HISTOGENIC DEMARCATION IN DISEASED PLANT

by

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Introduction

Defensive reactions are known as vital processes initiated in a host plant by a pathogenic agent and directed more or less specifically against that agent itself. Gäumann (5) has classified defensive reactions in plants into three groups based on the object of reaction and into six kinds as to the type of reaction (Table 1).

Table 1. Types of defensive reactions in plants (5)

Object of the reaction	Historical development	Type of reaction
The pathogen itself (anti-infectional reactions)	Autonomous	1. Plasmatic anti-infectional reactions
		2. Necrotic reactions
	Induced	3. Premunity
The metabolic products of the pathogen (anti-toxic reaction)	Autonomous	4. Histogenic demarcations
		5. Gummous demarcations
The innate Sensitiveness of the host (induced tolerance)	Induced	6. Desensibilization

The histogenic demarcation is a defensive reaction in which the host protects itself against toxic metabolic products emanating from the pathogen and from its own necrotic cells (5).

Many necrogenic fungi parasitic upon the leaves of plants make only limited growth, which results in definite lesions. The reason for such limitations of growth are purely physiological in character and have to do with the biochemical relation of pathogen and suscept (3).

Histogenic demarcations occur in the shot-hole disease of plums, peaches, and cherries (4), and in varieties of tobacco resistant to black root rot (2). In some woody plants, the demarcation tissues are not shed after they dam up the toxic substance (6).

The Cicatrice Formation and Histologic Changes in Plants.

There are numerous references in literaure to the healing of wounds by the histological changes resulting in callus formation in woody stem and cicatrice formation in leaf. Upon the death and collapse of exposed cells along the edges of wounds, a temperary protection is developed and is called a pseudocicatrice. Beneath this covering, the living parts slowly give rise to new layers of cells, which in time mature into the true cicatrice. This structure

varies greatly, but in thicker leaves may be a compact cork-like tissue with heavily suberized and highly modified cell walls(11). This cork-like tissue is largely formed by the development of new cells resulting from mitoses (10). Mitoses rarely occur within 24 hours after wounding. In thin mesophytic leaves, the cicatrice is complete in about 10 days, whereas in thick and xerophytic forms, up to 20 days might be required for its completion (11).

Blackman and Matthaei (1) removed leaves of *Prunus laurocerasus var. rotundifolia* and placed the petioles in a beaker of water. No healing tissue was formed when these leaves were wounded. If a sufficient number of cells were killed about the edge of the wound, however, an abscission layer was formed. The wounded portion eventually dropped out and the walls of the callus cells became cuticularized. Leaves wounded while stile attached to the plants formed an abscission layer very quickly. The wounded portion did not fall out, but division of the newly formed cells continued until a periderm of several layers of cork resulted (1).

Cunningham (3), in studies of some species of *Prunus*, *Pyrus communis*, and *Beta vulgaris*, also found that the natural reaction of the suscept to injury is the formation of a cicatrice. That the formation of a cicatrice is a common occurrence in the leaves of many higher plants is evident from the work of Wylie (10,11), Blackman and Matthaei (1), Cunningham (3), and others.

The Histogenic Demarcation Reaction in Diseased Plants

There are comparatively few reports in the literature about the reaction of plants to invasion by necrogenic pathogens. In 1892, Pierce (8) stated the shot-hole lesion in lesves of almond caused by *Cercospora circuncissa* is bounded by plesionecrotic and thickened tissues (8).Working with *Coccomyces sp.* in *Prunus*, Higgins (7) found that there was considerable variation in the same species. He states that the separation of tissues is due to the abrupt enlargement of a layer of cells at some distance from the ends of the mycelium. The enlarged cells lose their chloroplasts and nuclei, and only a thin layer of protoplasm lines the walls (7). Cunningham (3) stated that the cicatrice formed at the edge of necrotic pathological lesions is similar in its general characters to that formed as a natural reaction of the suscept to mechanical injury.

There is also variation with the position of the initial infection; different types of histogenic demarcation tissue formed in different parts of plants after infection by different pathogenic organisms.

Demarcations in leaves:

Samuel (1927) states if the leaves of *Prunus spp.* are infected by the conidia of *Clasterosporium carpophilum*, the causal agent of shot-hole disease ,the germ tube penetrate either through the epidermis or through the stomates. Once inside, the fungus proliferates in all directions in and between the cells. Within 38 hours after infection, the cells entered by the pathogen are seriously injured and about to collapse. Their walls are thickened and brown. As a consequence of the distant action by toxins, the nearest hypodermal cell, although not directly attacked, shows a brown discoloration (hyperergic effect). Moreover,

in neighboring cells, the chloroplasts have disappeared and, still farther away, they have shrunk and become light green.In the spongy mesophyll, no changes are yet apparent except for a dark coloring of two cells (again the result of action at a distance). Fifty-six hours after infection the lesion is already 1-2 mm wide and, therefore, visible macroscopically. The mycelium has invaded the spongy tissues and the neighboring vascular bundles, in which it has obliterated the phloem parenchyma. A few epidermal cells are brown and collapsed. The hypodermis is disorganized and collapsed. The infection spot then appears on the leaf surface as a small pit. Numerous cells of the spongy mesophyll are in the process of degeneration, and the toxins of the parasite are causing disintegration of the chloroplasts right up to the palisade tissues. After 4 days, the hyphae have reached the palisade tissues. Macroscopically, the necrotic cells in the center of the lesion are seen as a brown patch, surrounded by a lighter green zone (the cells containing the disorganized chloroplasts.).

Histogenic defense reactions set in between the fourth and eighth days. At a distance of about 20 cells from the brown necrotic zone, a narrow band of cells which involves all the leaf tissues begins to swell slightly; the cells are squeezed closely so that the intercellular spaces disappear, as do the chloroplasts; the protoplasm becomes denser, the nuclei increase slightly in size, and the cells assume a maristematic character. As a consequence of the disease, tissues which had already matured have again become capable of undergoing division. Vigorous cell division also sets in throughout the vascular bundles, whose parenchyma cells have been rejuvenated, and the xylem elements are squeezed to the periphery of the bundle. On the one hand, this abscission tissue interrupts the supply of nutrients from the healthy tissues to the lesion so that this gradually shrivels.

In young leaves, begining at the lower epidermis, the middle lamellae in a certain zone began to disintegrate, usually in such a way that one cell row accrues to the focus, while the three and four others remain part of the healthy leaf. In this way the focus of infection becomes spatially cut off from the healthy tissues and subsequently is actively thrust away from the lower side of the leaf.

Scarring now begins along the edge of the wound by the initiation of a wound periderm. A middle cell row, the phellogen, forms on its outer side two or three slowly lignifying and then suberizing of phellem cells and, on the inside, three or four rows of only cellulose thickened phelloderm. With this, the histogenic demarcation is completed (6,9).

Cunningham (1928) studied on the histological changes induced in leaves by certain leaf-spoting fungi and showed that species of *Prunus*, *Pyrus*, and *Beta* may be expected to react, when attacked by nectogenic pathogenes, by the formation of a cicatrice which isolates the infected portion from the healthy tissues. The character of this cicatrice and the extent of healing tissue differs with the relation of the infected portion to veins and islet borders (3).

The character of the cicatrice is essentially the same in all plants, The most striking features of this structure are the renewal of the meristomatic condition in the mature mesophyll cells. This results in a multiplication of cells in this region, the occlusion of intercellular spaces, and the formation of a definite wound periderm with its phellogen layer. Cork cells produced by the phellogen are frequently greatly enlarged (3).

Demarcations in roots and branches:

Demarcation reactions in branches and roots is sometimes accompanied by an expulsive or scaling reaction (6). Conant (1927) studied the root rot of tobacco caused by *Thielavia basicola*, and stated that the rapidity with which a tobacco plant can initiate and continue cork formation beneath a lesion is an accurate criterion of its resistance to *Thielavia*. Different degrees of histogenic demarcation reactions occur among different varieties of tobacco. In strongly resistant strains of tobacco, or in strains made resistant by exposure to high temperatures, the host tissue several layers of cells deeper than those actually invaded are stimulated to active cell division, forming a phellogen which further limits the progress of the fungus by proliferating cork cells toward the point of invasion. In other varieties of tobacco, however, the reaction occurs first in that part of the pericycle which lies immediately beneath the lesion. In the semi-resistant varieties, the histogenic demarcation fails to keep the fungus out of the stele in all cases, especially where the attack is made by a large waft of hyphae (2,6).

The Characteristics of Histogenic Demarcation in Diseased Plant.

The evidence presented above shows that there are histogenic demarcations formed in plants after penetration by pathogens. There are also two important characteristics of such kinds of defensive reaction in diseased plant: it is either an anti-toxic reaction or an autonomous reaction.

Anti-toxic reactions:

Duggar (4) first drew attention to the fact that the shot-hole effect on leaves of species of *Prunus* was produced not only by a number of fungi, but also by spraying with injurious solutions such as copper sulphate, formalin, corrosive sublimate, and others. McWhorter (3) has shown that this layer (cicatrice) may be penetrated to a slight extent by *Cladosporium papaya*. In the case of shot-hole disease in stone fruit tree (*Prunus spp.*) caused by *Clasterosporium carmophilum*. Gäumann states that the shot hole effect is not directed against the invading parasite but against its toxins and the host's own necrogenous products (6). The parasite causes the necrosis, giving rise to necrogenous substances. These, in the usual way, stimulate the formation of wound periderm.

Gloeosporium fructigenum (Bitter rot), possess fully developed antigenic properties but they develop more rapidly than the demarcation reac tion can follow, so that this lags behind; the race between pathogen and host is here won by the pathogen (7). The result is that the parasite successively colonizes the whole leaf.

So far as histogenic demarcation posses chiefly anti-toxic, not anti-infectional properties; therefore the parasite is sometimes able to penetrate them(5).

Autonomous reactions:

It is natural for plants to form a healing tissue around the wound and thereby recover from injuries. In some plants, wounds are healed simply by drying of the wound surface, whereas some form a special tissue such as cicatrice along the wounded edge (11). These are autonomous reactions of the plant itself.

Conclusion

The chain of reactions in histogenic demarcations is not initiated by the parasite itself, but by the necrosis caused by it. To this extent, demarcation reaction is non-specific, being indifferent to the particular source of the necrosis of the lesions, toxins of the parasite, or any other organic or inorganic poisons (5,6).

The defensive reactions classified by Gäumann include three groups and six different kinds. Here, histogenic demarcations are one kind of protection which is effected by means of the antitoxic reactions; their aim is not to prevent infecton, but to prevent disease.

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罹病植物之組織隔離反應

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植物對疾病之防禦反應一向被認爲是因爲病原體在植物體內之進展而引起之對該病原菌本身之一種或多或少具有特殊性之抗禦作用。Gäumann 曾將植物之防禦反應，據防禦對象分成3種，另按防禦反應之類型分成6種。本文論述之組織隔離反應（Histogenic demarcation reaction）即爲該氏依據反應類型而分類之一種。

組織隔離反應是寄主植物因抵制從病原菌或植物腐爛細胞所產生之異化物質而產生的保護植物自身的一種防禦反應，所以與其他類型之植物防禦反應不能混爲一談。

梨、桃等許多植物之穿孔病爲植物組織隔離反應的典型例子。這種防禦反應不僅可發生在葉片，亦可發生在根、莖等部位。然而因發生部位之不同而反應過程及結果亦稍有差異。以 *Clasterosporium carpophilum* 引起之櫻桃穿孔病爲例，當葉片被病原菌侵染後4至8日間，組織防禦反應開始發生。距離被侵染之腐敗斑點約20個細胞處的健全細胞，圍成一狹帶，並稍膨脹，這些細胞互相擠集，細胞間隙及葉緣體消失，原生質濃縮，細胞核增大，而具活性。當病情擴展時，在病斑四週的已成熟之細胞再次恢復分裂，此極活潑之細胞分裂，透過維管束，而將導管擠在週緣，另方面這些隔阻組織，中止營養分從健全組織進入病斑內，終使腐敗之罹病斑點脫落。

罹病植物之組織隔離反應具有兩大特性，是抗毒質反應，亦是植物體之自動反應。因爲這種組織隔離反應是緣賴病原菌引起腐爛而導致，並非病原菌自身所引起，所以這種組織隔離反應並無特殊性，不論何種腐爛病斑內之物質，寄生菌產生之毒質，或是有機或無機物所發生之結果均無差異性。

基於上述原因，植物組織隔離反應乃是植物抗禦有毒物質而產生之保護自身健康之反應，其目的不在防止病原體（菌）之侵染，而在於防止疾病之擴展。