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Research Article

Phytotoxicity Assessment of Several Herbicides on Oil Palm Seedlings-Post Topical Canopy Spray

Hafiz Ikhram Osman*

Department of Agronomy and Advisory Services, Kulim (M) Berhad, Malaysia

***Corresponding Author:** Hafiz Ikhram Osman* Department of Agronomy and Advisory Services, Kulim (M) Berhad, Malaysia.

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Abstract

Phytotoxicity identification and selection of appropriate herbicides are important in nursery upkeep. Young oil palm seedling is susceptible to phytotoxicity that caused short-long term effect or worse lethal. The herbicides of various mode of action that commonly used in oil palm environment were selected for screening. The phytotoxicity observation and visual rating were done at 7, 14, 30 and 60 days after treatment. Topical spray was done at standard and high concentration rates. ANOVA with Fisher LSD method analysis at 95% confidence showed significant symptoms between treated and untreated treatment. There were two significant phytotoxicity effect groups. Which were- (i) severe to complete destruction - 2,4-dimethylammonium 60%, fluroxypyr-meptyl 45.5%, indaziflam 45.5%, triclopyr-butotyl 32.1% and glufosinate ammonium 24.5%. (ii) Slight to moderate - glyphosate isopropylamine 41%, MSMA 39.5% + diuron 7.8%, diuron 40% + ametryn 40%, metsulfuron-methyl 20% and diuron 80%. Use of hormonal and cellulose biosynthesis inhibitor mode of action close to seedling in nursery and during field planting should be avoided.

Keywords: Herbicide Phytotoxicity; Oil Palm Seedling; Mode of Action

Abbreviations

DAS: Day After Spray; MOA: Mode of Action; HRAC: Herbicide Resistance Action Committee

Introduction

Herbicide is used commercially for oil palm nursery upkeep to maintain the seedlings quality. Chemical control is cost-effective, but care must be taken to choose the correct herbicides to ensure bio efficacy and crop safety [2]. Phytotoxicity refers to plant injury caused by exposure to chemical [6]. It can occur if the chemical is applied improperly by means of vapor or particle drifts, chemical run-off or persistent residues accumulate in the soil or on the plant. Phytotoxicity can cause temporary or long-lasting damage. The symptoms could be modifications in development cycle, thinning (mortality), modifications in colour, necrosis, deformation and effects on yield performance [3]. Pre- and post-emergence herbicides or combinations are used to ensure good weeds killing and long-term control. Phytotoxicity symptoms identification and knowledge are important to avoid losses and serve as the key point for herbicides selection. The objectives of this experiment were to identify chemical specific phytotoxicity symptoms and determine the appropriate herbicides for nursery stage and also in relation to immature area.

Materials and Methods

The experiment was carried out at the nursery in Kulim Agrotech Center, Malaysia. Main nursery oil palm seedlings aged 9 months and around 2.5ft height were selected for treatments. The treatments consist of 10 herbicides that were common in oil palm environment namely- (T1) glyphosate isopropylamine 41%, (T2) MSMA 39.5% + diuron 7.8%, (T3) diuron 40% + ametryn 40%, (T4) 2,4-dimethylammonium 60%, (T5) fluroxypyr - meptyl 45.5%, (T6) metsulfuron-methyl 20%, (T7) indaziflam 45.5%, (T8) triclopyr-butotyl 32.1%, (T9) diuron 80% and (T10) glufosinate ammonium 24.5%. Herbicide application was carried out to each seedling using low volume (LV) sprayer at 250 l/ha. There were two chemical rates, Standard and High concentration. Phytotoxicity symptom observation and visual rating were made at 7, 14, 30 and 60 days after spray.

Results and Discussion

Phytotoxicity symptoms were identified and described. The herbicide's active ingredients were grouped based on major injury symptoms that observed to be related to the mode of action and Herbicide Resistance Action Committee (HRAC) group (table 1). The injury levels were then evaluated in the form of visual rating

A Leaf defoliation	Primarily contact	GLUTHAMINE SYNTHETASE INHIBITOR *(Glufosinate ammonium - H - 10)* Affect nitrogen metabolism by Inhibiting convertion of glutamate and ammonia to glutamine. Consequently leads to accumulation of ammonium ions that inhibit photosynthesis and destroy cells. Translocation is limited which only occurs within contacted leaves. *Injury symptoms: Leaf burning appearance within hours followed by necrosis from tips margin towards centre within 7DAS.* ORGANIC ARSENICALS *(MSMA - Z - Ø)* Unknown mode of action. Multi actions that inhibit plant growth, distrupt plant metabolism and uncoupling phosphorylation. Rapid dessication indicates cell membrane destruction. *Injury symptoms: Leaf burning appearance within hours followed by necrosis from tips margin within 7DAS.*
	Post -contact	PHOTOSYSTEM II *(Ametryn - C1, Diuron - C2) - 5* Acts contact on foliar (post) but translocate through xilem on soil (pre) application. The chemical inhibits the electron transport and halt photosynthesis. The blocking cause lipid and membrane destruction. The chemical is classified based on attachment sites at D1 protein example of triazine and urea. *Injury symptoms: Intervienal chlorosis due to destruction of chlorophyl. Necrosis after lipid membrane destroyed within 14-30DAS.*
B Growing point rots	Shoot necrosis	EPSP SYNTHASE INHIBITOR *(Glyphosate-isopropylammonium - G - 9)* EPSP synthase produce aromatic amino acids that important in protein synthesis and growth. Chemical translocated to the actively growing point. *Injury symptoms: Spear rotting and collapsed within 14DAT with below the dead parts are still green. High dosage cause dieback that can be lethal. Epinasty could occur at certain point that cause bending and cells destruction.* ALS SYNTHASE INHIBITOR *(Metsulfuron-methyl - B - 2)* ALS synthase produce branch chain amino acids that important in protein synthesis and growth. Inhibition leads to cessation of cell division and halting the growth. Principally, chemical translocated to the apex of the plant. *Injury symptoms: Interveinal chlorosis and shoots collapsed within 30DAS.*
	Meristem collapsed and death	SYNTHETIC AUXINS *(2,4-dimethylammonium, Fluroxypyr, Tryclopyr) - O - 4* Translocation with accumulation principally at the shoots/roots and meristematic tissue. Interfere with cell formation and callus growth that cause abnormalities in multiple essential metabolism and growth processes. *Injury symptoms: Obvious whole shhoot apical meristem bending at 30DAS. Complete rotten and collapsed within 60DAT.* CELULOSE BIOSYNTHESIS INHIBITOR *(Indaziflam - L - 29)* The chemical inhibits cellulose (cell wall) biosynthesis and cell division that mainly occurred at the meristem thus higher effect to young oil palm seedling that yet to have fully callused matured brown bark stem. *Injury symptoms: Whole deterioration and necrosis at 30DAT. Complete rotten and collapsed within 60DAS.*

Table 1: Phytotoxicity grouping, injury symptoms and mode of action (MOA) based on Herbicide Resistance Action Committee (HRAC) [1,4,5].

Effect	Rating	Phytotoxicity symptom
None	0	No injury, normal
Slight	1	Slight stunting, injury or discolouration
	2	Some stand loss, stunting or discolouration
	3	Injury more pronounced but not persistent
Moderate	4	Moderate injury, recovery possible
	5	Injury more persistent, recovery doubtful
	6	Near severe injury no recovery possible
Severe	7	Severe injury stand loss
	8	Almost destroyed, a few plants surviving
	9	Very few plants alive
Complete	10	Complete destruction

Table 2: Visual scoring scale of 0 to 10 [7].

based on the scoring scale 1 - 10 (table 3) at 7, 14, 30 and 60 days after spray (DAS).

Phytotoxicity symptoms

Rapid symptom exhibited by contact action in glufosinate ammonium and MSMA which of leaves scorching and necrosis as early as 24 to 76 hours after spray.

Figure 1: Early contact herbicide phytotoxicity symptom at 7DAS. a) MSMA 39.5% + diuron 7.8% (T2) b) Glufosinate ammonium 24.5% (T10).

Initial herbicides phytotoxicity symptoms was chlorosis (interveinal or venal) that followed by dieback necrosis from the tips margin towards center. Whilst translocation and hormonal herbicides phytotoxicity showed mainly to the growing points that caused by the shoot or meristem deterioration.

The symptoms can be divided into two major groups

- **Leaf defoliation:** Leave injury can be observed clearly within 14DAS when the necrotic symptoms appeared resulted from desiccation due to cell membrane destruction usually started from the leave tip, margin or interveinal.
- **Growing point rots:** The effect of deterioration of apex or meristematic tissue can be observed as early as 7DAS or late at 30DAS. The symptoms could be of shoot rots, shoot bending or desiccation of young fronds. Whilst complete destruction was observed within 60DAS for hormonal and cellulose biosynthesis inhibitor chemicals.

Generally, phytotoxicity effect increases with degree of exposure and concentration. Higher rates exert quicker and more severe symptoms in this screening test. Phytotoxicity injury level was observed increased or correlated with chemical rate but the hormonal and cellulose biosynthesis inhibitor showed constant effect of lethal in both rates. This flagged the cautious of the chemical usage in nursery and immature area.

Figure 2: Photosystem I & II herbicides phytotoxicity symptoms. a) Interveinal necrosis at 7DAS b) Necrosis from tips margin at 14DAS c) Necrosis margin towards center mainly in older fronds.

Figure 3: Glufosinate ammonium 24.5% (T10) contact symptoms a) Persistent necrosis margin towards center within 14DAS b) Complete defoliation and destruction within 60DAS.

Figure 4: Shoot rots phytotoxicity symptoms in glyphosate isopropylamine 41% (T1), and metsulfuron-methyl 20% (T6), a) Necrosis at the new growing parts b) Complete destruction of the young parts whilst other remain green.

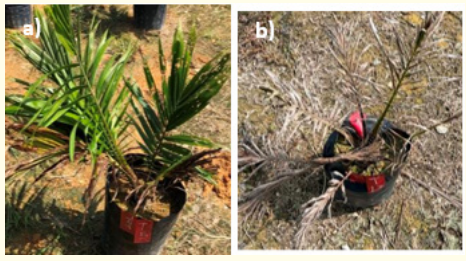

Figure 5: Meristem deterioration in hormonal and cellulose biosynthesis inhibitor chemicals a) Shoot bending at 14 - 30DAS b) Complete destruction due to shoot collapsed or meristem death within 60DAS.

Phytotoxicity rating

All treatments caused phytotoxicity under direct topical spray application at least of slight discoloration or chlorosis. There were 5 chemicals namely 2,4-dimethylammonium, fluroxypyr- meptyl, tryclopyr-butotyl, indaziflam and glufosinate ammonium that caused complete destruction (average 9.5 - 10) within 60DAS. All the above are translocation chemicals except for glufosinate ammonium. The translocation action transported the chemical by xylem or phloem to the shoot or growing point induced growth abnormalities and meristem death whilst the contact action or with minimum translocation destroys only the contacted cells area which highly depending on degree of exposure. Whole canopy defoliation and destruction in this experiment impeded physiological process that finally caused complete destruction.

Glyphosate isopropylamine (3) and metsulfuron methyl (4.8) also translocated towards growing shoots or younger fronds but with lesser severity effect. Glyphosate isopropylamine observed with much faster effect as compared to metsulfuron methyl and demonstrated quicker recovery within 60DAS.

Chemicals with contact action, MSMA + diuron (4.5) early symptoms was quite similar with glufosinate ammonium but less persistent. Diuron + ametryn (5.7) and diuron (5.7) also showed effect mainly to the mature leaves. Herbicide translocation through xylem accumulate in mature leaves that transpiring the most water. There was sign of recovery on the new shoots after the defoliation effect within 60 days.

The phytotoxicity rating was analyzed using ANOVA Fisher LSD method test ($P > 0.05$) for each observation timeline. Significant difference group of severe phytotoxicity to complete destruction were recorded in 2,4-dimethylammonium 60%, fluroxypyr - meptyl 45.5%, indaziflam 45.5%, triclopyr-butotyl 32.1% and glufosinate ammonium 24.5%. Photosystem HRAC group chemicals of diuron 40% + ametryn 40%, diuron 80% and MSMA 39.5% + Diuron 7.8% were generally moderate. Metsulfuron-methyl 20% was moderate. Glyphosate isopropylamine 41% showed slight to moderate symptoms increasing with concentration.

In standard rate, the severe and complete destructive chemicals were forming the group that with above 7 phytotoxicity rating whilst others were lower than 5. In high concentration rate, all treatments were above 4 phytotoxicity rating. The effects of severe and complete destruction were highly consistent in both rates (refer diagram 1 and 2).

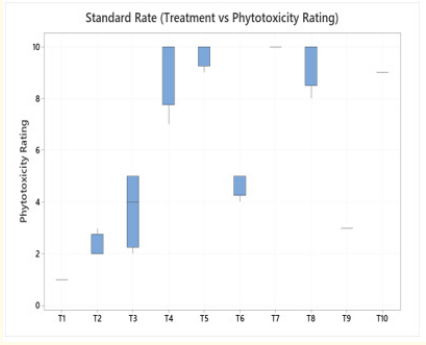

Diagram 1: Box plot of standard rate herbicide application. Treatment vs Phytotoxicity (60DAS) rating.

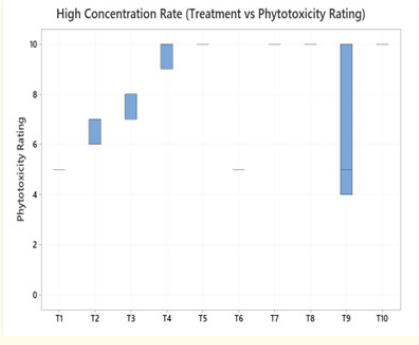

Diagram 2: Box plot of high concentration rate herbicide application. Treatment vs Phytotoxicity (60DAS) rating.

Conclusion

The screened herbicides within the same mode of action exert similar phytotoxicity symptoms. The specific action mechanism caused phytotoxicity symptoms either to new shoots, meristem tissue or leaves defoliation. There were two significant phytotoxicity effect groups. 1. Severe to complete destruction - 2,4-dimethylammonium 60%, fluroxypyr-meptyl 45.5%, indaziflam 45.5%, triclopyr-butotyl 32.1% and glufosinate ammonium 24.5%. 2. Slight to moderate - glyphosate isopropylamine 41%, MSMA 39.5% + diuron 7.8%, diuron 40% + ametryn 40%, metsulfuron-methyl 20% and diuron 80%. Phytotoxicity symptoms can be used to identify the causal chemical at least to the possible mode of action level. Understanding the effect can help to minimize phytotoxicity and determine safe chemical usage. Use of hormonal and cellulose biosynthesis inhibitor mode of action close to seedlings should be avoided in nursery and during field planting.

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