Multi-author Review

Phytotoxins and their involvement in plant diseases

The Editors wish to thank Prof. A. Ballio and Prof. A. Graniti for coordinating this multi-author review.

Phytotoxins and their involvement in plant diseases. Introduction

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Abstract. Bacterial and fungal plant pathogens are known to produce diffusible toxins in infected plants. These phytotoxins are harmful to plants in very low concentrations, and many reproduce at least some of the symptoms of the relevant bacterial or fungal disease. Several phytotoxins have been shown to be involved in pathogenesis. Recent years have seen substantial progress in our knowledge about the nature, structure and mode of action of phytotoxins, and this is briefly reviewed. Finally, possible applications in fields other than plant pathology are mentioned.

Key words. Toxins; phytotoxins; plant diseases; plant pathogenic fungi and bacteria.

The assumption that chemical factors are involved in plant diseases caused by pathogenic microorganisms dates back to the second half of the 19th century; that is, to the time when the microbial etiology of several plant diseases was experimentally demonstrated. This assumption was first made for infectious plant diseases whose symptoms could not be related to a direct effect of the pathogen on the host tissues. In such diseases chlorosis, necrosis, rotting and wilting often occur in advance of the invading fungal hyphae or bacterial cells, and symptoms may be shown by plant organs located at a distance from the site of infection and even far away from it.

Following the successful attempt made by de Bary[15] to reproduce soft rot by applying a sterile extract from rotten carrots to healthy carrot tissues instead of the pathogen, the cell-wall degrading enzymes produced by plant pathogenic fungi and bacteria were investigated. It is still controversial whether pectolytic enzymes are toxic per se, being able to cause cell death independently from their effect on the structural integrity of the plant cell-walls. Recent studies[9, 28] indicate that the macerating activity of purified preparations of microbial polygalacturonase and pectate-lyase is lost or becomes undetectable at certain pH values or in the presence of an inhibiting protein (PGIP), whereas the killing activity on plant cells is maintained. Therefore, if toxicity is not due to contaminants or other molecules, the enzyme does also act as a toxin.

Like enzymes, plant growth regulators produced by pathogenic microorganisms, for example IAA from the olive knot bacterium *Pseudomonas syringae* pv. *savastanoi*[65, 66], are not regarded as toxins[52]. On the other hand, the fact that some microbial metabolites, like fusicoccin, may mimic or reproduce the effects of plant growth hormones, does not exclude the possibility that they act as toxins in the diseased plant.

There is little agreement on how toxins should be defined in plant pathology, and which classes of active substances they include, and this has been discussed several times[12, 24, 25, 32, 43, 53, 56, 57, 68]. Most reviews on the subject begin with a discussion on the meaning of toxins in relation to the diseased plant. This is mainly due to the constant evolution of the toxin concept in plant pathology, and to the advancement of our knowledge of the function and mode of action of the various kinds of substances produced by pathogens in the infected plant. As this knowledge progresses, terms need to be redefined, synonyms discarded, and definitions adjusted or simplified. The term 'phytotoxins', for example, although ambiguous (the same term has been used for substances produced by higher plants which are toxic to animals) has been widely used in the literature to stress the fact that toxicity is chiefly – or – solely exerted on plants.

However, more important than defining terms is to point out what confers on a substance the character of a toxin. In plant pathology, substances described as toxins are usually microbial metabolites that are harmful to plants at very low concentrations. Many plant pathogenic bacteria and fungi produce phytotoxins in culture and in their hosts. In several cases, especially if they are produced during the early stages of plant disease development, these compounds have a function in pathogenesis and reproduce some or even all of the symptoms of the disease caused by the producer organisms. Unlike microbial toxins excreted by animal or human pathogens, most phytotoxins are small molecules and have a variety of structures, many of which are quite unusual. They are able to diffuse from the site of infection into the surrounding tissues, or are translocated within the plant *via* the apoplasts.

For microbial products which play a role in pathogenesis but have no intrinsic toxicity for plant cells the term 'phytoaggressins' has been proposed[25].

A large number of toxins, often produced as mixtures of related or unrelated substances, have been extracted

from cultures of many bacterial and fungal pathogens, but only some of their structures have been elucidated. The virulence of plant pathogens, and sometimes their pathogenicity, may depend on their ability to produce one or more toxins and to use them as chemical weapons [18, 43, 44, 54].

A limited number of toxins, the so-called host-selective (or host-specific) toxins, are known to affect only plants of a genotype susceptible to the pathogen. At low (physiological) concentrations they reproduce the symptoms of the natural infection. Cultivars resistant to the pathogen are not damaged even by relatively high concentrations of the toxin. Mutants of the pathogen lacking the genes for toxicity (Tox^-), i.e., unable to produce toxin, are non-virulent [13, 14, 23, 35, 45, 47, 48, 56 – 58, 73, 74].

A great number of non-selective phytotoxins are known which do not reproduce the patterns of resistance and susceptibility of the host to the pathogen. They include all the bacterial phytotoxins known so far and the majority of the fungal toxins. Because these biologically active substances are broad-spectrum toxins which can affect many economically important crops, their total impact on agriculture may be very serious [3, 4, 17, 18, 27, 44, 53, 63, 72].

For a long time, the role of toxins in pathogenesis in plants remained controversial. Although in culture many pathogenic bacteria and fungi were shown to produce extra-cellular metabolites which could mimic plant disease symptoms, for many years the inability to purify and characterize the active substances limited the interpretation of biological experiments designed to elucidate the role of these substances in causing disease.

Preceded by a number of early studies, and some intuition [67], research on phytotoxins was initiated and carried out by E. Gäumann and his school in Switzerland between the mid-1940s and the 1950s [21]. These studies stimulated further research in other countries by plant pathologists working in near-isolation, or by small groups of pathologists and organic chemists. Finally, in more recent years, research has extended all over the world, attracting an increasing number of scientists, especially as cooperative projects involving scientists with different fields of expertise.

The first plant/pathogen system to be investigated was the tomato wilt caused by *Fusarium oxysporum* f. sp. *lycopersici*. Several phytotoxic compounds, such as fusaric acid and aspergillomarasmins, were extracted and purified from culture filtrates and proved to induce wilt when applied to tomato cuttings in small concentrations [31]. The involvement of fusarial toxins in infected tomato plants, however, was not supported by rigorous experimental evidence [8].

In most vascular diseases such as tomato wilt, macromolecules produced by the pathogen, as well as viscous substances resulting from the enzymic breakdown of the cell walls of the host, increase the resistance to the flow of water through the xylem and cause occlusion of the vessels. The xylem dysfunction results in water stress, and the plant wilts because the leaves do not receive an adequate water supply for the maintenance of cell turgor [8, 71]. Toxins which could damage the integrity of plasma membranes or disrupt their function, thus causing an irreversible loss of cell turgor, are hardly translocated from the plugged vessels in which the pathogen is growing to the transpiring plant organs. Thus in diseases caused by potentially toxigenic pathogens, plants may wilt as a result of reduced water conductance through the xylem rather than in consequence of a toxin-induced inability of leaf cells to retain water [71].

Some recent findings have led to a reevaluation of the involvement of certain toxins in wilt diseases. The role of fusaric acid [22] in tomato wilt has been repeatedly questioned [8]. It was ascertained that there was no correlation between the ability to produce fusaric acid and the virulence of the pathogen [37]. However, it has recently been shown that treating tomato roots with soil bacteria able to degrade and detoxify fusaric acid, as well as with toxin-degrading mutants of *Pseudomonas solanacearum* and clones of *Escherichia coli* transformed with engineered plasmids from *Cladosporium werneckii*, protected plants from wilting caused by *F. oxysporum* f. sp. *lycopersici* [50, 70]. Moreover, plants regenerated from tomato leaf protoplasts insensitive to fusaric acid were found to show resistance to the pathogen [60].

Much of the scepticism about the role of toxins in plant diseases, chiefly based on the data on fusarial toxins, was overcome when the production of host-selective toxins by *Cochliobolus victoriae*, cause of the disastrous 'Victoria blight' of oats in the USA, was demonstrated in 1947 [42]. Another selective toxin, produced by a race of *Cochliobolus heterostrophus*, was shown to be an essential factor in the development of the 'Southern leaf blight of corn', a disease that devastated the widespread and susceptible 'Texas male sterile' maize lines in North America in 1970 and 1971, causing huge losses [34, 61].

In the first two decades after World War II, the rapid development of techniques and instrumentation for the separation and purification of natural substances and microbial products such as antibiotics provided new opportunities for the chemical elucidation of toxin structure. The first application was with non-host-selective toxins. Some bacterial toxins capable of inducing chlorosis, e.g., tabtoxin produced by the tobacco wilfire bacterium *Pseudomonas syringae* pv. *tabaci*, as well as other toxins of plant parasitic species of *Pseudomonas*, were thoroughly investigated [18, 19, 44].

The structure of fusicoccin was elucidated in 1968 [7]. This is a wilt toxin produced by a non-vascular pathogen, *Fusicoccum amygdali*, a fungus which causes bud cankers on shoots of almond and peach trees. Fusicoccin is perhaps the best understood of the toxins involved in plant diseases. After the producer pathogen has infected the host, the toxin is readily translocated by the transpiration stream from the invaded bark tissues to the leaves. Stomatal guard cells are induced to open irreversibly and the

uncontrolled water loss results in a rapid reduction of available water in the intercellular spaces and in a consequent loss of turgor of the mesophyll cells[69]. Large areas of the leaf blade wilt and wither. Pure fusicoccin solutions absorbed by practically any plant species, or sprayed onto the leaves, reproduce wilt by the same physiological mechanisms as that operating in naturally-infected stone fruit trees[26]. The finding that fusicoccin has the properties of a plant growth regulator attracted the attention of many physiologists and stimulated extensive research throughout the world[40,41,50].

The first comprehensive attempt to bring together current knowledge on phytotoxins was made in 1970 when a 'NATO Advanced Study Institute' on the subject was held in Italy. The proceedings were published as a book in 1972[72]. The meeting greatly stimulated research on toxins in many countries. During the ensuing 10 years several reviews were published[53,55] and a book entitled 'Toxins in Plant Disease' was printed in 1981[17]. A considerable number of meetings, discussions and reviews followed. The outcome of a Symposium held at the 4th International Congress of Plant Pathology (Melbourne, Australia) in 1983 gave rise to a book[12]. Two volumes of proceedings were published in 1989: the first, those of the 'NATO Research Workshop on Phytotoxins and Plant Pathogenesis' held in Italy in 1988, and the second, those of a conference on host-selective phytotoxins held in Japan (Tottori University) after the 5th International Congress of Plant Pathology (Kyoto)[27,33]. A selection of books, chapters and review articles on phytotoxins is given at the end of this Introduction. They offer the reader a wide spectrum of information and show how substantial the progress in the knowledge about phytotoxins has been in recent years.

The development of highly sensitive analytical and biological techniques and of new means for structural determination have reduced the time required to separate and purify the active substances, in many cases from years to months, and have permitted structural elucidation using milligrams of material, rather than grams. The first structure of a selective toxin was reported in 1974[49], although this kind of toxin had been known for 40 years[67]. Since then, about 20 such host-selective toxins have been recognized for diseases of considerable worldwide importance, and for more than half of them the structure has been elucidated[14,33,35]. Knowledge of the structures has led, in turn, to the application of advanced methods for the detection of phytotoxins in diseased plant tissues and to biochemical demonstrations of their sites of action, e.g. on the host cell membranes. How toxins act, i.e., their targets and mechanisms of action, is less well understood, but substantial information is now available for many bacterial and fungal toxins.

Finally, following the use of traditional approaches to the genetic analysis of toxin production, the powerful and precise technologies of recombinant DNA and other bioengineering methods, in particular transformation and complementation, have been applied to toxin systems, giving some insight into the genetic mechanisms of toxin production. Genes for toxin synthesis or detoxification have been successfully cloned and characterized[1,27,36,38,51,73,74].

Resistance of plants to disease is often expressed as tolerance of or insensitivity to the toxins produced by the pathogen. Some phytotoxins are known to be able to activate metabolic pathways leading to the synthesis of substances involved in plant defence reactions, such as phytoalexins. In this respect, they can act as 'elicitors', causing localized death of plant cells and hypersensitive responses of the host which may restrict growth of the pathogen[6]. In other cases, toxins act as 'suppressors' of induced resistance[29,33].

Apart from their obvious importance in the field of plant pathology, where several of them have been used to establish the molecular basis of disease production[2,5,11,36,39,59], phytotoxins are useful tools for gaining an understanding of the mode of action of many biologically active substances. Experimentally, phytotoxins are initially identified by potent pathological effects on susceptible host tissues. In such tissues they affect basic biochemical processes. Sometimes, phytotoxins have also been tested on non-host tissues for effects other than pathological ones.

Thus phytotoxins may have considerable potential as regulators of, or as probes for investigating, the biochemical and genetic processes which occur during normal development of crop species. Their use in plant physiology and – to a lesser extent – in microbiological investigations has greatly increased, and sometimes even become routine. Thanks to the use of certain phytotoxins, our understanding of certain biochemical and physiological processes in plants, such as the functions of membrane receptors and ion pumps, photosynthesis, genetic male sterility, transpiration and stomatal opening, water uptake and translocation of nutrients, has made substantial progress.

Although phytotoxins act primarily on plants, their effect may extend beyond the plant kingdom, for example to certain microorganisms or animals. A number of phytotoxins have been reported to be antibiotics or mycotoxins, that is they exhibit antimicrobial activity or, if ingested with contaminated food, may be hazardous for animals and humans[10,46,62]. As knowledge about the occurrence and range of sensitive organisms expands, utilization of phytotoxins as biocides may become possible. Because phytotoxins are biologically active compounds, a variety of applications have been successfully attempted. Currently, phytotoxins are being used as probes for the rapid screening of plant clones or the progeny from crosses for resistance to diseases. In agriculture, certain fungal toxins are considered to be promising agents for the biological control of noxious organisms, for example potential herbicides[16,30,46,64]. Others, like fusicoccins, may be used to promote the germination of dormant

seeds or to speed up the drying of leaves or hay. Approaches to using toxins with a wide biological activity in chemotherapy are also being explored [20].

This multi-author review is intended to give up-to-date information on some of the most important aspects of current research in the field.

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0014-4754/91/080751-05$1.50 + 0.20/0

Host recognition by toxigenic plant pathogens

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Abstract. Certain fungal pathogens release host-selective (or host-specific) toxins (HST) as a host recognition factor during spore germination at the infection site on plants. Prior to penetration of the pathogen into its host, the released toxin specifically binds to a putative receptor on the host cells and initiates signaling mechanisms leading to pleiotropic effects on cells. Of these, the crucial one negates the general and inducible defense reactions of the cells. This is accomplished by a signal from the HST, which is transduced through a path way at or near the step of plasma membrane modulation, which is directly or indirectly triggered by the HST. This mechanism operates even though the toxin may affect mitochondria or chloroplasts as the primary target organelle. The fungal spore is able to penetrate the so-called 'narcotized cell' and completes the initial colonization of the host. The host recognition process may take place without necessitating host cell death, even in the case of perthophytic parasites. At the molecular level, HST-mediated recognition of the host by a pathogen requires strict stereochemical precision like a lock and key.
Key words. Host recognition; host specificity factors; host-specific toxins; host-selective toxins; *Alternaria*; *Cochliobolus*; plasma membrane; mitochondrion; chloroplast; toxin receptor.

Introduction

In spite of repeated and massive exposure to diverse fungal parasites, higher plant species actually suffer from only a very minute fraction of the potential plant pathogens they come in contact with. In general, most plants have evolved 'non-host' defense mechanisms which can act against all but a few specialized parasites[16]. Such 'specificity in parasitism' or 'host recognition' has been of great interest to plant pathologists for a long time. It is not an easy task, however, to define 'recognition' precisely. Sequeira's definition[53], proposed in 1978, is most