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Defense mechanisms of vegetables towards diseases

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Abstract
Plant bears its own defense mechanism (s) against the development of diseases caused by various fungi, bacteria, viruses, nematode and other microorganisms. The attribution of the hosts that reduce the chances of infection or the further development of the pathogen are considered to be defense mechanism'. The defense may be used against pathogen at any stage of infection *i.e*. during pre-penetration, penetration or post penetration. In nature disease resistance seems to be the rule rather than the exception. Plants also have induced structural and biochemical defense like histological defense structure, hypersensitive response, PR protein, phytoalexine and systemic acquired resistance. All these defense mechanism are slow down or inhibit the initial attack of pathogens.

Keywords: pre-existing structural defence, Pre-existing chemical defence and induced biochemical defense

Introduction
Vegetables are considered as protective food in our diet as they are rich source of minerals and vitamins. Vegetables faced lots of diseases problems which lead to lower its production and hinder our nutritional security. To copup with this problems, plants have evolved several defense mechanism itself.

Understanding how plants defend themselves from pathogens is essential in order to protect our food supply and develop highly disease-resistant plant species. Disease in plants can be defined as the series of adverse changes in plant cells and tissues in response to a pathogen or environmental factor which results in alterations in the form, function or integrity of the plant and may lead to partial impairment or death of plant parts or the entire plant.

Resistance against any 'deleterious act' has become a natural and universal response of plant system. The resistance against parasites/pathogen is the heritable trait of plants by virtue of which they resist attack by parasites/pathogens or their activities. The defense mechanism(s) has ensured the survival of plants in spite of living amongst some of the potentiality devastating pathogens in addition to abiotic stresses.

Most of the plant diseases are caused by pathogenic microorganisms such as fungi, bacteria and viruses. Plants have developed an array of structural, chemical, defenses to prevent themselves from extensive damage caused by pathogens. Constitutive defenses are those which are already present in the plant and include many preformed structural barriers such as cell walls, waxy epidermal cuticles, thorns and bark and antimicrobial chemical compounds. They act as first line of defense and inhibit or slow down the initial attack of pathogens. Inducible defenses are produced or mobilized when the plant gets injured or detects invading pathogens. These include toxic chemicals, pathogen-degrading enzymes, secondary metabolite products and deliberate cell suicide (Agrios, 2005) [1].

Review of Research Work
Pre-existing structural defence
Zhang *et al.* (2015) [10] investigated the role of leaf structure in resistance to powdery mildew in watermelon. They observed stomata number in the highly resistant variety PMR 5 was 40.4 per visual field, while the stomata number in the same area on the leaves of the most susceptible variety Huang HeMi 3 was almost half at 21.6. Moreover, the size of stomata was smaller (mean length 6.1 μm2) in the highly resistant variety PMR 5 than in the most susceptible variety Huang HeMi 3 (9.0 μm2). They also noted the wax content of the resistant varieties was higher than in the susceptible ones after fungal inoculation and reached its maximum value (21.2 mg/ g) on the ninth day in the highly resistant variety PMR 5. Stages in the development of the necrotic defense reaction in a cell of a very resistant potato variety infected by *Phytophthora infestans* are given in Figure 1.

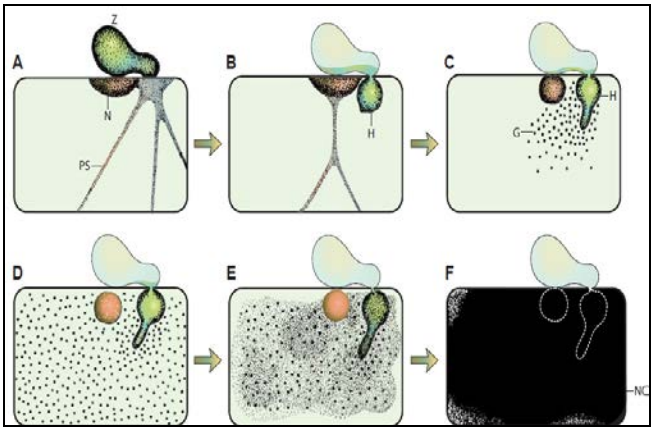

Fig 1: Stages in the development of the necrotic defense reaction in a cell (N: Nucleus; PS: Protoplasmic strands; Z: Zoospore; H: Hypha; G: Granular material; NC: Necrotic cell)

Pre-existing chemical defence

Mithen *et al.* (1987) [7] found that the glucosinolate content of leaves of 'resistant plants, *i.e.* those exhibiting small localized lesions was significantly greater than that in leaves of susceptible plants of *Brassica* species.

Younes and Abo-Elyousr (2014) [9] studied the phenol content in screening of some okra (*Abelmoschus esculentus* L.*)* genotypes to powdery mildew resistance. They observed the highest phenol content in Luxor genotype.

Induced biochemical defense

Chen and Heath (1992) [4] studied the effect of elicitor produce by *Uromyces vignae* causing cowpea rust and they noted that resistant cultivar showed higher level of necrotic cells than susceptible cultivar due to hypersensitive response.

Hong *et al.* (2013) [5] reported that the higher doses of 0.02 and 0.05 mM H_2O_2 decreased the bacterial growth compared to that of untreated control. No bacterial growth was found in the bacterial cultures treated with 0.1 and 0.2 mM of H_2O_2 in tomato. Bernards and Ellis (1991) [2] recorded that tomato cell cultures inoculated with *Verticillium alboatrum* accumulated up to fivefold higher levels of wall-bound phenolics than in uninoculated control.

Sudhamoy Mandal (2010) [8] observed that all elicitors could increase lignin deposition in eggplant roots in varying degrees, starting from 12 h of elicitation, when compared to the corresponding control. Lawrence *et al.* (2000) [6] reported that resistant genotypes exhibiting higher total enzyme activity levels of chitinase as well as *b*-1,3-glucanase than susceptible ones in tomato. Brindle *et al.* (1983) [3] studied the accumulation of phytoalexin in potato cell suspention cultures inoculated with either an incompatible (Race 4) or a compatible (Complex) race of *Phytopthora infestans.* They found that phytoalexine concentration was more in race 4 *i.e*. resistant as compared to compatible (Table 1).

Table 1: Examples of phytoalexin produced by plants

Structure	Name	Plants involved
Inorganic	sulphur	cocoa
Phenolic	chlorogenic acid	potato, tobacco, apple
	avenalumins	some cereals
Terpenoid	capsidiol	capsicum, tobacco
	rishitin	potato, tobacco, tomato
	ipomeamarone	sweet potato
	gossypol	cotton
Phenylpropanoid	pisatin	pea
	phaseollin	french bean, cowpea
	kievitone	french bean, cowpea
	glyceollins	soybean
	medicarpin	alfalfa, clover, broad bean, chickpea
	scoparone	citrus
Acetylenic	wyerone	broad bean
	safynol	safflower
Stilbene	resveratrol	grape, peanut
	batatasins	yam
Indole-sulphur	camalexin	*Arabidopsis*
	brassinins	cabbage, rape, turnip

Conclusions

Defense mechanism(s) is/are inbuilt in vegetable crops in nature. Some resistance mechanism may induced by application of external chemicals and/ or microorganisms. Most of the microorganism penetrate inside the host cell wall through wound or injury or through natural openings like stomata, lenticel and hydathodes. Plants have different types of defense mechanism *viz.* Pre-exixsting structural and chemical defense *i.e.* wax, cuticle, cell wall and in chemicals such as phytoanticipants and several phenolic compounds which are present in the host cell. Plants also have induced structural and biochemical defense like histological defense structure, hypersensitive response, PR protein, phytoalexine and systemic acquired resistance. All these defense mechanism are slow down or inhibit the initial attack of pathogens.

References

1. Agrios GN. "*Plant pathology"*, Elsevier Academic Press, London, U.K, 2005, 208-242.
2. Bernards MA, Ellis BE. Plant Physiol. 1991; 97:1494-1497.
3. Brindle PA, Kuhn PJ, Threl fall DR. Phytochemistry. 1983; 22:2719-2724
4. Chen CY, Heath MC. Physiol. Mol. Plant Pathol. 1992; 40:23-28.
5. Hong J, Kang S, Kim Y, Yoon D, Kim H, Sung C, *et al*. Plant Pathol. J. 2013; 29(4):386-396.
6. Lawrence CB, Singh NP, Qiu J, Gardner RG, Tuzun S. Physiol. Mol. Plant Pathol. 2000; 57:211-215.
7. Mithen RF, Lewis BG, Heaney RK, Fenwick GR. Trans. Brit. Mycol. Soc. 1987; 88:525-531.
8. Sudhamoy Mandal. African. J Biotechnol. 2010; 9(47):8038-8047.
9. Younes N, Abo Elyousr K. Int. J Agric. and Econ. Dev. 2014; 2(1):59-76.
10. Zhang H, Yu Ge, Wang Q, Zhang J. Indian J Genet. 2015; 75(2):237-241.