Mycology Answers

Some Fungi produce Toxins that affect the Physiological Functioning of Higher Plants: what is the Role of these Compounds in the Development of Plant Diseases?

Fungi that are capable of invading and infecting higher plants may be pathogenic i.e. causing disease, or symbiotic such as mycorrhizal forms. Pathogens interact with potential hosts in many different ways. A wide range of biochemical factors (e.g. cell wall degrading enzymes, growth regulators, toxic compounds) is used to disrupt normally integrated physiological processes in the host plant and to bring about disease.

Pathogenic fungi are often grouped according to the means by which they derive nutrients from the host plant. Biotrophic fungi exploit living hosts, invading but causing minimum physical disruption and spreading slowly through the tissues. Nutrients are absorbed from the host, but the cell membranes are not disrupted and the host cells do not die (e.g. rust diseases, powdery mildews, downy mildews). Necrotrophic fungi on the other hand, are highly destructive to host tissues and plant cells are often killed prior to the invasion of tissues (e.g. fungal vascular wilt diseases). The spread of necrotrophs is often very rapid. In these cases contacts with living host tissues are minimal and in most instances host cells are killed in advance of invasion, usually by cell wall degrading enzymes that disrupt tissue integrity. Necrotrophic invaders only reproduce when the host cells are dead.

Plant pathogenic fungi produce a range of toxic compounds (phytotoxins), usually secondary metabolites, that affect the physiological functioning of higher plants. Phytotoxins often cause wilting, chlorosis and necrosis, although the importance and actual role in disease establishment is variable and, for some diseases, hotly disputed. In general, toxins are low molecular weight compounds and are active at very small concentrations. They are a very diverse group of molecules and include polypeptides, glycoproteins, phenolics, terpenoids, sterols and quinones. The term does not apply, however, to compounds having other effects such as hormones, enzymes, and genetic determinants, nor to compounds liberated by the disruption of plant tissues.

Toxins are usually classed as host-selective or non-selective. Host-selective toxins are usually very fast acting, produce disease symptoms in susceptible plants and are usually regarded as important for disease production. Host non-selective toxins, however, have toxicity that is not related to the host range of the fungus and can be differentially toxic to plants which are not susceptible.

Among the host-selective toxins are some of the most notorious fast acting compounds, which have affected crops world-wide and made headlines. Species of *Helminthosporium* produce toxins which exert multiple effects on the host plants with devastating consequences. *Helminthosporium maydis* produces HmT-toxin and causes southern corn leaf blight, a serious disease of plants carrying Texas male sterility (T-maize). This cultivar was used widely in southeastern USA to give good cropping plants. The fungus caused massive crop destruction in the early 1970s although the severity of the effect was compounded by the planting of genetically uniform cultivars over huge areas, which allowed rapid spread of the pathogen. The toxin (composed of ten linear polyketols) affects leaf function, including photosynthesis and respiration, and inhibits closure of stomata. In addition, membrane permeability is increased, resulting in leakage of potassium, and root growth is also impared. *Alternaria mali* produces AM-toxin and is very host specific. It causes blotch in susceptible apple cultivars by the action of three related toxins. Toxin I is a cyclic peptide and is more toxic than Toxins II or III. AM-toxins cause membrane disruption and affect chloroplast function and photosynthesis.

Host non-selective toxins are less fast acting but also result in serious disruptions to the normally integrated metabolism of affected plants.

Fusicoccum amygdali produces the toxin Fusicoccin. It is the causal agent of wilt diseases of almond and peach. Fusarium wilts impair water movement in the host plants following the obstruction of the vascular system (xylem pathway) by the presence of fungal mycelium, fungal spores and other materials produced by the pathogen and by biochemical responses of the invaded plant. The disease is caused by complex interactions between cell wall degrading enzymes, plant growth regulators and toxins. In this case, it is likely that toxins are secondary determinants of the disease. Water transport processes are impaired which results in increased respiration rates, together with the development of water stress symptoms. Some *Fusarium* species produce fusaric acid (5-n-butylpicolinic acid) which increases respiration rates in infected plants and contributes to the severity of infections.

In order to fully establish a role for toxins in the development of a plant disease, it is important that they are detected in naturally infected plant tissues. The same compounds should also be extracted from lesion tissue. However, this may be difficult since active concentrations are often very low. Toxins occurring in multiple forms may also confuse experiments. Toxins produced in culture may not necessarily be formed in a host plant, and studies using toxins from cultures have caused much controversy in the past. Additionally, some fungi liberate toxins early in their encounters with the host plant as an aid to penetration and establishment, while others are produced much later in the infection process and may enhance the senescence of the plant leading to its more rapid demise.

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