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Non-host-selective fungal phytotoxins: Biochemical aspects of their mode of action

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Abstract. During the last decade increasing attention has been directed towards the biochemical mechanisms responsible for the biological activity of phytotoxins. Studies on the mode of action of some non-host-selective phytotoxins, some following on from previous observations, have demonstrated a very specific interaction with particular components of the cell machinery, and have suggested the possible use of these phytotoxins as tools for the investigation of important biochemical processes. This review article reports and discusses results of studies carried out in the 1980s with seven non-host-selective fungal toxins: brefeldin A, cercosporin, *Cercospora beticola* toxin, fusicoccin, ophiobolins, tentoxin, and zinniol. Each of these interferes with the life of the host by interacting with a different biochemical target.

Key words. Non-host-selective phytotoxins; brefeldin A; cercosporin; *Cercospora beticola* toxin; fusicoccin; ophiobolins; tentoxin; zinniol; receptors; plasma membrane; endoplasmic reticulum; ATPase; calmodulin; oxygen radicals.

Introduction

Until 1980, when the state of knowledge of the mode of action of phytotoxins was reviewed by Daly [32], very little work had been devoted to understanding the molecular mechanisms responsible for the toxicity of these plant metabolites. In recent years, interest in this aspect of phytotoxin research has increased, and it is to be hoped that the results will stimulate future investigations.

So far, more attention has been directed towards host-selective than non-host-selective phytotoxins; the obvious involvement of the former in disease makes them more attractive for biochemical and genetic investigations. On the other hand, non-host-specific phytotoxins often act

by affecting specific mechanisms which occur widely, if not ubiquitously, among plants. Consequently, the study of their modes of action can uncover new and unexpected physiological aspects and biochemical mechanisms, so that phytotoxins may be very useful tools for the study of particular physiological processes in plants (e.g., fusicoccin and tentoxin) and/or in animals (e.g., brefeldin A).

The present article is intended to extend to fungal phytotoxins the information provided by Durbin in the preceding article, dealing with the mechanism of action of bacterial phytotoxins. The discussion will be restricted to the biochemical aspects of the mode of action of a limited number of non-host-selective fungal phytotoxins; in fact, although the number of fungal metabolites involved in plant pathogenesis is quite high, the way in which they interact with the cell machinery has been established only for very few of them.

Before any study of the mode of action is begun, it is of the utmost importance to make sure that the phytotoxin is a pure compound, of known chemical structure. At least a partial knowledge of structure-activity relationships will turn out to be very helpful in devising procedures for the preparation of derivatives needed for assay systems, for the detection and the localization of binding sites, etc., namely radioactive, fluorescent or photoaffinity probes, or antigens for obtaining monoclonal or polyclonal antibodies.

Brefeldin A

Brefeldin A (fig. 1), originally isolated from a strain of *Penicillium decumbens* and named decumbin [102, 103], is a biologically active metabolite of a wide range of fungi [108]. Its phytotoxicity was first reported by Suzuki et al. [112]; more recently, its importance in the development of the stem and head blight of safflower caused by *Alternaria carthami* has been established [117].

Brefeldin A has not been found to cause electrolyte leakage from discs of safflower leaf or of carrot tissue, or to affect the integrity of safflower leaf protoplasts, or the membrane potential of cultured safflower cells. Thus the plasma membrane is excluded as the primary site of action of this toxin [117]. The suppression by brefeldin A of polyacetylenic phytoalexin biosynthesis in cultured safflower cells challenged with an appropriate elicitor is of remarkable interest for studies of the development of the disease caused by *A. carthami* [115], but is probably the consequence of a mechanism unrelated to that responsible for the induction of toxic symptoms [116]. The availability of ^{14}C- and ^{3}H-labeled toxin and of specific anti-brefeldin A antibodies [116] will be of great value for further studies in this area.

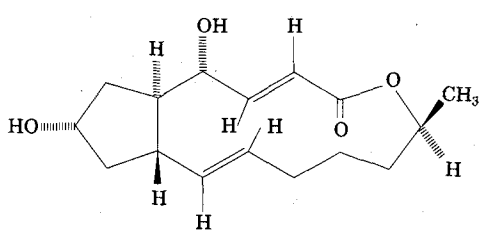

Figure 1. Brefeldin A.

Future investigations directed towards the identification of the cellular target of the toxin should consider the possible effects of brefeldin A on the secretory pathway through the endoplasmic reticulum and the Golgi apparatus. In fact, in animals the use of brefeldin A to perturb intracellular traffic [81, 83, 120] has produced new information about this fundamental process [48, 66, 67, 122]. Until now its action on glycoprotein processing in plants has not been explored.

Cercospora beticola toxin

Cercospora beticola toxin (CBT) is the major phytotoxic metabolite of the fungus causing the leaf spot disease of sugar beet [97]. It is a yellow compound not to be confused with the red cercosporin (see the next section). It exhibits antibiotic activity against bacteria, impairs root formation in tomato cuttings, and when infiltrated into sugar beet leaves it induces the necrotic lesions characteristic of leaf spot disease [97, 98]. The structure of the toxin is currently being investigated in Milan by Assante, Merlini and Nasini, who have generously donated samples of the compound to several research groups in Italy and France.

Evidence so far accumulated suggests that this toxin has an effect on the plasma membrane ATPase. Proton pumping is inhibited by low concentrations of CBT in microsomal vesicles from both pea stems [71] and maize roots [22]. In the latter vesicles, as well as in microsomal preparations from maize coleoptiles [68, 119], the hydrolytic activity of the ATPase is also inhibited by CBT [22].

Experiments with purified preparations of H^+-ATPase from maize roots reconstituted into artificial proteoliposomes [96] by the octylglucoside dilution procedure [90] have confirmed that on addition of CBT the ATP-driven proton translocation is strongly inhibited; the maximal velocities of proton transport and of ATP hydrolysis are independent of toxin concentration. The inhibition follows Michaelis-Menten kinetics and could be due to the competition of CBT with the enzyme for substrate binding, thus conforming to a mechanism which involves the direct interaction of the toxin with the proton pump of the plasma membrane.

The inhibition by CBT of the binding of fusicoccin to its specific receptors [119] was not confirmed in recent competition experiments carried out with a very pure sample of toxin which was quite active in inhibiting ATP-driven proton transport and ATP hydrolysis in maize root preparations (Ballio et al., to be published). The new results are consistent with the great structural dissimilar-

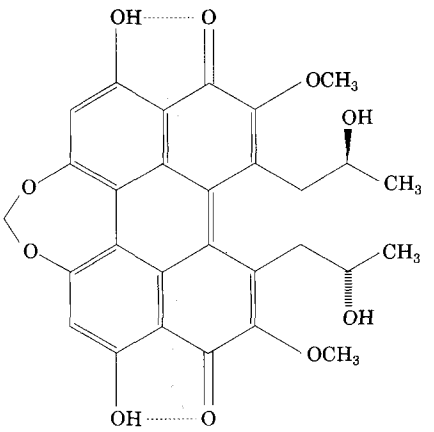

Figure 2. Cercosporin.

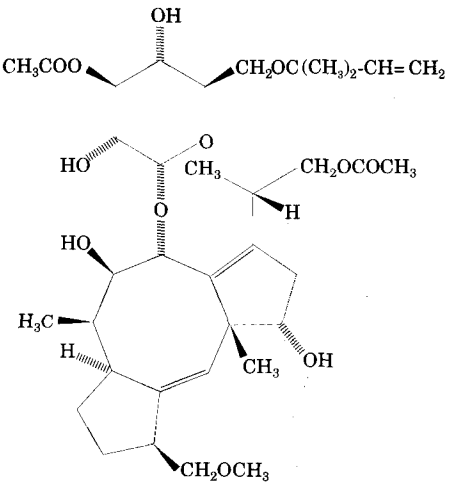

Figure 3. Fusicoccin.

ity between fusicoccin and CBT (G. Nasini, personal communication) and with the different type of interaction of the two toxins with the plasma membrane proton-transport system[96]. The complete structural characterization of CBT, followed by structure-activity correlation studies, should allow the preparation of a radioactively labeled active derivative which could be used to obtain conclusive results on the above-mentioned effects.

Cercosporin

Cercosporin (fig. 2) is a member of the relatively large group of naturally occurring perylenquinones[125], which includes several phytotoxic fungal metabolites. It was first isolated from the soybean pathogen *Cercospora kikuchii*[62], and thereafter from many other species of *Cercospora*[125].

Cercosporin is a non-selective toxin suspected of being involved, together with CBT, in *C. beticola* leaf spot disease of sugar beet[11,45,124] and in other plant diseases. It has been isolated from plants infected by species of *Cercospora*[45,62,124], and when applied to a wide range of host species it reproduces many symptoms of the disease. It was observed that cercosporin kills plant cells rapidly only in the presence of light[69,128], which was the first evidence that its toxicity might be related to photodynamic properties. In fact, on illumination cercosporin produces singlet oxygen[33,40], as well as superoxide anion[36,53]. The amount of the first is decreased by reducing agents with concomitant initiation of superoxide production – a modulation which might yield fluctuating amounts of these species of active oxygen. Both species are probably responsible for the lipid peroxidation observed in vitro[25] and in vivo[34,35] and consequently for the membrane damage observed in plant cells[106,107].

Several other phytotoxic perylenquinones and partially reduced perylenquinones are produced by pathogenic strains of *Cercospora*, *Alternaria*, and *Stemphylium* species[58,70,95,125]. Only for some of them have photodynamic activity[54,70] and a production of active oxygen species[54] been demonstrated.

Fusicoccin

Fusicoccin (fig. 3) is the major phytotoxic metabolite of *Fusicoccum amygdali*, the causative agent of peach and almond canker. It was isolated in 1962[15] and its structure reported in 1968[14,21]. The leaf wilt caused by the pathogen is reproduced by the pure toxin, which induces the opening of stomata and consequently impairs the water balance in the infected plant[121]. The mechanism by which fusicoccin affects stomatal movement is the same as that which is responsible for the promotion by the toxin of growth by cell enlargement, seed germination, and the transport of ions and other solutes across the plasma membrane of higher plant cells[74]. Circumstantial evidence led to the proposal that fusicoccin acts directly and primarily at the plasma membrane level, where it selectively activates the H^+-ATPase responsible for electrogenic proton extrusion, with consequent influence on the activity of a number of metabolic and physiological processes[74–77]. Recently, several research groups have produced results which strongly support the above hypothesis. It has been shown that in vesicles isolated from radish seedlings[38,91,92] and broad bean tissues[23] the hydrolytic and H^+-transfer activities of the plasma membrane H^+-ATPase are stimulated by fusicoccin acting at the membrane level. Direct evidence for the involvement of the plasma membrane ATPase in the fusicoccin-induced membrane hyperpolarization has been obtained by showing that in tobacco protoplasts anti-ATPase antibodies completely inhibit the response induced by fusicoccin[20]. Furthermore, evidence has been produced that the stimulation of proton extrusion is triggered by a signal that originates from the interaction of fusicoccin with specific high-affinity receptors detected in plasma membrane-enriched fractions of a large number of higher plants, and located at the apoplastic side of the plasma membrane[7,46]. These receptors have been characterized in tissues of several monocots and dicots[1,17,39,41,46,79,109,110], and partially purified, either

in their original state[37], or after photoaffinity labeling[47]; enzymatic inactivation tests have demonstrated that they are phosphorylated glycoproteins[4].

The influence of the phospholipid environment on some kinetic properties of the receptors solubilized from maize[8] and spinach[5] tissues has been investigated by inserting them into liposomes. A marked stimulation of proton pumping was observed on addition of fusicoccin to liposomes which had incorporated both a partially purified plasma membrane H^+-ATPase from maize roots or shoots and a crude preparation of fusicoccin receptors from maize shoots[1,3]. This result provides convincing support for the functional relationship between fusicoccin receptors and the H^+-ATPase, already evidenced by the above-mentioned specific stimulation of the ATPase activity and proton pumping in isolated membrane vesicles from radish[38,91,92] and from broad beans[23]. This dual reconstituted system might become a useful tool for the study of the signal transduction following fusicoccin binding to its receptors, a process which is still obscure except for the preliminary evidence favoring the participation of inositol-1,4,5-triphosphate[2], and the involvement of at least one protein between the receptors and the ATPase[38].

In 1985 it was proposed[52] that the stimulation by fusicoccin of the plasma membrane proton-extruding system might be ascribed to a reduction in cytoplasmic pH resulting from the enzymatic liberation of acetate from the toxin. Clear-cut experimental evidence against this ester hydrolysis mechanism has been reported recently[99]. Older results obtained in the course of studies on structure-activity relationship[16,18] had already left little doubt of the irrelevance of acetyl groups in the mechanism of fusicoccin action. In fact, dideacetylfusicoccin, several minor non-esterified metabolites isolated from *F. amygdali*, and the cotylenins, which are non-esterified fungal metabolites closely related to fusicoccins[12], retained the in vivo and in vitro activities typical of fusicoccin.

Research on fusicoccin receptors, besides demonstrating their prominent role in the mode of action of the toxin, has led to the detection in higher plants of endogenous ligands that compete with fusicoccin in specific binding to microsomal preparations[6]. The existence of these compounds offers an explanation for the paradoxical contrast between the very broad occurrence of fusicoccin receptors in the tissues of higher plants and the exceedingly limited number of plant species that can serve as hosts for the fusicoccin-producing pathogens. The endogenous ligand – probably a single compound – has been purified by HPLC[13] and by immunoaffinity chromatography[72,73], but the minute concentration found in all plant tissues so far examined has frustrated efforts to determine its chemical identity.

Ophiobolins

Studies on the mode of action of these sesterterpenes have mainly concerned ophiobolin A (fig. 4), the first-

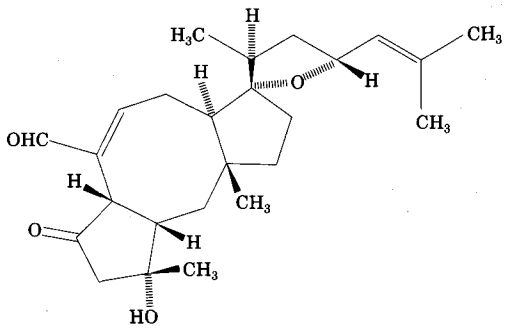

Figure 4. Ophiobolin A.

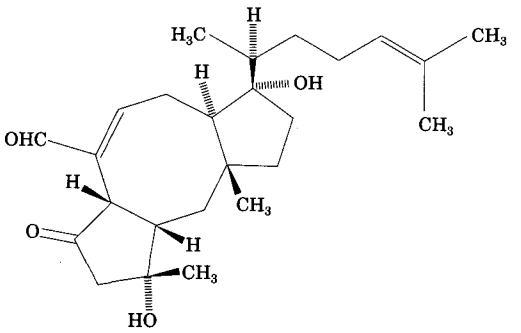

Figure 5. Ophiobolin B.

discovered member of this group of phytotoxins[85]. Ophiobolin B (fig. 5) is the immediate biogenetic precursor of ophiobolin A from which it only differs by its higher content of four hydrogen atoms. The probable role of ophiobolin A in the development of brown spot of rice is supported by its presence in rice leaves infected by *Cochliobolus miyabeanus*[84]. Early studies on its toxicity towards tissues from both monocots and dicots indicated effects on cellular permeability[26,86,118], probably caused by the inhibition of specific membrane transport processes[118]. These results, as well as those showing interference with the fusicoccin-stimulated proton extrusion activity, and the effect of ophiobolins A and B on K^+ permeability, have suggested that ophiobolins and fusicoccin interact at the plasma membrane level[27,50,75]. It is likely that the occurrence of a similar carbotricyclic system in ophiobolins and fusicoccins has been a factor leading to this suggestion[50,51,75]. More recently it was shown that ophiobolins A and B do not compete with fusicoccin in the binding assay to fusicoccin receptors (Ballio, unpublished results).

Furthermore, it has been demonstrated that in vitro ophiobolin A irreversibly inhibits the calmodulins of bovine brain, of maize and of spinach[64,65]. The inhibition is correlated with toxicity on excised maize roots and depends, at least for bovine brain calmodulin, upon reaction of the toxin with the epsilon-amino groups of lysine residues[63]. Thus, consideration must be given to the possibility that in the plant cell the ubiquitous calcium-bind-

ing calmodulin is a target molecule for the action of ophiobolins. Since the Ca^{2+}-calmodulin complex is responsible for the regulation of a number of cellular functions, the interaction of ophiobolins with calmodulin might bring about many different disturbances of the cell machinery. One example is the enhancement by ophiobolin A of stomatal opening[82], namely of a Ca^{2+}-inhibited process[100,127]. As often happens, a broad activity spectrum arises from a specific biochemical reaction.

Tentoxin

Tentoxin (fig. 6) is a cyclic tetrapeptide produced by the plant pathogenic fungus *Alternaria alternata*[49,80], which causes chlorosis in seedlings of several higher plants[49,113]. Early literature on the mode of action of tentoxin is discussed in an excellent 1980 review article[32] that particularly stresses the relation between the development of chlorosis and the inhibition of photophosphorylation[9], and the role of the CF_1 domain of the chloroplast coupling factor as primary target of the toxin[105]. More recently, some elegant experiments have indicated that the beta subunit of CF_1 might be the target of tentoxin[93]. In fact, the successful reconstitution of native and hybrid *Rhodospirillum rubrum* chromatophores, containing the native beta subunit and that isolated from CF_1, respectively, provided two types of F_0-F_1 complexes. Both were catalytically active, but only the hybrid one was sensitive to the toxin.

The observation that tentoxin induces stomatal closure with loss of K^+ from the guard cells[43,44] was suggestive of another site for toxin action[75], a hypothesis which until now has not gained convincing support. Negative evidence for the interaction of the toxin with the plasma membrane has been produced[24]. A recent extended investigation of the effects of tentoxin on stomatal movement has yielded results which favor the inhibition of photophosphorylation as the primary cause of stomatal closure[31]. The same investigation has also ruled out a role for tentoxin as a K^+-carrier. This role was suggested by its interaction with lipid bilayer membranes[59], but later put in question by experiments on biological membrane systems[29,30]. Similarly, the formation of transmembrane ionic channels, observed on the incorporation of tentoxin into a lipid bilayer[55,56], might be of negligible relevance in in vivo systems.

The widely-accepted conclusion that the primary site of action of tentoxin is the CF_1 domain of the chloroplast ATPase has made this toxin a useful tool in some functional and biochemical studies with chloroplasts[57,60,87–89,93,101]. The discovery that plastids of tentoxin-treated plants are deficient in polyphenol oxidase activity[123] has provided a way to determine the function of this enzyme in phenolic metabolism. In fact, it was found that elimination of polyphenol oxidase activity in developing mung beans by tentoxin had no effect on the type and amount of caffeic acid derivatives[42]. This result was followed by the discovery in tentoxin-treated mung bean seedlings of a membrane-bound *p*-coumaric acid hydroxylase, a new enzyme probably involved in the biosynthesis of phenolic compounds in those tissues[61].

Zinniol

Zinniol (fig. 7) has been isolated from cultures of several plant pathogenic species of *Alternaria*[19,28,104,126] and more recently from *Phoma macdonaldii*, as well as from tissues of sunflower inoculated with this pathogen[111]. ^3H-labeled zinniol, synthesized according to a two-step procedure[78], was used to demonstrate the occurrence of specific binding sites in protoplasts and microsomal preparations of carrot cells[114]. The binding was saturable and reversible, and was much decreased in cell lines resistant to the toxin. It was shown[114] that occupancy of the binding sites results in the stimulation of Ca^{2+} uptake by protoplasts, with an optimum at 0.1–1.0 μM concentration; again, the resistant cell lines were much less sensitive. Experiments in competition with Ca^{2+}-channel blockers showed an interference at the plasma membrane level with the functioning of Ca^{2+}-channels. Some of these were affected by both the toxin and the blockers, and others by the toxin only. The increased intracellular Ca^{2+} concentration consequent to zinniol stimulation of Ca^{2+} uptake may upset some important Ca^{2+}-regulated processes and thus explain the toxic effects of the compound.

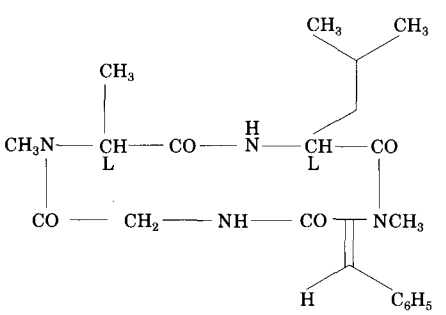

Figure 6. Tentoxin.

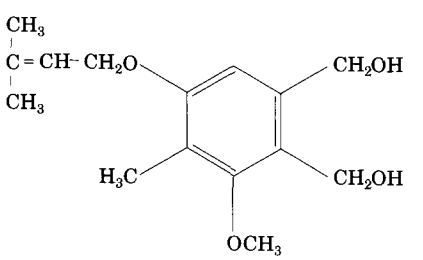

Figure 7. Zinniol.

Further studies on the zinniol target might shed new light on Ca^{2+} transport in plants, and zinniol could become a useful tool for investigating this process.

An unexpected chlorophyll retention effect 'green islands', in cereal leaf sections, produced by zinniol, was reported some years ago[94]. The effect, which could not be ascribed to increased levels of cytokinins or cytokinin-like compounds during zinniol treatment, was induced by relatively high (mM) amounts of toxin, and, until now, has been observed only in three cereal species resistant to the toxin. The biochemical significance of these data is still unexplained, as is their relevance for plant pathogenesis.

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