

A New Working Definition of the Term "Phytoalexin"

At a recent NATO Advanced Study Institute on "Active Defence Mechanisms in Plants," a group of plant pathologists assembled to revise the definition of the term "phytoalexin." A similar attempt at the 1978 International Congress of Plant Pathology in München, Germany, failed, and it was considered important to create a new working definition of phytoalexin reflecting some of the advances in our field since Müller and Börger first proposed the term. (Müller, K. O., and H. Börger. 1940. *Arb. Biol. Reichsanst. Land. Forstwirtschaft. Berlin* 23:189-231.)

In attendance at this meeting were Drs. P. Albersheim, J. Bailey, D. Bateman, J. Callow, D. Clarke, G. Defago, B. Deverall, D. Elgersma, M. Esquerre-Tugaye, J. Friend, B. Fritig, A. Fuchs, M. Heath, D. Ingram, N. Keen, J. Kuc, P. Langcake, G. Lazarovits, J. Mansfield, J. Paxton, W. Rathmell, P. de Wit, and R. Wood, representing a wide range of phytoalexin researchers. The working definition arrived at by consensus is: Phytoalexins are low molecular weight, antimicrobial compounds that are both synthesized by and accumulated in plants after exposure to microorganisms.

This definition may be contrasted with J. Hardy's direct translation of Müller and Börger's definition of a phytoalexin:

1. The premature death of the parasite on the tubers of the resistant W varieties is *not* due to any toxic "principle" already present in the tuber before the infection nor to the absence of any substance necessary for the normal development of the fungus, but to a *change in the state of the host cells which come into contact with the parasite*. This change of state results in a "paralysis" or the premature death of the fungus (cf. Meyer [29]; Müller, Meyer and Klinkowski [35]). The principle inhibiting the development of the fungus is formed or activated only in the course of this change of state which we have termed the "defensive reaction."

2. The defensive reaction is linked with the living state of the host cell. This does not mean, however, that a tissue which is parasitized by a virulent *Phytophthora* strain, but is still alive, may not at the same time be capable of responding to the attack of an avirulent strain with the changes of state characteristic of the defensive reaction.

3. The inhibiting principle must be of a material nature. It is formed or activated in the reacting host cell and may be regarded as the end product of a "necrobiosis" released by the parasite.

4. This not yet isolated and therefore still hypothetical "defensive substance" is "non-specific." It has an inhibiting action

on other parasitic fungi of the potato tuber as well as *Phytophthora infestans*. Saprophytic fungi are also inhibited in their development by this substance. However, the various parasitic species differ in their sensitivity towards this "phytoalexin."

5. The decisive factor for the fate of the parasite and hence also for the "immune" behaviour of the host is discovered only in the sensitivity of the host cells to certain material influences emanating from the *Phytophthora* fungus: the greater the sensitivity, the higher the resistance. The reaction product is accordingly not specific, but only the genotypically determined *readiness of the host cell to react*, which is manifested in the speed with which the hypothetical defensive substance is formed.

6. The defensive reaction is confined to the tissue colonized by the fungus and its immediate neighbourhood. There is no immunization embracing the whole individual.

7. In the resistant varieties we find an "immunization" of the portions of tissue invaded by the parasite; in the susceptible varieties the opposite is the case: the host cells invaded by a virulent *Phytophthora* strain, but still alive, also become "sensitive" to fungi which are incapable of attacking an intact potato tuber after association with the parasite for some time. Here, too, we note gradual differences in the capacity of the individual fungus species to colonize the tissue attacked by *Phytophthora*.

8. What is inherited is only the capacity to "acquire" the resistance at the place of infection and only here, but not the resistant "state" in itself. This state must first be "acquired," and this happens only after the plant has come into contact with the pathogenic agent. This serves to release the "mechanism" which transforms the portions of tissue attacked by the parasite from the "indifferent" to the "resistant" state.

Müller in 1956 revised his definition of phytoalexin to "Phytoalexins are defined as antibiotics which are the result of an interaction of two different metabolic systems, the host and the parasite, and which inhibit the growth of microorganisms pathogenic to plants." (Müller, K. O. 1956. *Phytopathol. Z.* 27:237-254.)

It is hoped that the revised definition presented here will help clarify what constitutes a phytoalexin and that continued research will clarify the role of phytoalexins in plant disease resistance.

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Voucher Specimens Should Support Pathogen Reports

We would like to raise a matter that has concerned us for some time. Whenever a new report of a pathogen is made, sufficient description of the material should be included in the journal paper so its readers could feel confident of the validity of the identification of the pathogen. With fungi, this could include sorus descriptions, spore measurements, and notes on spore ornamentation.

Most important, a voucher specimen of all such pathogens should be deposited in a recognized herbarium, and this should be reported in the article. Without this documentation, such reports can be considered as only tentative. Researchers must be willing to preserve representative samples so that their reports may be reconfirmed if necessary. In the course of our work, we have found many specimens that were misidentified. Such errors would have stood forever had not voucher specimens been kept.

We have recently been trying to determine the distribution of sugarcane rust in the Western Hemisphere. Some of the journal reports have published descriptions and photographs, so we can accept their accuracy. Other articles, however, offer no supporting evidence and no indication that specimens were kept. Since we cannot examine their collections, we feel that those reports simply cannot be accepted as valid distribution records.

Consequently, we recommend that in PLANT DISEASE, reports of new pathogen distribution records or new host reports be supported by the preservation of representative voucher specimens and that the location of the (se) specimen(s) be noted. Also, the pathogen should be briefly described.

The Arthur Herbarium is willing to keep and safeguard voucher specimens of any collections of rust fungi (Uredinales). The Arthur Herbarium, with over 90,000 specimens, is the world's largest herbarium devoted solely to the study of rust fungi. We would also be happy to assist in the identification of rust fungi.

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