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Insecticide Use Impacts on Pest Resistance: An Evidence from Diamondback Moth

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Abstract

Diamondback moth (DBM) has been important pest of crucifers since years ago worldwide. The first DBM resistance was reported on dichlorodiphenyltrichloroethane (DDT), from then it has developed resistance to other insecticides. Its ability to build resistance to almost every insecticide introduced is a great concern to the agriculture world, owing mainly to its short life cycle, continuous host availability and its genetic elasticity. Numerous resistance mechanisms displayed by DBM to counterattack effect of insecticides, either behaviorally, physiology or by biochemical. This paper is focused on diamondback moth (DBM) resistance to insecticides, its biochemical mechanism and the potential to become cross resistance to other insecticides. We highlighted the biochemical reactions in DBM resistance and emphasized on enzymes responsible to the resistance. The information on the mechanism could provide valuable information to other researchers in designing a rapid and sensitive biochemical assay in detecting DBM resistance to many insecticides that has yet to be discovered. The potential of DBM to develop cross resistance to other insecticides is also stressed since the issue is always co-related with biochemical resistance mechanisms.

Keywords: Biochemical resistance mechanisms; diamondback moth; resistance.

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1. Introduction

Diamondback moth (DBM), particularly known as *Plutella xylostella* (Lepidoptera: Yponomeutidae) has been acknowledged as an important pest of crucifers worldwide. It is a specialist phytophagous and the diet is exclusively restricted only to crucifers due to the presence of glucosinolates. Crucifers grouped under family Brassicacea where the family consists of many economically important crops such as oilseeds (e.g., mustard), cole crops (e.g., cauliflower and cabbage) and root vegetables (radish and turnip). There are many studies reported on vegetables / crop damages by the DBM, highlighting it notorious pest status [1, 2, 3, 4]. The insect plays a vital pest role, covering both lowland and highland areas whether in temperate, tropical or subtropical regions [5, 6, 7, 8]. DBM is well distributed wherever its host plant available and is considered as the most universal Lepidopteran pest. Availability of host crop throughout the year, the diversity and abundance of the host, serious overlap of generation and limited alternative control method implemented are attributed to the pest status of the DBM [3, 8, 9].

The insecticides have been misused greatly due to unavailable technical assistance and this contributed to selection of resistant individuals. Table 1 summarized the history of DBM resistance to several insecticides. Resistance of the DBM to insecticides has lead farmers to adapt rotational insecticides techniques in the farms. However, by times, the condition become worsens because of cross resistance incidences due to exposure of the DBM to various insecticide groups. In most cases, cross resistance occurs when the insect is rendered to newer insecticides that are chemically–related to previous insecticides. It could seriously disrupt pest control program by reducing effectiveness of many insecticides. According to [10], the insecticide fipronil suffer cross-resistance from endosulfan and dieldrin. The ability of broad spectrum insecticides in controlling DBM has seriously declined resulting from resistance and cross resistance by the insect [8, 11].

It is essential to implement a resistance monitoring program at the early stage of development, due to ability of DBM to rapidly evolve resistance to almost all insecticides introduced to it. On that principle, the biochemical basis of the resistance must be truly understood in order to devise strategies to delay resistance in the insect. The understanding of the resistance will allow us to apply existing insecticide to which resistance has already developed in a wiser manner. It could also provide an idea of cross resistance pattern among the insecticides, either the insecticides are in the same or different groups. Therefore, the constituents that contribute to resistance in insect and its mechanism could serve as keys in managing the resistance problem in DBM. Subsequently, excellent resistance monitoring program can be achieved by designing novel approaches to prevent or minimize the evolution of resistance.

This paper considers ways in which an improved understanding of resistance in DBM, the biochemical resistance mechanisms developed by the insect and cross–resistance pattern. Such knowledge is important as it could be integrated in insecticide resistance management.

2. Resistance mechanism

In general, resistance arises through alteration in target site of the insect [34, 35], metabolic detoxification and

reduces in cuticular penetration [36]. This paper will only emphasize on metabolic resistance in insecticide resistance. Table 2 shows insecticides compounds with its mode of action and resistance mechanisms developed by the insects.

Table 1: History of resistance cases in DBM

Year	Active Ingredients	Group	Country of first case recorded	References
1953	DDT	DDT	Indonesia	[12]
1967	Aldrin	Cyclodiene	Vietnam	[13]
1967	Carbaryl	Carbamate	Vietnam	[13]
1967	Diazinon	Organophosphate	Vietnam	[13]
1967	Dieldrin	Cyclodiene	Vietnam	[13]
1974	Trichlorfon	Organophosphate	Malaysia	[14]
1978	Metamidophos	Organophosphate	Malaysia	[15]
1981	Cypermethrin	Pyrethroid	Taiwan	[16]
1981	Deltamethrin	Pyrethroid	Taiwan	[16]
1981	Fenvelerate	Pyrethroid	Taiwan	[16]
1983	Cynofenphos	Organophosphate	Japan	[17]
1983	Cynophos	Organophosphate	Japan	[18]
1983	Dialifos	Organophosphate	Japan	[17]
1989	Bt *kurstaki*	*Bacillus thuringiensis*	United States of America (USA) (Hawaii)	[19]
1991	Chlorpyrifos	Organophosphate	Australia	[20]
1991	Esfenvelerate	Pyrethroid	Australia	[20]
1992	Carbofuran	Carbamate	USA	[21]
1992	Cyhalothrin	Pyrethroid	USA	[21]
1993	Bt*aizawai*	*Bacillus thuringiensis*	USA	[22]
1995	Abamectin	Abamectin	Malaysia	[23]
1995	Bt Cry1Da	*Bacillus thuringiensis*	Malaysia	[23]
1996	Bt Cry1C	*Bacillus thuringiensis*	USA	[24]
1998	Bt Cry1Aa	*Bacillus thuringiensis*	India	[25]
1998	Bt HD73	*Bacillus thuringiensis*	India	[25]
2000	Emamectin benzoate	Avermectin	USA	[26]
2000	Spinosad	Spinosad	Thailand, USA	[27]
2000	Acetamipirid	Neonicotinoid	Japan	[28]
2001	Indoxacarb	Oxadiazine	USA	[29]
2004	Cyhalothrin	Pyrethroid	Pakistan	[30]
2004	Tebufenozide	Diacylhydrazine	China	[31]
2008	Bt Cry1Ac	*Bacillus thuringiensis*	Malaysia	[32]
2008	Bt Cry1Ca	*Bacillus thuringiensis*	Malaysia	[33]

Table 2: Insecticides and enzymes responsible in biochemical resistance possessed by the insects.

Insecticide Group	Detoxification Enzymes	References
Avermectin	Glutathione S – transferases (GST);	[37]
	Cytochrome P-450;	[38]
	General esterases	[39]
Pyrethroid	Cytochrome P-450;	[40]
	Esterases	
Organophosphates	Carboxylesterases;	[41]
	GST	[42]
Organochlorines	GST	[43]
Spinosyns	Microsomal-O-demethylase	[44]
Indoxacarb	Esterases	[45]
IGR (Pyriproxyfen, tebufenozide)	Cytochrome P-450	[46]
Neonicotinoids	Cytochrome P-450	[47-48]
Chlorfenapyr	Esterases	[49]

In metabolic resistance, the detoxification enzymes act in cascade [35]. Metabolic detoxification occurs when toxins are modified by several enzymes, resulting in excretion of the modified toxin before reaching its target. The detoxification of the toxin involving three phases: recognization and hydrolysis of the toxin, conversion of toxin into non toxic molecule and excretion of the molecule out from insects' body.

Previous findings have highlighted the role of the enzymes in resistant DBM and other insects [50-52]. Several important enzymes are well known to participate in metabolic detoxification, including hydrolases (general esterase, carboxylesterase, β–glucosidase, carboxylamides), reductases (juglonereductase, cytochrome c reductase) and microsomal oxidases (epoxidases, hydroxylases, sulphoxidase, N–demethylase, O-dealkynases) [53-57].

2.1. Metabolic detoxification

2.1.1 Glutathione-S-transferases (GST)

Many studies have suggested GST as essential enzymes in resistance based on its high level incorporated in

insecticide resistant strain insects. GSTs are dimeric multifunctional enzymes that function to detoxify various xenobiotics substances [58]. There are two families of GST have been found in insects; microsomal and cystolic, classified according to their location within the cell, both are important in insecticide resistance in insects.

The function of GST is mainly to detoxify both endogenous and xenobiotic compounds either directly or by catalysing the secondary metabolism of compounds that have been oxidized by cytochrome P450 family [34]. GST degrade insecticides through O–dealkylation, O–dearylation, dehalogenate or/ and conjugate, depend on the insecticide involved in resistance development in the insect. During O–dealkylation, alkyl portion of the insecticides will be conjugated with glutathione [59] while O–dearylation referred to reaction of glutathione with the leaving group [60].

Previous studies proved the elevated level of GSTs enzymes is frequently associated with resistance towards the insecticides. GST usually implicits degradation to some OPs [41, 61] in DBM. Among the four GST isoenzymes that have been acknowledged in DBM, GST-3 and GST-4 have several biochemical and toxicological similarities. Their conjugation activity was higher toward 2-dichloro-4-nitrobenzene (DCNB) and some organophosphates compared to the other two isoenzymes [60]. As stated by [62], there is A 3–4 fold increase in GST activity was found in resistant strain of diamondback moth field population compared to the sustainable strain.

GST also plays a role in abamectin–resistant strain of DBM [46]. In 2004, [63] revealed the methamidophos resistant-DBM showed 1.5 fold higher GST level over unselected strain. Similarly, [64] suggested that higher GST activity in methamidophos-exposed field population DBM compared to its natural enemies in the same population is due to resistance it developed to the insecticide. Recently, observation showed that GST activity in the whole body of homogenate DBM was markedly higher than the susceptible strain [65], strengthen the hypothesis that the enzyme is important in detoxifying strategies developed by the lepidopterans.

2.1.2 Monooxygenases

Monooxygenases (mixed function oxidases - MFO) is one of the most significant detoxification enzymes that are ubiquitously found in insects [66]. The enzymes have a major role in endogenous metabolism activity and important for adaptation of toxins in host plants dwelled by the insects. They catalyze the formation of a substrate from single molecular atom oxygen together with reduction of the other atom to water. Enhancement of the enzymes had been recorded to build up resistance towards neonicotinoid and pyrethroid in many insect pests including DBM [67], *Bemisia tabaci*, *Blatella germanica* and *Tribolium castaneum* [68-70].

Monooxygenases–mediated resistance is caused by increase of the detoxification activity of the enzymes. The resistance could happen due to altered catalytic activity of P450 or change in expression level of the protein [71]. A study by [72] suggested that P450 can be considered to contribute to resistance only when it sequester the compound to which the strain has monooxygenase–mediated resistance and secondly, when P450 is higher in resistant strain or the protein encodes for resistant strain allele have greater catalytic activity compared to that

of the susceptible ones. P450s involves in the detoxification of organophosphate as well as activation of the parent compound of the insecticide, such as diazinon (a phosphorothionate) into a phosphate diazoxon insecticide [73].

Earlier study found out that resistance to OPs in many insect pests and arthropod was caused by increased levels MFO mediated detoxification enzymes [74]. Similarly it is revealed that enhanced detoxification by P450 enzymes is the commonly found resistance mechanism to at least four insecticides classes which are OPs, carbamates, pyrethroid and macrocyclic lactones [75].

In addition, laboratory studies conducted by [46] found that increase of MFO activity in the tebufenozide–resistant DBM could confer significant cross–resistant to abamectin. Activity of cytochrome P450 was recorded 2.5 times higher in the carbaryl and methyl parathion resistance Lepidopteran compared to the susceptible strain [76]. A rapid decline of monooxygenase activity together with GST and carboxylesterase was in line with increased susceptibility to fufenozide in resistance DBM during the first generation [77].

2.1.3 Esterases

Insect esterases involve in both physiological and defense system and are found in soluble and membrane found forms. Numerous studies have revealed that esterases is crucial in contributing to insecticide resistance among insects and other arthropod species, specifically to the OPs, carbamates and pyrtehroid [9, 25, 46]. The esterases contribute to broad spectrum insecticide resistance via rapid – binding and slow turnover of the insecticide. The enzyme also produces narrow – spectrum resistance through metabolism of a very limited range of insecticide that shared a common ester bond [78].

According to [79], resistance to prefonofos was highly correlated with esterase activity, suggesting role of the enzyme in the resistance. Similarly, the linked between esterase with OPs and pyrethroid was also evident in the insecticide resistance cases [80-81]. Therefore, [82] suggested that esterase assay can be utilized as a biochemical marker in the monitoring insect resistance in the field population.

According to [55], resistance to malathion is attributed to elevated carboxylesterase activity, which the insecticide become less toxic after hydrolyzation process and more easily excreted from the cell. In addition, the resistance caused by esterase in malathion in some insect has been found to be associated by a single autosomal gene which inherited as dominant trait by the progeny. In resistance to pyrethroid, major route of the resistance is hydrolysis of the ester bond that is similar in almost all pyrethroids.

Trans isomers pyrethroid is more preferable by insect esterases because of the position of ester and substituted vinyl groups that located opposite sides of the cyclopropane ring. According to [83], mutant carboxylesterases play a role in esterase–mediated metabolic resistance by degradation or sequestration of the insecticide. During sequestration, an esterase that binds to any insecticide with high affinity is overexpressed that usually caused by gene amplication to a certain degree that permit the effective sequestration of the toxin.

3. Potential of cross – resistance

Cross resistance is defined as the ability of an insect to confer resistance to an insecticide without being exposed to the insecticide. Insecticides having specific chemical group will have similar target site, therefore the mode of action is similar. In such group, when an insect become resistant to an insecticide, it is possible for the insect to confer cross resistance to another insecticide in the group [84]. In chemically unrelated insecticides, cross resistance commonly happen through enhancement of enzymes responsible for resistance. Pattern of cross resistance in DBM is crucially important. They provide information on couple of insecticides that would be safely used and delay the resistance in pest management program.

Tebufenozide, an insect growth regulator was used in selection with susceptible DBM strain in China. The selected strain exhibited a high resistance ratio (RR=93.8) and later the strain showed high cross resistance to abamectin (RR= 35.7) and methoxyfenozide (RR=29.1) and low resistance to deltamethrin (RR=3.9) [31]. Although both tebufenozide and abamectin having different mode of action, the study indicated obvious cross resistance between the insecticides. Further study confirmed that the resistance is caused by metabolic detoxification, with high mixed function oxidase (MFO) activity recorded in tebufenozide and abamectin-resistant strain [46].

Some *Bt* Cry such as Cry1Ja, Cry1Ab and Cry1Ac shares common binding site in midgut of DBM, suggesting the event of cross resistance. Although Cry1Ja binds with high affinity to only a small part of Cry1Ac and Cry1Ab binding sites, any changes in the binding site might as well confer resistance to Cry1Ja [85]. In contrast, Cry1Ja was not competence to Cry1Ab and Cry1Ac in other lepidopteran pest *Helicoverpa armigera* [86]. In other cases, extremely reduced binding of Cry1A (Cry1Ab and Cry1Ac) and Cry1Fa were detected in resistant DBM [87-88].

Previous studies showed that DBM resistance to Cry1A *Bacillus thuringuensis* subsp. *Kurstaki* confers cross – resistance to Cry1F, but not to Cry1B, Cry1C, Cry1D and Cry9C due to differences of their binding sites from Cry1A [89-91]. Cross resistance to spinosyn analogs (3'-O- ethyl spinosyn) and spinetoram are well established in spinosad resistance DBM strain [92]. Study conducted by [33] demonstrated that the Cry1Ab-SEL sub-population showed fairly high cross resistance to Cry1Ac. The cross–resistance within Cry1A family is expected because they bind to same receptor site [93] and share more 80% homology sequence [94].

Potential of cross-resistance between two diamide (flubendiamide and chlorantraniliprole) insecticides were studied [95]. Both insecticides has same mode of action; they selectively binds to ryanodine receptoes (RyR) in insect muscles and release calcium, uncontrolly. While chlorantraniliprole was extensively applied in the field, flubendiamide is not yet used in field scale when the study was conducted. It is interesting though to note that significant toxicity correlation was documented between the diamides, implying that there is cross-resistance. It is suggested that resistance to chlorantraniliprole is mediated by metabolic detoxification at low level. However, the information on cross-resistance between the insecticides is limited because to date, no report recorded its cross-resistance to insecticides addressing ryanodine receptor. Therefore, target-site resistance must not be excluded as molecular studies should be considered to investigate the target-site resistance in diamides.

Study on potential of cross resistance between two sodium channel blocking insecticide (metaflumizone and indoxacarb) disclosed no cross resistance occurred between the compounds [96]. This suggested that although both compound shares same target site, different primary resistance mechanism may takes place. Evidences revealed that detoxification enzymes have primary role in indoxacarb resistance [45, 97-98]. Meanwhile, spinosad-resistant DBM CH_1 strain revealed to confer high level of cross resistance to abamectin [99]. Although both compound are from different groups and have different target sites, but they both are macrocyclic lactones therefore cross resistance might happened due to metabolic detoxification. However, argument arose corresponding to the finding because CH_1 strain was exposed to abamectin prior selection with spinosad. Several cases of cross resistance were summarized in Table 3.

Table 3: Cases of cross resistance of insecticides in DBM.

Strain (Resistant)	Cross resistance to	Resistance Ratio	References
Fufenozide	Methoxyfenozide	37.6	[100]
	Hexafluron	28.7	
	Tebufenozide	27.9	
	Diflubenzuron	17.2	
	Abamectin	7.02	
Tebufenozide	Abamectin	35.7	[31]
	Methoxyfenozide	29.1	
	JS118	16.5	
Phentoate	Phosphamidon	29.3	[101]
	Chlorpyrifos	26.5	
	Chlorpyrifos-methyl	23.4	
	Parathion	51.1	
	Pyrachlofos	59.9	
	Phosmet	72.5	
	Prothiofos	52.7	
	Phosphonothiolate	38.7	
	Prothiofos	120.0	[102]
	Cynophos	95.0	
	Methomyl	381.0	
Abamectin	Fenvelerate	20.2	[103]
Cry1Ab	Cry1F	> 240	[91]
Cry1Ab	Cry1Ac	40	[33]

4. Insecticide resistance management

4.1 Focus on IPM

An effective resistance management program does not depend solely on a single approach. Reducing input on conventional insecticides and its rotation is only a part of management program. In fact, effective management strategy relies on early resistance detection and monitoring the resistance potential in the pest population [82]. For that purpose, integrated pest management (IPM) and integrated resistance management (IRM) have been commenced. IRM was developed to overcome evolving resistance problems of the pests towards various insecticides including the newer ones. It works by reducing the selection pressure to any given insecticides to slower resistance development in the resistance–developing target pest species. To attain a successful IRM, one must understand nature of the pest and the insecticides together with appropriate IRM tactics that will be devised. This includes analyses and capabilities to comprehend the risk of resistance in a particular system and its possibility to mitigate [104-105]. Nevertheless, these scientific assessments of resistance risk and proper management strategies are only the initial step in an effective IRM.

Compared to developed countries, smallholders in developing countries usually have less access on educational materials and the number of supportive consultants and extension services are inadequate [106]. Therefore, in the countries, the main focus is to make good management decisions especially related to the insecticides application procedure. On this basis, various regional and international organizations played major roles in developing proper educational programs on pest management. Farmer Field School (FFS) established by the Food and Agricultural Organization (FAO) implementing IRM and IPM in over 30 developing countries worldwide, which aimed to make farmers more experts through hands–on training. The principles included in this program is to grow healthy crops, conserve natural enemies in the field and to observe crops frequently to make suitable decisions on pest control [107]. In addition, extensive effort has covered the identification of natural enemies of diamondback moth, educating farmers on the benefits of the insects and supplying the insects via inundative release [108].

Effective IRM program relies on early resistance detection and accurate characterization of the spatial resistance distribution by monitoring [29]. To conduct monitoring program, it needs suitable assay system for evaluating pest susceptibility to many insecticides. A trustworthy assay system must be simple, cost-effective yet sensitive enough to obtain accurate result including on large number of insect [109]. In fact, effective IRM greatly depends on consistent scouting and resistance monitoring to make appropriate decisions on insecticide applications. Meanwhile, IPM comes as a concept to solve pest problem after chemical control did not meet the expectations in managing the pest problem and created more problems than solutions.

As stated by [110], the program is not a static one for it must be adjusted and improved as new management means become available. At present, IPM is accepted as the most promising approach in pest management program as it implements systematic insecticides application in combination with improved cultural and biological – based techniques. In the program, the chemical input is strictly controlled where it will be used when absolutely needed to avoid the pest from reaching economically damaging densities. Reduced input of

conventional insecticides has benefited the natural enemy in a way that it can build up the population and survives in the field. Therefore, selection for more 'soft' insecticides that has less effect to natural enemies is important for conservation, enhancement and collaboration of natural enemies such as predators and parasitoid. It is suggested that insecticides should be applied according to regular crop sampling for occurrence of pests and natural enemies, accurate pest identification and the economic threshold defined for the pests [111-112].

5. Conclusions

Recent IPM strategies widely proposed on controlling DBM resistant management includes the biological, cultural and chemical techniques. However, insecticide is still the main approach use to control DBM as well as other pests until improved alternatives are found. Therefore, it is a need to recognize the relationship between the insect and the chemicals. An ample understanding of resistance basis in insect shall be a key step in managing the resistance problem. Knowledge on the aspects is fundamental to devise effective strategies for resistance management programs. It appears that unless appropriate management strategies are devised, the resistance problem may become unmanageable in the foreseeable future. Baseline susceptibility should be prepared for every insecticides compound especially for a new chemistry insecticide. In addition, it should be tested with appropriate procedures towards resistant population before its introduction to the field. Prior to the introduction of an insecticide with a new chemistry, it is suggested that proactive monitoring program can be developed. A refined resistance management that includes cooperative insecticide rotation would further enhance the durability of each insecticide.

Good collaboration between farmers, academic and industry researchers are strongly suggested since all of them are important parties in making the planned resistance management worked successfully. Many a problem would arise when dealing with top – down messages. Learning from the successfulness on rice IPM, the farmers that firmly understand IPM can established good biological control practices in their rice field. Thus, looking on positive side, the approach can be implemented in managing diamondback moth resistance.

Since the farmer need information on proper means in combating the insect and its resistance ability, it would be great if researchers can share their knowledge and work together with farmer in the field. The farmers should be able to make the right decision in pest monitoring, choose the effective and environmental friendly tools and apply it at the right time. It is also proposed that authorized staff provide guidance and monitor the farmers from initial stage of management program until it is well understood and established. Therefore, in managing the resistance in diamondback moth, it is not only planning that matters. The core thing is, all parties involved should understand their role and carried the program well.

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