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Chapter – 24

Induced resistance in plants and counter-adaptation by insect pests

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Abstract

Insect and plants have coevolved for millions of years. Plants respond to herbivory through various morphological, biochemical, and molecular mechanisms to counter/offset the effects of herbivore attack. These defense strategies against herbivores are wide-ranging, highly dynamic, and could be direct and/or indirect. Direct defense affects the herbivore's growth and development due to antibiosis because of secondary metabolites produced constitutively and/or induced upon infestation by the insect pests. The indirect defense involves the recruitment of natural enemies of the insect pests. The natural enemies (parasitoids and predators) are attracted by the volatiles produced by the plants in response to insect herbivory. The direct and indirect defensive strategies either act separately or in conjunction with each other. However, insects have the ability to adapt to the plant defensive responses through physiological processes, metabolism and behavior to offset the adverse effects of the host plants' defense systems. This process of defensive responses by the host plants and counter defense by the insect pests results in the breakdown of resistance, and evolution of new populations/biotypes of the insect pests. This co-evolution between the plants and insects poses a major threat for developing crop cultivars with stable resistance to the target pest for pest management.

***Key words*:** Host plant resistance, induced resistance, insect adaptation, secondary metabolites, pest management.

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1. Introduction

Plant damage by insect pests and the response of the host plants to insect herbivory is a central component in evolution and speciation of both insects and

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Over view

plants (1). In the course of evolution, a number of insect-plant interactions have evolved that are sometimes symbiotic and/or competitive. Quite often, most of the interactions are harmful to one or both the species. Plants respond to insect herbivory through a dynamic and multifaceted defense system, which is mediated physiologically and biochemically. Host plant defense against herbivores is a complex array of structural, chemical, and physiological traits intended to perceive the attacking organisms (Fig.1), and restrain them before they are able to cause extensive damage (1-5). Plant defensive responses against herbivory can be either constitutive or induced. Constitutive defense occurs in plants irrespective of the external stimuli such as insect damage and/or elicitor application. Induced defense is activated in plants in response to the external stimuli such as insect damage, pathogen infestation, abiotic stress and/or elicitor application. Induced defense is an important component of plant defensive strategy, and has gained high momentum in insect control programs (2, 3, 5). Induced defenses make the plants phenotypically pliable, and thus, less prone to adaptation by insects (3, 6-8). However, the timing of induced resistance is very important, faster the response, the greater the benefit for the plant. It not only prevents the insects from infesting other parts of the same plants, but also from the other insects waiting to attack it. A better understanding of insect-plant interactions and the counter adaptation by the insects will provide avenues for designing of new strategies for controlling insect pests.

Since plants lack the physical mobility, they have evolved a number of strategies which enable them to withstand insect pressure. Plant defense against insect pests is mediated through morphological (toughness, thorns, thickness, and hairiness) and biochemical (nutritional composition of the plant tissue, and the nature and amounts of secondary metabolites) factors (4, 9). Plant defense against insect pests is highly sophisticated and precise, however, the insects have developed mechanisms to counter this defense (1,5,10). Insect adaptation to plant allelochemicals is the basic determinant of ecological and evolutionary patterns of host plant selection by the insect pests (10, 11, 12).

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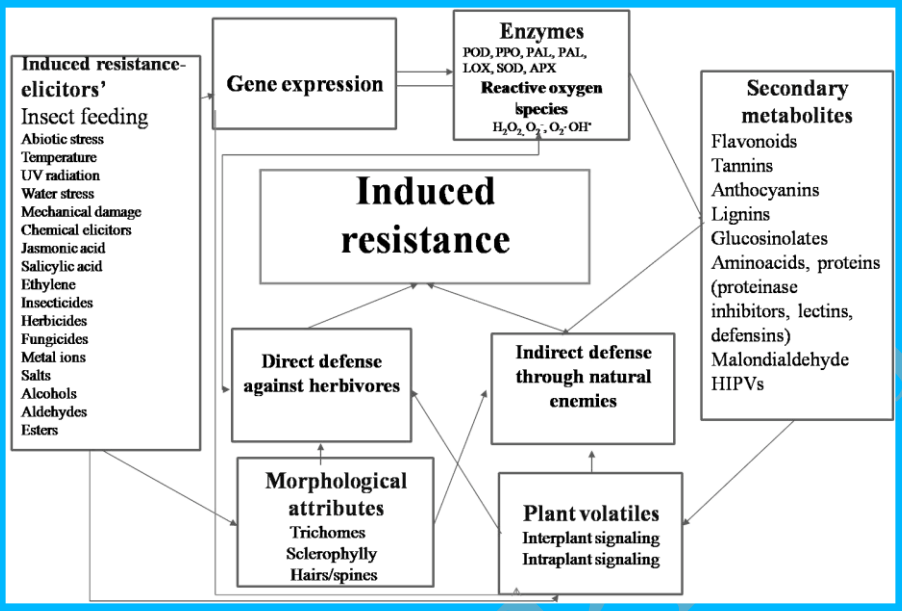

Fig.1. Mechanism of induced resistance in plants and adopted from War et al. (2012). POD= peroxidase; PPO= polyphenol oxidase; PAL= phenylalanine ammonia lyase; TAL= tyrosine alanine ammonia lyase; LOX= lipoxygenase; SOD= superoxide dismutase; APX= ascorbate peroxidase; HIPVs= Herbivore induced plant volatiles

On the basis of feeding habits, insects could be generalist herbivores, where the insect feeds on a range of host species or specialist herbivores, where the insect feeds on few host species or, in extreme cases, feeds on a single species. The successful herbivory by insect pests depends on their potential to combat the plant's defense strategies. Insects avoid plant defense chemicals through behavioral, morphological, biochemical and physiological adaptations. However, biochemical adaptations of insects to plant allelochemicals are more abrupt, dynamic and effective (1). Avoidance of plant-defensive compounds by insects has been suggested to be genetically determined and/or achieved after frequent encounter with the toxic plant foods.

Genetic engineering has been used to produce high yielding varieties that are resistant to various biotic and abiotic stresses (13) by introducing various genes, which enable them to produce higher levels of toxic secondary metabolites. However, one of the major concerns of transgenic crops is the development of resistance by insect pests. A number of lepidopteran pests have been reported to have developed resistance to *Bacillus thuringiensis* (Bt) under field conditions. For example, *Busseola fusca* Fuller has been reported resistant to Bt corn producing Cry1Ab in South Africa (14). Pink bollworm, *Pectinophora gossypiella* Saunders has developed resistance to

Bt cotton producing Cry1Ac in western India (15). Similarly, cotton bollworms, *Helicoverpa zea* (Boddie) and *Helicoverpa punctigera* (Wall.) have developed resistance to Bt cotton producing Cry1Ac and Cry2Ab in the southeastern United States and Australia, respectively (16,17). *Spodoptera frugiperda* (J.E. Smith), in Puerto Rico has been reported resistant to Bt corn producing Cry1F (18). Development of resistance by insects will lead to the severe outbreaks of the insect pests and high yield losses. In this paper, we will focus the discussion on counter adaptations by the insects to secondary metabolites (glucosinolates, tannins), protease inhibitors (PIs), and the role of various enzymes in insects for detoxification of plant allelochemicals.

2. Signal transduction in plant defenses

Plants respond to elicitors derived from oral secretion of insect herbivores, mechanical damage and/or the exogenous application of inducers (1, 19). Insect oral secretion/regurgitant contains a number of elicitors of plant defense; the important ones are fatty acid conjugates (FACs). The first FAC isolated from the oral secretion of beet armyworm larvae, *Spodoptera exigua* (Hub.) was N-(17-hydroxylinolenoyl)–L -glutamine (volicitin) and stimulates maize plants to produce volatiles, which attract natural enemies of the pest (20). Similarly, regurgitant of tobacco hornworm, *Manduca sexta* L. contains N-linolenoyl-glu is a potential elicitor of volatile emissions in tobacco plants (21). In addition, some FACs activate mitogen-activated protein kinase (MAPK) pathway, producing a number of plant defensive compounds, that play an important role in activating signal transduction pathways in response to heat and drought stress, and pathogen and insect attack (22). Some FACs induces accumulation of 7-*epi*-jasmonic acid, a potent elicitor of herbivore-responsive genes in tobacco plants (21). FACs induce the production of nicotine and proteinase inhibitors (PI) in *Nicotiana attenuata* (Torr. ex Watson) (23).

Herbivory leads to the accumulation of phytohormones in plants, the important ones being salicylic acid (SA), jasmonic acid (JA) and ethylene. The phytohormones mediate various signal transduction pathways involved in plant defense against various biotic and abiotic stresses. The main transduction pathways involved in plant defense against herbivorous insects are phenylpropanoid and octadecanoid pathways mediated by SA and JA, respectively (1). These pathways lead to synthesis and accumulation of toxins at the feeding site or in other parts, which are then transported to the feeding site (24, 25). In addition, antioxidative enzymes involved in plant defense accumulate in plant tissues on account of insect damage (26, 27). The insect adaptation to plant defensive chemicals involves overcoming penetration barriers and special excretions, sequestration of secondary metabolites in the mid gut, temporary binding with carrier proteins, and storage of toxins in adipose tissue, target-site mutation and behavioral avoidance (11, 28).

3. Avoidance of plant defenses by insect pests

Defensive strategies by the host plants pose a considerable selection pressure on insect pests, which have led to the development of counter adaptation of insect pests to these defenses (10**)** (Fig. 2). The mirid bug *Pameridea roridulae* (Reut.) walks freely on the sticky surface of *Roridula gorgonais* Planch., a proto-carnivorous plant (29), which is considered as an important plant defensive trait in these plants. Similarly, *Helicoverpa armigera* (Hub.) was found to feed on *Arabidopsis thaliana* (L.) leaf tissue areas with low concentration of glucosinolates (30). Glucose oxidase in the saliva of *H. zea* suppresses the induced plant resistance and reduces the amounts of toxic nicotine in *Nicotiana tabacum* L. (31). Similarly, glucose oxidase in saliva of *S. exigua* has been found to regulate the expression of defensive genes in *Medicago truncatula* Gaertn. (32). Insects even eavesdrop the presence of JA and SA and up-regulate their detoxifying systems to face the future plant defense (33). Some insects remove the hairs from leaves, which restrict the insect feeding (34), while others cut the leaf veins or latex channels (35) or even impinge the plants to isolate the feeding site so that the defense compounds produced distally are not transported to the site (36, 37). Contact avoidance has been reported in monarch butterfly, *Danaus plexippus* (L.) larvae, which exclusively feed on milkweed (38). Milkweed has a specific feature of presence of a variety of toxins stored in pressurized latex canals. However, larvae cut the veins and drain these toxins before feeding (38).

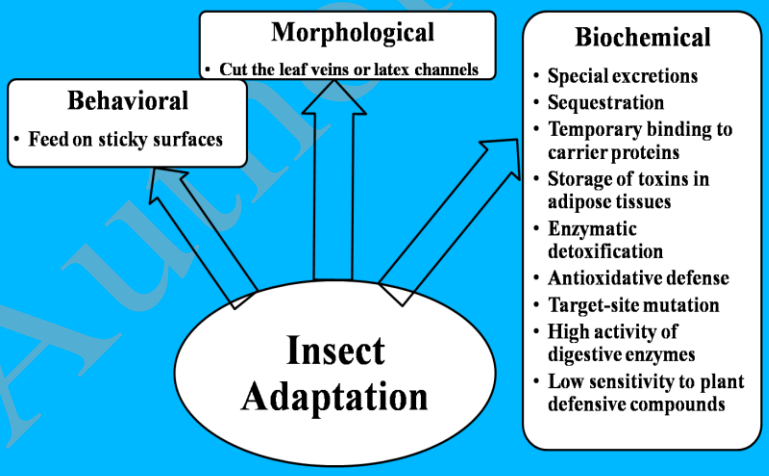

Fig.2. Counter-adaptations of insects to plant defensive systems.

Gall-forming insects avoid the plant defense by takingover the plant tissue and make it a reservoir for the photosynthesis products so that they can draw substances from it (39). Furthermore, these insects reduce the levels of toxic phenols in the gall and make it a site convenient for the larval development (40).

The sap feeding insects such as aphids draw the nutrition from phloem of the plants by piercing the stylet into the vascular bundle. These insects deplete photosynthates, act as vectors of many viruses, and inject chemicals, which alter plant defense signaling, and effect plant growth and development (41). Although there is not much physical damage caused by sap feeding insects as is caused by the chewing insects, and the plant defensive response is not as strong as against the large tissue damaging pests, plants do respond to them by sealing the puncture used by stylet piercing (42). However, insects have developed a strategy to avoid this sealing by producing the proteins in saliva, which antagonizes the plant depositions on the punctures (43).

3.1. Adaptation to protease inhibitors

Protease inhibitors (PIs) constitute an abundant and important group of compounds in plants, which have a defensive function against herbivores, including insect pests. The PIs inhibit the activities of various enzymes in insects especially, insect peptidases including serine, cysteine and aspartate proteinases and metallo-carboxypeptidases, which are involved in insect growth and development (44, 45). The PIs reduce the digestive ability of the insect pests, thus leading to the shortage of important food constituents such as amino acids resulting in slow development and/or starvation (46). A large number of PIs have been reported in plants, which are effective against many insect pests including lepidopteran and hemipteran insects (47, 48). Production of PIs is induced in plants in response to insect damage (47). However, many insect pests have adapted to plant PIs, which has resulted in even greater damage to the plants (27, 49). This counter defense of PIs by insect pests is a major barrier to the manipulation and utilization of PIs for a long-lasting plant defense, and thus warrants an understanding of the mechanisms by which insects counteract the PI-based plant defense. Adaptation by insect pests to PIs has attracted the interest of researchers to understand the adaptation mechanisms, and ultimately design better strategies so that PIs can be utilized in crop protection (49-51). Insect pests have developed two types of resistance or adaptation to protease inhibitors. One depends on having the alternative proteases that are insensitive to the inhibitors (48). These insensitive proteases can occur constitutively in the plant and/or induced when the other proteases are inhibited to compensate their loss (49,50,52). The second mechanism involves the presence of the alternative proteases, which degrade the protease inhibitors so as to reduce their inhibitory activity (26, 53). *S. exigua* has been reported to adapt to potato proteinase inhibitor II by induced gut proteinase activity, which is insensitive to the inhibitors (51, 52). Further, when fed on the Soybean Proteinase Inhibitor (SPI) containing diet, larval proteases showed insensitivity to the inhibitor (51, 54). Similarly, Brioschi et al. (51) reported that *S. frugiperda* adaptation to SPI involves *de novo* synthesis and also up-regulation of existing enzymes such as chymotrypsins and trypsins. The diamondback moth (DBM), *Plutella xylostella* (L.) larvae have been found to be insensitive to Mustard

Trypsin Inhibitor 2 (*MTI2*) (55), which have been attributed to degradation of MTI2 by DBM, thus avoiding the effect of the protease inhibitor.

Gene expression studies have revealed that 12 digestive serine proteinases were either up- or down-regulated in *H. armigera* when fed on soybean Kunitz-type trypsin inhibitor (56, 57). Bown et al. (57) further observed that the trypsin-like proteinases genes are both up- and down-regulated two to 12-fold in *H. armigera* when fed on PI containing diet. Trypsins insensitive to plant PIs have been characterized from *Agrotis ipsilon* (Ffn.), *Trichoplusia ni* (Hub.) and *H. zea* (58). The larvae contain higher levels of inhibitor resistant proteolytic enzymes when fed on the artificial diet containing soybean trypsin inhibitor (59). Cysteine proteinases insensitive to inhibitors have been reported in the larval guts of Colorado potato beetles, *Leptinotarsa decemlineata* (Say), when fed on potato leaves with high levels of endogenous proteinase inhibitors (50). Similarly, the expression of cysteine proteinases, intestains A and C, which are resistant to the PIs, increase in Colorado potato beetle feeding on potato plants with induced PIs (60). A new trypsin-like enzyme is produced in *S. frugiperda* larvae when fed on artificial diet with soybean PIs (54). The new enzymes resistant to the inhibitors synthesized and/or secreted in insects are regulated by the ingestion of PIs in a dose- and time-dependent manner (59), and has been confirmed that insects may contain a large number of genes for proteinases. For example, about 28 genes of serine proteinase family occur in *H. armigera* (57). Proteolytic inactivation is an important adaptation developed by insects to withstand the proteolytic inhibition by PIs. Some coleopteran and lepidopteran larvae showed proteolytic inactivation of the PIs mediated by the insect's midgut proteinases (53). Insects express PI resistant enzymes when exposed to the PIs. For example, when *Heliothis virescens* (Fab.) larvae were fed on PI containing diets, they expressed the putative PI-resistant trypsins (52, 56, 57).

In *Callosobruchus maculatus* (Fab.), about 30 different cDNAs encoding major digestive cathepsin L-like cysteine proteases (CmCPs) have been cloned, and the transcripts and protein products undergo modulation (61). These CmCPs can be CmCPA and CmCPB, based on the sequence similarity. CmCPB has higher proteolytic activity, highly efficient in converting zymogens into active forms and greater protease inhibitor activity against soybean cysteine protease inhibitor N (scN) (62). Bruchids fed on scN containing diet expressed more CmCPB than CmCPA (63), thus can cope with PIs easily. The PIs, though considered as important and highly effective defense components of plant resistance, in most of the cases, no longer serve as a resistant components in plants against insect pests.

3.2. Glucosinolate–myrosinase system and insect adaptations

The most studied insect-plant defensive system is the glucosinolate-myrosinase system of Brassicae and is considered as the most effective defense system against

insect pests. In Brassicaceae, glucosinolates and myrosinases are hydrolyzed to toxic isothiocyanates (mustard oils) and other biologically active products (64). Together the glucosinolates and myrosinases constitute an activated plant defense system known as the ‘‘mustard oil bomb’’. The abrupt release of these compounds produces toxicity in insect pests (64, 65). Glucosinolate–myrosinase system is as defensive in plants as commercial insecticides. High glucosinolate and myrosinase containing lines of *Brassica juncea* L. are more defensive against *Spodoptera eridania* (Cramer) larvae than the ones with less glucosinolate and myrosinase content (65). However, adaptation of the *P. xylostella* and many other insect pests to glucosinolate-myrosinase system has nullified the value of this system as a defensive strategy. The glucosinolates and the specific enzyme (myrosinase) are stored in separate compartments of the cells (idioblasts and guard cells), and are distributed in different parts of an organ unevenly (30). They are stored in special sulfur-rich cells called as S-cells situated close to the phloem (66). When the tissue is damaged, the glucosinolates and myrosinases come in contact with each other, and the glucosinolates are hydrolysed to highly toxic products, such as isothiocyanates. These isothiocyanates are the important plant defensive compounds, however, insect pests have developed adaptations to reduce and/or evade the toxicity of glucosinolates, and have even evolved strategies to sequester them and use them for their own defense. For example, *P. xylostella* modifies the glucosinolates by sulfatase gut enzyme avoiding their hydrolysis (12). *Myzus persicae* (Sulz.), *Athalia rosae* (L.) and *Pieris rapae* (L.) sequester glucosinolates into their hemolymph and body tissues (67-69). When a predator attacks, the haemolyph oozes out glucosinolates that deter the predators such as ants and predatory wasps (67). Aphids such as *Brevicoryne brassicae* L. and *Lipaphis erysimi* (Kalt.) sequester glucosinolates from phloem sap (68, 69).

Furthermore, many lepidopterans especially those belonging to Pieridae family such as *P. rapae* possess nitrile specifier protein (NSP) in their midgut, which evades the toxicity of glucosinolates (70). The NSP activity in the guts of *P. rapae* modulates the hydrolysis of glucosinolates and leads to the formation of nitriles instead of toxic isothiocyanates (70). The unstable intermediate formed during glucosinolate hydrolysis serve as a direct substrate for NSP (71). *Spodoptera littoralis* Bios. larvae develop faster on *A. thaliana* lines producing nitrile as compared to those producing isothiocyanate (71). However, some evidences suggest that NSP switch the plant defense from direct to indirect, which are more effective against the specialist herbivores (72). The glucosinolates in cabbage stimulate oviposition by *P. rapae* on the leaf surface (73), and also act as feeding stimulants for the larvae, which feed on glucosinolate containing plants (64). When *A. thaliana* plants are infested with *P. rapae* larvae, they modulate the plant defense system in such as way that more nitriles are produced at the expense of isothiocyanate by inducing AtNSP1 (74).

3.3. Adaptation to tannins

Tannins are the polyphenolic compounds involved in plant resistance against insect pests. They strongly bind to proteins or to digestive enzymes in the gut, thereby reducing their digestibility by insect pests and thus affecting insect growth and development. In addition, tannins also act as feeding deterrents to many insects because of their astringent (mouth puckering) nature (75). Tannins form hydrogen or covalent bonds with the protein $–NH_2$ groups, which leads to precipitation of proteins and the digestive enzymes of herbivores. Furthermore, chelation of metal ions in insects by tannins reduces their availability to the insect pests, thus affecting growth and development. Tannins have also been reported to inhibit feeding, cause midgut lesions, and pharmacological toxicity (75, 76). However, insects have developed several adaptive mechanisms to avoid the toxicity of tannins. The potential mechanisms insects use to avoid toxicity of tannins include alkaline gut pH (77), tannin absorption through peritrophic membrane (76), polymerization (78) and excretion of the polyphenols after concentration (79). The surfactants formed as products of lipid digestion in the gut lumen prevent precipitation of proteins (80). Oxygen levels in foregut also play an important role in toxicity of tannins. At higher pH, oxygen levels are low and reduce auto-oxidation of tannins, thereby, lowering their toxicity. However, it is still a mystery whether the oxidation is due to the reaction of tannins to low oxygen and/or with the ferric ions. The antioxidative system of insects also plays an important role in reducing the tannin toxicity. For example, ascorbate reduces the oxidation of tannins and formation of reactive oxygen species (ROS) in insect gut (81). The grasshoppers possess a strong midgut antioxidative defense, which enables them to withstand tannins. This antioxidative defense mainly comprises of glutathione, α-tocopherol and ascorbate (82). The tolerance to tannins and peritrophic membrane association in *Schistocerca gregaria* (Forsk.) has been attributed to the ultrafilteration of tannins (76). In some species including *Melanoplus sanguinipes* (Fab.), tannic acid does not bind to the peritrophic membrane (83). In addition, peritrophic membrane prevents insect epithelium from the lesions and damage by ROS by adsorbing highly reactive ferrous ions (75).

3.4. Detoxification of plant metabolites

Enzymatic detoxification of toxic chemicals mediates the adaptation of insects to plants allelochemicals and plays an important role in chemical based insect-plant interaction (84). Insects deploy various enzymes for detoxification of insecticides and plant allelochemicals, however, of which cytochrome P450s and glutathione-S-transferases (GST) are the most important (84, 85). Insects react strongly to the toxic allelochemicals, when provided with the natural host plant diet or incorporated in the artificial diet, by increasing the metabolic mechanisms that result in the production of monoxygenases and GST (81, 85). The mechanisms of detoxification that operate in insects depend on the host plant chemistry, and its levels are generally influenced by the concentration of allelochemicals in the plant (85, 86). The role of insect detoxification enzymes in the metabolism of insecticides,

allelochemicals, and other xenobiotics has been studied in considerable detail (84-87).

3.4.1. Role of Cytochrome P450 in insect adaptation to plant defense system

The P450 enzymes constitute a diverse category of enzymes involved in insect resistance to insecticides and host-plant chemicals. They metabolize the plant chemicals and pesticides, and convert them into highly reactive, unstable polar compounds, which in turn are metabolized by secondary enzymes. Insects contain about 100 P450 genes and thus possess a great diversity in their structure and function (88).

The P450s are regarded as one of the important players in insect-plant co-evolution, since they are used both by plants (to produce toxins) and by insects (as a means of detoxification) (89). The desert dwelling species of *Drosophilla mettleri* Heed. feeding on cactus containing toxic allelochemicals possess inducible amounts of P450 involved in the metabolism of these toxins (90). The metabolism of isothiocyanates such as 2-phenylethylisothiocyanate, indole-3-carbinol and indole-3 -acetonitrile in *S. frugiperda* midgut microsomes is CytP450 dependant (91). Adaptation of lepidopteran insects to plant secondary metabolites such as furanocoumarins has been attributed to P450s, depending on the host plant. For example, *Papilio polyxenes* Fab. (black swallowtail) feeding on plants containing furanocoumarins tolerate up to 0.1% xanthotoxin in diet (92), which is detoxified by P450 monooxygenases (93). A number of P450s involved in detoxification of plant chemicals have been isolated from insect herbivores such as *Depressaria pastinacella* Dup. (94), *M. sexta* (95) and *Helicoverpa* species (33). A clearer picture of involvement of P450 in detoxification of plant allelochemicals came after the sequencing of CYP6B1 from *P. polyxenes*, which codes for P450s. Expression of CYP6B161 and CYP6B162 coding for P450s are induced in lepidopteran cell lines indicating the involvement of P450s in metabolism of linear furanocoumarins, such as xanthotoxin and bergapten (96). Furthermore, the conversion of dihydrocamalexic acid to camalexin, which are the major *Arabidopsis* phytoalexins, is catalysed by cytochrome P450 PAD3 (97). Aphid resistance to glucosinolates is attributed to the CYP81F2, which is a downstream part of the indolic glucosinolate pathway (98).

P450s have also been characterized from many other insects where they play an important role in metabolizing plant secondary metabolites. For example, in *Musca domestica* L., CYP6A1 metabolizes the terpenoids (99), in *H. armigera*, P450 mono-oxygenase CYP6AE14 detoxifies gossypol (100), in *Anopheles gambiae* Giles, CYP6Z1 metabolizes xanthotoxin and bergapten (furanocoumarins), furanochromones, and natural myristicin, safrole and isosafrole (101), while as CYP6Z2 metabolizes xanthotoxin, lignan, piceatannol and resveratol (102), and in *Diploptera punctata* (Esch.), CYP4C7 hydroxylates sesquiterpenoids (103). Bark beetles such as *Ips pini* Say and *Ips paraconfusus* Lanier detoxify the monoterpenes, sesquiterpenes and diterpenoid resin acids by P450s (104).

3.4.2. Role of Glutathione-S-transferases (GSTs) in insect adaptation to plant defense

The GST is involved in insect resistance to host plant defense by detoxification of xenobiotics and catalyzation of the conjugation of electrophilic molecules with the thiol group of reduced glutathione, which results in their rapid excretion and degradation (84, 86). This family of enzymes has been implicated in neutralizing the toxic effects of insecticides that are neurotoxic and/or affect insect growth and development. These include spinosad, diazinon, DDT, nitenpyram, lufenuron and dicyclanil (105). A number of reports have advocated the role of GST in insect adaptation to plant glucosinolates or other plant secondary metabolites incorporated into the artificial diet in *S. frugiperda*, *S. litura*, *T. ni*, *M. persicae*, *Aulacorthum solani* (Kalt.) and *Acyrthosiphon pisum* (Harris) (84, 104, 106). The overproduction of GST in *M. persicae* has been attributed to insect adaptation to glucosinolates and isothiocyanates in members of Brasicaceae, although there is no direct confrontation of isothiocyanates, because aphids directly insert their stylets into the phloem (84, 107).

4. Conclusions and perspectives

The counter defense by insects to constitutive and induced defens~~ive~~ response in the host plant is highly complex, and ~~has~~ poses major ~~d a big~~ challenge to develop cultivars with stable resistance to insects for ~~insect~~ pest management ~~programs~~. It is ~~highly~~ therefore important to ~~broaden the base of our~~ gain an understanding of ~~the~~ insect adaptation to plant defense, and develop the ~~measures~~ strategies to minimize the effect of such ~~prevent~~ such adaptations on expression of resistance to insects. Induced resistance in plants against insect pests ~~will be a better option to deal~~in one of the important components of resistance to insects, and makes the plants ~~with this as it would make plants~~ phenotypically plastic and indeterminable for the insect pests. The insects will have limited time to respond to induced defense of the hot plant as such mechanisms are expressed only when the plants are challenged by the hevbivores. ~~Furthermore, the d~~Development of plants with a capability to respond to herbivore attack ~~defensive compounds having wide range of mode of actions~~ will not only be effective for pest management, but also slowdown the counter adaptation by the insect pests. ~~be effective against insect adaptation.~~

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