens	Medicarpin	*H. carbonum*	+	−	Ingham 1976b
T. subterraneum	Trans-resveratrol	*A. pist*	+	−	Ksendzova & Nilova 1975
Triticum aestivum	Not identified	*Aspergillus pallidus*	+	−	
		M. fructigena	+	−	
		Puccinia coronata avenae	+	−	
		Stemphylium botryosum	−	+	Duczek & Higgins 1976
		S. sarcinaeforme	−	+	
		Phoma sp.	+	−	Sakuma et al. 1976
		H. maydis	+	−	
		Kabatiella caulivora	−	+	
		Stemphylium botryosum	−	+	
Ulmus hollandica	Monsonene E	*Ceratocystis ulmi*	−	+	Overeem & Elgersma 1970
	Monsonene F	*Botrytis allii*	+	−	
		Penicillium italicum	+	−	
		Cladosporium cucumerinum	+	−	
		Aspergillus niger	+	−	
Vicia faba	Wyerone acid	*B. fabae*	−	+	Deverall & Vessey 1969
		B. cinerea	+	−	Hargreaves & Mansfield 1975
		B. allii	+	−	
	Wyerone ester	*B. elliptica*	+	−	
		B. narcissicola	+	−	
	Not characterised	*B. paeoniae*	+	−	
		B. tulipae	+	−	
	Wyerone	*B. cinerea*	+	−	Mansfield & Devarall 1974
		B. fabae	−	+	
Vigna sinensis	Kievitone	*Phytophthora vignae*	+	−	Keen 1975
Zea mays	Phenol ?	*H. turcicum*	−	+	Lim et al. 1970
		H. turcicum (from Johnson grass)	+	−	

(4) Lack of sensitivity to phytoalexins and their detoxification by the infecting pathogen.

In other words, incompatible reaction is characteristic of any of the following: (1) The infected plants rapidly synthesize phytoalexins; (2) Phytoalexins readily accumulate in the infected tissues at concentrations toxic to the pathogen; (3) Uninterrupted synthesis of phytoalexins; and (4) The parasite is sensitive to the phytoalexin and does not detoxify it. Existence of any one of these or a combination of these will necessarily determine the fate of the invading parasite.

1. Delayed synthesis of phytoalexins

Susceptible cultivars respond belatedly in synthesizing phytoalexins. During this delayed response, the invading pathogen has the required "respite" to establish in the host. Consider the following examples. In bean, phaseollin (VII) occurs in both compatible (*Colletotrichum lindemuthianum* B race) and incompatible (γ-race) infection sites, but it occurs much earlier in the incompatible infection zone (Rahe 1973). This occurrence coincided in time with the onset of visible necrotic cells at the infection sites. Similar results were reported in soybean [*P. megasperma sojae* interaction (Keen & Kennedy 1974)], barley [*Erysiphe graminis* f. *hordei* (Oku et al. 1975)].

2. Reduced accumulation of phytoalexins

In compatible host-parasite interaction, the host synthesizes reduced concentrations of phytoalexins which are clearly insufficient to prevent the onslaught of the pathogen. As a result, infection becomes unabated and disease develops. This situation prevails in potato [*P. infestans* (Varns & Kuc 1971)], soybean [*P. megasperma sojae* (Keen & Paxton 1975)], pea [*E. pisi* (Oku et al. 1975)], to cite only a few examples.

3. Inhibition of the formation of phytoalexins

Any process that leads to the inhibition of formation of phytoalexin naturally results in compatibility between the host and parasite. Direct evidences in support of this were presented by Bailey (1973) and Oku et al. (1975). In the bean hypocotyls inoculated with race γ of *C. lindemuthianum* which causes compatible reaction, when incubated at 17°C, spreading lesions appeared. But in the plants kept at 25°C, the lesions were limited. Correspondingly the concentration of phytoalexin in these lesions varied significantly.

Clearly in plants incubated at 25°C, the phytoalexins restrict the hyphal growth of *C. lindemuthianum* not only during hypersensitive reaction but also during the formation of limited lesions (Bailey 1974).

4. Insensitivity to and detoxification of phytoalexins by pathogens

In the recent years, attention has been focused on the capacity of pathogenic micro-organisms to degrade phytoalexins. The evidences are impressive (table 1) and it has become axiomatic that pathogenic micro-organisms are insensitive to phytoalexins and obviously metabolize them more rapidly than do the non-pathogenic forms. By this means, pathogenic strains prevent the accumulation of phytoalexins in the infection court and cause disease.

Conclusions

Admittedly the infecting parasites have developed several mechanisms to detoxify the biochemical shield, prohibitins and phytoalexins of plants. No single mechanism is likely to decide localization and not a single experimental approach is going to provide all the answers. We have just got glimpses of this fascinating and exceedingly

complex field. Although only a handful of investigators are presently involved, it is my optimistic hope that the future offers much scope to Biochemical Plant Pathologists and Molecular Biologists.

The mechanism of specificity of host involves either rejection or acceptance. The moment a plant recognizes the "foreignness", it elicits a series of reactions including the mobilization of prohibitins and synthesis of phytoalexins that culminate in the containment and/or destruction of the foreigner.

Within the framework of the theory, the host specificity of plants can be well explained against bacteria and fungi. Only viral pathogenesis cannot be explained, as these being intracellular parasites nothing is known about the mechanisms of host colonization by viruses. Does virus infection trigger some of the detoxifying enzymes in the host and remove the toxic barrier? We know that some of the viruses are tolerant to a variety of prohibitins (Fukushi 1930). Whether this relationship exists in other viruses? Surely answers to this and related problems will be available in the morrow.

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