Lipotoxicity and induced suppression of fungi associated with decay of melon seeds in Nigeria

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Received on: 23/03/2021 Accepted on: 23/06/2021 Published on: 26/06/2021

ABSTRACT

Aim: The study was aimed to eradicate fungal contaminants in edible melon seeds by the use of lipids (a simplistic human and eco-friendly approach) to inhibit microbial growth.

Materials and Methods: Nine (9) identified fungal pathogens of edible melon seeds were induced with Lipotoxins using Coconut oil, Palm oil and Peanut oil in an in-vitro experiment. The Lipotoxicity test conducted showed that all the fungal pathogens were susceptible at varying degrees to the presence of saturated and unsaturated lipids extant at different levels in the test oils.

Results: Aspergillus niger, Cladosporium spp, Penicillium spp, A. flavus, Mucor spp, Absidia corymbifera and Rhizopus oryzae were totally (100%) eliminated i.e. 0.00cm mycelia diameter produced in the in-vitro culture. Seven (7) out of nine (9) pathogens used for this experiment were killed and two (2) were biologically deactivated by the lipids.

Conclusion: It was concluded that use of lipids in the pre-treatment of melon seeds before storage ensure the availability of disease free melon seeds for human consumption, eradicate mycotoxin contamination leading to food poisoning and ensure safety of life.

Keywords: Edible melon seeds; Food poisoning; Lipotoxic treatment; Mycotoxin contamination; Postharvest deterioration.

How to cite this article: Etaware PM (2021). Lipotoxicity and induced suppression of fungi associated with decay of melon seeds in Nigeria. J. Agri. Res. Adv., 03(02): 09-16.

Introduction

Colocynthis citrullus L. (Edible melon) seeds are one of the few natural sources that are very rich in protein (34.86%), essential oils (42.29%), vital Sodium (Na=162.76ppm), minerals like Potassium (K=8.28%), Calcium (Ca=1.49%) (Oyedele *et al.*, 2018), Iron (Fe=39.71pph), Copper (Cu=3.37ppm)and Zinc (Zn=13.46ppm)(Abiodun and Adeleke, 2010). Edible melon seeds also contain starch (11%), soluble sugars (2.50%), crude fibre and ash (12%) which is very essential in human diets. One major factor responsible for decreased shelf-life, depleted nutrient, reduced seed viability and unavailability of melon seeds all year round is postharvest deterioration of stored melon seeds by fungal pathogens (Oke et al., 2009).

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Although, improper storage conditions also play an important role in the deterioration of melon seeds while in storage, Donli and Gulani (2001) initially reported that fungi are the major causes of grain and seed spoilage in local store houses amongst other implicated spoilage organisms especially insects and rodents.

During harvest, melon gourds are sometimes partially broken and kept in the soil to ferment (post-harvest ripening) for few weeks, a process that signals multiple entry of infectious pathogens resulting in spoilage while in storage (Chiejina, 2006). After processing (seed wash, sun-drying and winnowing to remove shaft) and storage of the melon seeds, the pathogens introduced during postharvest ripening are rendered dormant (but still viable) for a long period of time if the seeds are kept in good storage conditions until the next planting season (Chiejina, 2006). The effects of these pathogens are translated in the earlier stages by the failure of the sown infected melons seeds to germinate or at the later stages by infecting germinated seedlings or at worst cause diseases in the

matured melon plants while in the field (Chiejina, 2006). Most storage fungi are also known to produce a large number of metabolites in seeds, some of which are toxic to humans, as a result of their metabolic activities in infected crop species under favourable conditions for fungal growth (Othman and Al-Delamiy, 2012).

Aflatoxins, one of the most potent mycotoxins produced by several species of Aspergillus (Most common storage fungi) are carcinogenic, hepatotoxic, deleterious to foetal growth and development, and are immunedepressant (Wild, 2007). Aflatoxins are not easily denatured by heat or high temperature, and are quite stable in many food processes, it is resistant to degradation and are not destroyed under normal cooking conditions (Obani et al., 2019). A more productive approach to minimize fungal occurrence on stored melon seeds is the use of lipid based substances (Lipotoxins) to inhibit the growth and metabolic activities of these noxious pathogens in stored melon seeds (Lipotoxic treatment of melon seeds), in order to ameliorate the problems of postharvest infection and spoilage of stored melon seeds and further strengthen the course for food security.

Materials and Methods

Source of fungal pathogens

The fungal pathogens of stored melon seeds used for this experiment were obtained from the stock culture of Oke *et al.* (2009), Botany Laboratory, Department of Botany, Faculty of Science, Lagos State University, Ojo, Lagos State, Nigeria.

Media used

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The following media were prepared according to the manufacturer's prescription (Industrial media)/Standard Laboratory protocols (Furbished Laboratory Media) using standard laboratory equipment and procedure. One litre of each media was prepared accordingly and sterilized at 121°C for 15 minutes in a stainless steel bucket autoclave.

Malt Extract Agar (MEA) Composition: A standard laboratory format was used in the formulation of MEA used in this experiment: Clarified Malt Extract (20g), Dextrose (20g), Peptone (6g), Agar (15g) and Chloramphenicol (0.1g).

Potato Dextrose Agar (PDA): Composition: The current composition of PDA used for this research was formulated based on the manufacturer's specification i.e. PDA powder

(39g) and Streptomycin (500mg) for one litre of pure culture media.

Carrot Agar (CA): Composition: Standard laboratory protocol for media formulation was the basic guide to the formulation of the desired Carrot Agar medium i.e. Clean and healthy carrot fruit (250g), Agar (20g) and Streptomycin (500mg).

Corn Meal Agar (CMA): Composition: A standard laboratory format was used in the formulation of CMA used in this experiment. The ingredients used were locally prepared i.e. Corn Meal (20g), Agar (20g) and Streptomycin (500mg).

Lipid Source

The lipids (oils) used for the Lipotoxicity test were extant in palm oil, coconut oil and peanut oil at varying degree as shown in Fig 1.

Lipotoxic Induction of melon seeds

The identified fungal pathogens of stored melon seeds [Oke et al., 2009] were each inoculated into Coconut oil, Palm oil and Peanut oil suspended in freshly prepared MEA, CA, CMA, and PDA media using standard laboratory procedure. Ten (10) millilitres of each sterilized oils was aseptically added to 90ml of freshly prepared medium culture (100% full strength). Homogenization was done in a water bath at 70°C for 10mins. At 37°C, 15ml of each lipophilic medium was aseptically dispensed disposable Petri dishes in replicates. The extrapolated fungal pathogens of stored melon [Oke et al., 2009] were inoculated on the culture media using a sterile 5mm diameter Cole Parmer cork-borer. Control experiments were setup using culture media placebos. The experiment was kept in a digital temperature calibrated incubator at 25 ± 2°C for 7days. Observations were made on daily basis.

Results and Discussion

Aspergillus niger was 100% Lipotoxic at the beginning of the experiment with 0.00cm mycelia diameter produced in the in-vitro treatment with peanut oil combined with CMA compared to the control setup for this experiment [Radial mycelia growth of *A. niger* on CMA only was 2.28cm at day 1] (Table 1). Also, at the end of the experiment, A. niger was highly susceptible to the lipid contents of coconut oil added to CA (1.95cm) and CMA (3.08cm), palm oil suspended in full strength CA (6.68cm), PDA (4.48cm) and CMA (4.75cm), and peanut oil combined with CA

(4.35cm) and PDA (3.90cm), respectively [Radial mycelia growth of *A. niger* on CMA only was 8.50cm, CA only (8.50cm), and PDA only (8.50cm) at day 7 of the Lipotoxicity test].

The growth of Cladosporium spp was totally inhibited by all the oils used for the experiment with 0% production of mycelia mass (0.00cm radial mycelia growth on the treatment medium) at day 1 of the lipid inhibition experiment (Table 2). At the end of the experiment, all the oils used as treatment significantly reduced the growth and mycelia production of Cladosporium spp (P<0.05) cultured in MEA (Coconut oil [4.38cm], Palm oil [5.73cm], and Peanut oil [5.05cm], respectively), CA (Palm oil [1.88cm]), PDA (Coconut oil [3.48cm], Palm oil [5.08cm], and Peanut oil [4.45cm], respectively), and CMA (Coconut oil [3.50cm], Palm oil [5.23cm], and Peanut oil [6.00cm], respectively) compared to the blanks used as control for this experiment [Radial mycelia growth of Cladosporium spp on MEA, PDA and CMA only was 8.50cm, while CA only gave 8.30cm at day 7 of the Lipotoxicity

The growth of *Penicillium* spp was also totally inhibited by all the oils used as treatment with production of 0% fungi mycelia growth at the beginning of the lipid inhibition experiment (Table 3). At the end of the experiment, Palm oil and Coconut oil suspended in PDA and CMA respectively were able to produce 100% inhibition of *Penicillium* spp in the in-vitro analysis conducted [0.00cm fungi mycelia growth for *Penicillium* spp] compared to the blanks used as control for this experiment [Radial mycelia growth of *Penicillium* spp on PDA and CMA only was 7.15cm and 6.40cm, respectively] at day 7 of the Lipotoxicity test (Table 3).

The growth of Aspergillus flavus was 100% inhibited by all the oils used for the Lipotoxicity test i.e. 0% mycelia mass was produced at the beginning of the lipid inhibition experiment (Table 4). Absolute control of the pathogen (Aspergillus flavus) was achieved only by the use of coconut oil suspended on CMA i.e. 0.00cm fungi mycelia mass produced at the end of the experiment too, compared to the control setup for this research [Mycelia diameter of Aspergillus flavus on CMA only was 7.50cm] (Table 4).

Mucor spp had a high level of Lipotoxins (100% treatment effect) compared to some of the test pathogens (Table 5). The microbial activities and growth of the test fungi "Mucor spp" was

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100% inhibited by peanut oil and coconut oil dissolved in PDA and CMA, respectively from the beginning (Day 1) of the experiment to the end (Day 7) with 0% (0.00cm) production of fungi mycelia mass (Table 5) compared to the control setup (P<0.05) for this experiment [Mycelia diameter of *Mucor* spp on CMA only was 1.28cm at Day 1 and 8.50cm at Day 7, while its mycelia diameter on PDA only was 1.70cm at Day 1 and 8.50cm at Day 7, respectively] (Table 5).

All the oils used for this experiment were totally lethal to the survival of Aspergillus fumigatus from the onset till the termination of the research as shown in the result obtained from the Lipotoxicity test (Table 6). A. fumigatus had the highest level of Lipotoxins (100% treatment effect) compared to other test pathogens with 83.33% [0.00cm radial mycelia growth on MEA (Coconut oil, Palm oil and Peanut oil), CA (Coconut oil and Palm oil), PDA (Coconut oil, Palm oil and Peanut oil), and CMA (Coconut oil and Palm oil), respectively] of the Lipotoxic treatment combinations causing total eradication of the pathogen (0% mycelia produced in-vitro). The remaining 16.67% of the treatment combination was able to minimize the microbial activities and growth of the test fungi below epidemic level (2.90cm and 3.60cm, respectively on peanut oil) compared to the control setup (P<0.05) for this experiment. Mycelia diameter of Aspergillus fumigatus on all the blanks was 6.95cm (MEA), 6.75cm (CA), 7.55cm (PDA) and 7.35cm (CMA), respectively at Day 7 (Table 6).

Absidia corymbifera also had a high level of Lipotoxic reaction to the lipid treatment used for this experiment (100% eradication of mycelia growth) compared to some of the test pathogens (Table 7). The microbial activities and growth of the test fungi "Absidia corymbifera" was totally (100%) inhibited by all the oils used in separate media combinations [CA, PDA and CMA, respectively] from the beginning (Day 1) of the experiment to the end (Day 7) with 0% (0.00cm) production of fungi mycelia mass (Table 7) compared to the control setup (P<0.05) for this experiment. Mycelia diameter of Absidia corymbifera on CA only was 8.33cm, 8.31cm on PDA only and 6.89cm on CMA only, respectively, at Day 7 of the Lipotoxicity test (Table 7).

The growth of *Curvularia* spp was totally inhibited by all the oils used at the beginning of the Lipotoxic test (0% production of mycelia mass on the treatment media) at day 1 (Table 8).

Table 1: Growth reactions of Aspergillus niger to Lipotoxic treatment

		Diameter Measurement of Aspergillus niger (cm)							
Media	Lipid Source	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	
Malt Extract	Coconut Oil	1.43±0.18 ^{de}	6.30±0.42ab	6.40±0.42b	7.88±0.04a	8.30±0.14a	8.30±0.14a	8.30±0.14a	
	Palm Oil	$1.30-\pm0.07^{de}$	5.40 ± 0.85 b	8.03±0.39a	8.10±0.49a	8.18±0.60a	8.18±0.60a	8.18±0.60a	
Agar [MEA]	Peanut Oil	1.08 ± 0.46^{e}	7.05 ± 0.07^{a}	8.20±1.13a	8.30±0.99a	8.40±0.85a	8.40 ± 0.85^{a}	8.40±0.85a	
Cannot A con	Coconut Oil	1.40 ± 0.28 de	1.68 ± 0.32^{de}	1.68 ± 0.32^{f}	1.68 ± 0.32^{d}	1.68±0.32e	1.83±0.39e	1.95±0.42e	
Carrot Agar	Palm Oil	2.23±0.53b	2.53±0.60d	3.40 ± 0.28^{de}	5.23±1.03b	6.15±0.49b	6.60 ± 0.07^{b}	6.68±0.18b	
[CA]	Peanut Oil	1.50±0.00 ^{c-e}	2.55 ± 0.07^{d}	3.60±0.42 ^{c-e}	3.90±0.71 ^c	4.13±0.67 ^c	4.35±0.35c	4.35±0.35 ^{cd}	
Potato Dextrose	Coconut Oil	1.83±0.11b-d	4.00±0.49c	4.65±0.64c	6.40±1.41b	7.35±1.63ab	7.35 ± 1.63 ab	7.58±1.31ab	
	Palm Oil	1.38 ± 0.18 de	1.70 ± 0.21^{de}	1.70 ± 0.21^{f}	2.50±0.00cd	3.35 ± 0.49 ^{cd}	4.08 ± 0.04 ^{cd}	4.48 ± 0.04 ^{cd}	
Agar [PDA]	Peanut Oil	1.50±0.14 ^{c-e}	2.20±0.92d	2.55±0.92ef	2.73±0.88cd	3.05±1.34 ^{c-e}	3.38 ± 1.38 ^{cd}	3.90 ± 1.56^{cd}	
Corn Meal	Coconut Oil	1.05 ± 0.07^{e}	1.05 ± 0.07^{e}	$1.60\pm0.14^{\rm f}$	2.10±0.35d	2.48 ± 0.32^{de}	2.65±0.42 ^{de}	3.08 ± 0.39^{de}	
	Palm Oil	2.03±0.32bc	2.18 ± 0.46^{d}	$2.65\pm0.64^{\rm ef}$	3.73±0.53 ^c	4.25±0.64 ^c	4.45±0.35c	4.75±0.35c	
Agar [CMA]	Peanut Oil	0.00 ± 0.00^{f}	1.70 ± 0.21^{de}	4.40 ± 0.57 ^{cd}	6.03±0.04b	7.00 ± 0.42^{ab}	7.48 ± 0.81 ab	7.68 ± 0.53 ab	
MEA	No Oil	4.22±0.09a	7.15±0.35a	8.50±0.00a	8.50±0.00a	8.50±0.00a	8.50±0.00a	8.50±0.00a	
CA	No Oil	2.33±0.11b	4.35±0.21c	6.00 ± 0.14 ^b	6.40 ± 0.14 ^b	7.50 ± 0.28 ab	8.30±0.14a	8.30±0.14a	
PDA	No Oil	3.75±0.21a	6.20 ± 0.28^{ab}	8.50±0.00a	8.50±0.00a	8.50±0.00a	8.50±0.00a	8.50±0.00a	
CMA	No Oil	2.38±0.32 ^b	6.20±0.57ab	8.10±0.42a	8.25±0.35a	8.40±0.14a	8.50±0.00a	8.50±0.00a	

Means with the same alphabets down the COLUMN are not significantly different at P<0.05 using Duncan Multiple Range Test (DMRT) for separation of statistically significant means. Data collected were represented as "Means ± SD" only

 Table 2: Growth reactions of Cladosporium spp to Lipotoxic treatment

		•	Diameter Measurement of Cladosporium spp (cm)							
Media	Lipid Source	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7		
Malt Extract	Coconut Oil	1.10±0.21bc	3.03±0.39a	3.38±0.39b	3.38±0.39 ^{cd}	3.50±0.42 ^{d-f}	4.15±0.71 ^{ef}	4.38±0.60 ^{cd}		
	Palm Oil	1.05±0.21bc	2.90±0.14a	3.20 ± 0.00 bc	3.53 ± 0.32^{cd}	4.90±1.27 ^{cd}	5.30±1.20 ^{c-e}	5.73±1.31bc		
Agar [MEA]	Peanut Oil	0.85 ± 0.21^{cd}	3.13±0.04a	3.28±0.11b	3.28 ± 0.11^{cd}	$4.58\pm1.59^{c-e}$	4.88 ± 1.52^{de}	5.05 ± 1.48 b-d		
Camat A aan	Coconut Oil	0.00 ± 0.00^{f}	1.28±0.53d	2.25±0.64d	3.08±0.67c-e	4.70 ± 0.14^{cd}	$6.00\pm0.42^{b-d}$	6.90 ± 0.14^{ab}		
Carrot Agar	Palm Oil	0.55 ± 0.49^{de}	1.48 ± 0.04 ^{cd}	1.48 ± 0.04^{e}	$1.48 \pm 0.04^{\rm f}$	1.50 ± 0.00 g	$1.80\pm0.00g$	1.88 ± 0.04^{e}		
[CA]	Peanut Oil	0.75±0.21 ^{c-e}	2.20±0.21b	5.45±0.64a	8.28±0.18a	8.28±0.18a	8.28±0.18a	8.28±0.18a		
Potato Dextrose	Coconut Oil	0.00 ± 0.00^{f}	0.35 ± 0.07^{e}	1.15±0.21 ^{e-g}	$1.20\pm0.14^{\rm f}$	2.50 ± 0.57^{fg}	2.85 ± 0.35^{fg}	3.48 ± 0.81^{de}		
	Palm Oil	0.00 ± 0.00^{f}	0.50±0.21e	0.50 ± 0.21 g	2.85 ± 0.28^{de}	$4.50 \pm 0.28^{c-e}$	$4.85 \pm 0.64^{\mathrm{de}}$	5.08 ± 0.67 ^{b-d}		
Agar [PDA]	Peanut Oil	0.40 ± 0.14 ef	1.15±0.64d	2.45±0.64 ^{cd}	2.83±0.74 ^{de}	3.75±0.64 ^{d-f}	$4.20 \pm 0.57 ef$	4.45 ± 0.57 ^{cd}		
Corn Meal	Coconut Oil	$0.00\pm0.00^{\rm f}$	$0.00\pm0.00^{\rm e}$	1.30 ± 0.42^{ef}	$2.03\pm0.74^{\rm ef}$	$2.68 \pm 1.45 ^{e-g}$	$2.78\pm1.31^{\rm fg}$	3.50 ± 1.77^{de}		
	Palm Oil	$0.00\pm0.00^{\rm f}$	0.58 ± 0.04^{e}	$0.58 \pm 0.04 \mathrm{fg}$	$1.65 \pm 0.57^{\rm f}$	$4.50\pm0.00^{c-e}$	4.90 ± 0.21^{de}	5.23±0.46b-d		
Agar [CMA]	Peanut Oil	$0.00\pm0.00^{\rm f}$	0.33±0.11e	3.25±0.07b	4.13 ± 0.74^{bc}	5.03 ± 1.38^{cd}	5.40±1.27 ^{c-e}	6.00±1.13bc		
MEA	No Oil	1.52±0.14a	2.32±0.14b	3.12 ± 0.14^{bc}	4.80±0.42b	6.00 ± 0.42^{bc}	8.25±0.21a	8.50±0.00a		
CA	No Oil	1.08 ± 0.14^{bc}	1.88 ± 0.14 bc	2.68±0.14 ^{b-d}	3.83±0.11 ^{b-d}	4.13±0.11 ^{c-f}	7.20 ± 0.42^{ab}	8.30±0.14a		
PDA	No Oil	1.30 ± 0.14 ab	2.10±0.14b	$2.90 \pm 0.14^{\text{b-d}}$	4.80±0.85b	7.20 ± 0.85 ab	8.20±0.28a	8.50±0.00a		
CMA	No Oil	1.10 ± 0.14^{bc}	1.90 ± 0.14 bc	$2.70\pm0.14^{b-d}$	3.50 ± 0.28^{cd}	4.30±0.28c-f	6.95±0.21a-c	8.50±0.00a		

Means with the same alphabets down the COLUMN are not significantly different at P<0.05 using Duncan Multiple Range Test (DMRT) for separation of statistically significant means. Data collected were represented as "Means ± SD" only

Table 3: Growth reactions of Penicillium spp to Lipotoxic treatment

			Diameter Measurement of Penicillium spp (cm)							
Media	Lipid Source	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7		
Malt Extract	Coconut Oil	0.73±0.25bc	4.03±0.67a	5.10±0.14 ^b	5.10 ± 0.14^{cd}	5.10±0.14 ^{b-d}	5.43±0.11 ^{cd}	5.50±0.00c		
	Palm Oil	1.03±0.25bc	2.38±0.04 ^{cd}	7.80±0.71a	8.15±0.21a	8.15±0.21a	8.15±0.21ab	8.15±0.21ab		
Agar [MEA]	Peanut Oil	0.75 ± 0.14^{bc}	2.70±0.28bc	3.35±0.78 ^{cd}	3.88 ± 1.17^{de}	4.55±1.77 ^{c-e}	5.10±1.84 ^{c-e}	5.78±2.16bc		
Camat A ann	Coconut Oil	0.88 ± 0.32^{bc}	2.50±0.28 ^{cd}	4.35±0.35bc	4.80 ± 0.57^{cd}	5.20±0.57 ^{b-d}	5.83±0.67 ^{b-d}	6.25±0.71a-c		
Carrot Agar	Palm Oil	0.00 ± 0.00^{d}	$0.70\pm0.00^{\mathrm{fg}}$	$0.70\pm0.00^{\mathrm{fg}}$	$0.70\pm0.00^{\rm f}$	$0.70\pm0.00^{\rm fg}$	$1.30\pm0.00^{\rm fg}$	2.23±0.18ef		
[CA]	Peanut Oil	0.00 ± 0.00^{d}	$1.48 \pm 0.11^{\rm ef}$	3.05±1.34d	3.90 ± 1.48^{de}	5.70±1.27 ^{b-d}	6.90±1.56a-d	6.93±1.52a-c		
Potato	Coconut Oil	0.00 ± 0.00^{d}	1.75 ± 0.28^{de}	2.45 ± 0.49^{de}	2.50±0.57e	2.60±0.71ef	2.93±0.81ef	3.05 ± 0.78^{de}		
Dextrose	Palm Oil	0.00 ± 0.00^{d}	$0.00\pm0.00g$	$0.00\pm0.00g$	0.00 ± 0.00^{f}	$0.00\pm0.00g$	$0.00\pm0.00g$	0.00 ± 0.00^{f}		
Agar [PDA]	Peanut Oil	0.00 ± 0.00^{d}	$0.78 \pm 0.67^{\mathrm{fg}}$	$0.78 \pm 0.67^{\rm fg}$	$0.78 \pm 0.67^{\rm f}$	$0.85 \pm 0.64^{\mathrm{fg}}$	$1.18{\pm}0.74^{\mathrm{fg}}$	1.33 ± 0.88^{ef}		
Corn Meal	Coconut Oil	0.00 ± 0.00^{d}	$0.00\pm0.00g$	$0.00\pm0.00g$	0.00 ± 0.00^{f}	$0.00\pm0.00g$	$0.00\pm0.00g$	0.00 ± 0.00^{f}		
	Palm Oil	0.00 ± 0.00^{d}	$0.00\pm0.00g$	1.63 ± 0.60^{ef}	2.53±1.87e	3.45 ± 3.18^{de}	4.48 ± 2.86 de	5.08 ± 2.65 ^{cd}		
Agar [CMA]	Peanut Oil	0.00 ± 0.00^{d}	1.95±0.78 ^{c-e}	5.55±0.07b	5.93±0.25bc	6.40±0.42a-c	6.85 ± 0.64 a-d	7.05±0.35a-c		
MEA	No Oil	1.40±0.35a	3.40 ± 0.35^{ab}	5.40±0.35 ^b	7.40 ± 0.35^{ab}	7.40 ± 0.35^{ab}	7.40±0.35a-c	7.40±0.35a-c		
CA	No Oil	0.85 ± 0.14 bc	2.35±0.57 ^{cd}	5.55 ± 0.57^{b}	8.38±0.04a	8.38±0.04a	8.38±0.04a	8.38±0.04a		
PDA	No Oil	1.08 ± 0.04^{ab}	2.40 ± 0.35^{cd}	4.40 ± 0.35^{bc}	7.15 ± 0.57^{ab}	7.15 ± 0.57^{ab}	7.15±0.57a-c	7.15±0.57a-c		
CMA	No Oil	0.70 ± 0.07^{c}	$1.28 \pm 0.04 ^{\rm ef}$	3.40 ± 0.35^{cd}	6.40 ± 0.35 bc	6.40±0.35a-c	6.40 ± 0.35 a-d	6.40±0.35a-c		

Means with the same alphabets down the COLUMN are not significantly different at P<0.05 using Duncan Multiple Range Test (DMRT) for separation of statistically significant means. Data collected were represented as "Means \pm SD" only

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Table 4: Growth response of Aspergillus flavus to Lipotoxic treatment

			I	Diameter Meas	surement of As	spergillus flavu	s (cm)	
Media	Lipid Source	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7
Malt Extract	Coconut Oil	0.68±0.32c	2.23±0.60bc	5.15±1.34ab	6.35±0.64a	7.55 ± 0.49 ab	7.70 ± 0.28 ab	7.75±0.21a-c
	Palm Oil	0.40 ± 0.00^{d}	2.20 ± 0.14 bc	5.00±0.28b	6.10 ± 0.64 ab	7.10±1.70a-c	7.10 ± 1.70^{ab}	7.10±1.70a-c
Carrot Agar [CA] Potato Dextrose Agar [PDA] Corn Meal	Peanut Oil	0.58 ± 0.04 ^{cd}	2.63±0.88a-c	6.05±0.07a	6.90±0.85a	8.25±0.07a	8.25±0.07a	8.25±0.07a
Commot A com	Coconut Oil	0.60 ± 0.00^{cd}	1.08 ± 0.11^{d}	1.08 ± 0.11^{cd}	1.20±0.28d	1.25 ± 0.35 ef	1.25±0.35d-f	1.25±0.35e-g
O	Palm Oil	$0.00\pm0.00^{\rm e}$	0.75±0.21 ^d	0.75±0.21 ^{c-e}	1.08 ± 0.60^{d}	1.48±1.03e	2.08 ± 0.95^{de}	2.55±0.64e
	Peanut Oil	1.58 ± 0.11^{b}	2.13±0.18 ^c	5.40 ± 0.57^{ab}	5.90±0.57ab	6.05±0.49 ^c	6.30±0.28b	6.35±0.21 ^c
Potato	Coconut Oil	0.63 ± 0.11^{cd}	0.98 ± 0.53 ^d	1.70±0.57c	1.70 ± 0.57 ^d	1.70±0.57e	1.70 ± 0.57 de	$1.70 \pm 0.57 ^{\rm ef}$
Dextrose	Palm Oil	$0.00\pm0.00^{\rm e}$	0.83±0.11 ^d	1.18 ± 0.04^{cd}	1.43 ± 0.18^{d}	1.80 ± 0.14^{e}	2.30±0.28d	2.63±0.25e
Agar [PDA]	Peanut Oil	0.00 ± 0.00^{e}	0.73±0.11 ^d	0.73 ± 0.11^{de}	0.73 ± 0.11^{de}	0.73 ± 0.11^{ef}	0.73 ± 0.11^{ef}	$0.73\pm0.11^{\rm fg}$
Com Most	Coconut Oil	0.00 ± 0.00^{e}	$0.00\pm0.00^{\rm e}$	$0.00\pm0.00^{\rm e}$	0.00 ± 0.00^{e}	0.00 ± 0.00^{f}	$0.00\pm0.00^{\mathrm{f}}$	$0.00\pm0.00g$
	Palm Oil	$0.00\pm0.00^{\rm e}$	0.75 ± 0.07^{d}	0.75±0.07 ^{c-e}	3.18 ± 0.74^{c}	6.13±0.81 ^c	6.68±1.03b	6.68±1.03bc
Agar [CMA]	Peanut Oil	$0.75\pm0.00^{\circ}$	1.15±0.07d	1.45 ± 0.07^{cd}	2.90±0.57c	3.28 ± 0.32^{d}	3.85±0.35c	4.80±0.99d
MEA	No Oil	1.92±0.14a	3.12±0.14a	5.12 ± 0.14^{ab}	5.92 ± 0.14 ab	6.72±0.14bc	7.52 ± 0.14 ab	7.92 ± 0.14^{ab}
CA	No Oil	1.48 ± 0.14 b	2.68±0.14a-c	4.68 ± 0.14^{b}	5.48 ± 0.14 ^b	6.28 ± 0.14 bc	7.08 ± 0.14 ab	7.48 ± 0.14 a-c
PDA	No Oil	1.70 ± 0.14^{ab}	2.90 ± 0.14^{ab}	4.90 ± 0.14^{b}	5.70 ± 0.14^{b}	6.50 ± 0.14 bc	7.30 ± 0.14^{ab}	7.70±0.14 ^{a-c}
CMA	No Oil	1.50 ± 0.14^{b}	2.70±0.14a-c	4.70 ± 0.14^{b}	5.50 ± 0.14^{b}	6.30 ± 0.14^{bc}	7.10 ± 0.14^{ab}	7.50±0.14a-c

Means with the same alphabets down the COLUMN are not significantly different at P<0.05 using Duncan Multiple Range Test (DMRT) for separation of statistically significant means. Data collected were represented as "Means ± SD" only

Table 5: Growth reactions of *Mucor* spp to Lipotoxic treatment

			Diameter Measurement of Mucor spp (cm)							
Media	Lipid Source	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7		
Malt Extract	Coconut Oil	$0.00\pm0.00^{\rm e}$	3.10±0.57ab	3.70±0.28c	3.70±0.28 ^{cd}	3.70±0.28c	3.70±0.28 ^{de}	3.70±0.28 ^{de}		
	Palm Oil	0.00 ± 0.00^{e}	2.98 ± 1.17^{ab}	4.13±0.32c	4.20±0.21c	4.33±0.04c	4.43±0.11 ^{cd}	4.43±0.11 ^{cd}		
Agar [MEA]	Peanut Oil	$0.00\pm0.00^{\rm e}$	2.43 ± 0.18^{ab}	3.35±0.35 ^{cd}	3.60 ± 0.42^{cd}	4.30±0.85c	5.35±1.34bc	5.58±1.52bc		
C 1 A	Coconut Oil	$0.00\pm0.00^{\rm e}$	$0.00\pm0.00^{\rm e}$	$0.00\pm0.00^{\mathrm{f}}$	0.00 ± 0.00^{f}	$0.00\pm0.00^{\rm e}$	0.00 ± 0.00^{h}	0.00 ± 0.00 g		
Carrot Agar [CA]	Palm Oil	$0.00\pm0.00^{\rm e}$	1.23±0.11 ^{cd}	1.33±0.25e	1.33±0.25e	1.33±0.25 ^d	1.33±0.25gh	$1.33\pm0.25^{\rm fg}$		
	Peanut Oil	1.10 ± 0.14^{d}	2.13±1.03bc	2.80±1.41 ^d	3.43±1.52 ^{cd}	3.80±1.84c	4.13±1.73 ^{c-e}	4.33±1.87 ^{cd}		
Potato	Coconut Oil	$0.00\pm0.00^{\rm e}$	$0.00\pm0.00^{\rm e}$	$0.00\pm0.00^{\mathrm{f}}$	0.00 ± 0.00^{f}	2.45±0.64 ^d	$3.15\pm0.78^{d-f}$	3.48 ± 0.74^{de}		
Dextrose	Palm Oil	$0.00\pm0.00^{\rm e}$	0.80 ± 0.28 ^{de}	1.83±0.04e	1.83±0.04e	1.83±0.04 ^d	$2.00\pm0.28^{\rm fg}$	2.20±0.57ef		
Agar [PDA]	Peanut Oil	$0.00\pm0.00^{\rm e}$	$0.00\pm0.00^{\rm e}$	$0.00\pm0.00^{\mathrm{f}}$	0.00 ± 0.00^{f}	$0.00\pm0.00^{\rm e}$	0.00 ± 0.00^{h}	0.00 ± 0.00 g		
Corn Meal	Coconut Oil	$0.00\pm0.00^{\rm e}$	$0.00\pm0.00^{\rm e}$	$0.00\pm0.00^{\mathrm{f}}$	0.00 ± 0.00^{f}	$0.00\pm0.00^{\rm e}$	0.00 ± 0.00^{h}	0.00 ± 0.00 g		
	Palm Oil	$0.00\pm0.00^{\rm e}$	0.93 ± 0.18^{de}	0.93 ± 0.18^{ef}	2.83±0.32 ^d	6.15±0.78 ^b	6.15±0.78 ^b	6.28±0.60b		
Agar [CMA]	Peanut Oil	$0.00\pm0.00^{\rm e}$	0.70 ± 0.28^{de}	1.35±0.35e	1.88±0.32e	2.35±0.07 ^d	2.75±0.21 ^{ef}	3.18 ± 0.25^{de}		
MEA	No Oil	1.85±0.09a	2.89 ± 0.09^{ab}	8.10±0.07a	8.50±0.00a	8.50 ± 0.00^{a}	8.50 ± 0.00^{a}	8.50±0.00a		
CA	No Oil	1.25±0.08c	3.23±0.39a	5.85±0.21b	6.40±0.14b	$7.50\pm0.28a$	8.30±0.14a	8.30±0.14a		
PDA	No Oil	1.70 ± 0.14 ^b	3.30±0.14a	8.50±0.00a	8.50±0.00a	8.50±0.00a	8.50 ± 0.00^{a}	8.50±0.00a		
CMA	No Oil	1.28±0.01c	2.63 ± 0.09 ab	6.60±0.14b	6.90±0.42b	8.40±0.14a	8.50 ± 0.00^{a}	8.50±0.00a		

Means with the same alphabets down the COLUMN are not significantly different at P<0.05 using Duncan Multiple Range Test (DMRT) for separation of statistically significant means. Data collected were represented as "Means \pm SD" only

Table 6: Growth reactions of Aspergillus fumigatus to Lipotoxic treatment

			Diameter Measurement of Aspergillus fumigatus (cm)							
Media	Lipid Source	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7		
Malt Extract	Coconut Oil	0.00 ± 0.00^{c}	0.00 ± 0.00^{d}	0.00 ± 0.00^{d}	0.00 ± 0.00^{c}	0.00 ± 0.00^{c}	0.00 ± 0.00^{c}	0.00 ± 0.00^{c}		
	Palm Oil	0.00 ± 0.00^{c}	0.00 ± 0.00^{d}	0.00 ± 0.00^{d}	0.00 ± 0.00^{c}	0.00 ± 0.00^{c}	0.00 ± 0.00^{c}	0.00 ± 0.00^{c}		
Agar [MEA]	Peanut Oil	0.00 ± 0.00^{c}	0.00 ± 0.00^{d}	0.00 ± 0.00^{d}	0.00 ± 0.00^{c}	0.00 ± 0.00^{c}	0.00 ± 0.00^{c}	0.00 ± 0.00^{c}		
Carrot Agar	Coconut Oil	0.00 ± 0.00^{c}	0.00 ± 0.00^{d}	0.00 ± 0.00^{d}	0.00 ± 0.00^{c}	0.00 ± 0.00^{c}	0.00 ± 0.00^{c}	0.00 ± 0.00^{c}		
O	Palm Oil	0.00 ± 0.00^{c}	0.00 ± 0.00^{d}	0.00 ± 0.00^{d}	0.00 ± 0.00^{c}	0.00 ± 0.00^{c}	0.00 ± 0.00^{c}	0.00 ± 0.00^{c}		
[CA]	Peanut Oil	0.00 ± 0.00^{c}	1.20±0.99c	1.95±1.20c	2.38±1.45b	2.78±1.94b	2.88±1.80b	2.90±1.77b		
Potato Dextrose	Coconut Oil	0.00 ± 0.00^{c}	0.00 ± 0.00^{d}	0.00 ± 0.00^{d}	0.00 ± 0.00^{c}	0.00 ± 0.00^{c}	0.00 ± 0.00^{c}	0.00 ± 0.00^{c}		
	Palm Oil	0.00 ± 0.00^{c}	0.00 ± 0.00^{d}	0.00 ± 0.00^{d}	0.00 ± 0.00^{c}	0.00 ± 0.00^{c}	0.00 ± 0.00^{c}	0.00 ± 0.00^{c}		
Agar [PDA]	Peanut Oil	0.00 ± 0.00^{c}	0.00 ± 0.00^{d}	0.00 ± 0.00^{d}	0.00 ± 0.00^{c}	0.00 ± 0.00^{c}	0.00 ± 0.00^{c}	0.00 ± 0.00^{c}		
Corn Meal	Coconut Oil	0.00 ± 0.00^{c}	0.00 ± 0.00^{d}	0.00 ± 0.00^{d}	0.00 ± 0.00^{c}	0.00 ± 0.00^{c}	0.00 ± 0.00^{c}	0.00 ± 0.00^{c}		
	Palm Oil	0.00 ± 0.00^{c}	0.00 ± 0.00^{d}	0.00 ± 0.00^{d}	0.00 ± 0.00^{c}	0.00 ± 0.00^{c}	0.00 ± 0.00^{c}	0.00 ± 0.00^{c}		
Agar [CMA]	Peanut Oil	0.00 ± 0.00^{c}	1.20±0.14c	2.30±0.14c	2.75±0.49b	3.10±0.71b	3.28 ± 0.81^{b}	3.60±0.71b		
MEA	No Oil	1.67±0.07a	3.95 ± 0.21^{ab}	4.55±0.21ab	5.75±0.21a	6.95±0.21a	6.95±0.21a	6.95±0.21a		
CA	No Oil	1.27 ± 0.12^{b}	3.75±0.21 ^b	4.35±0.21 ^b	5.55±0.21a	6.75±0.21a	6.75±0.21a	6.75±0.21a		
PDA	No Oil	1.55±0.21a	4.55±0.21a	5.15±0.21a	6.35±0.21a	7.55±0.21a	7.55±0.21a	7.55±0.21a		
CMA	No Oil	1.35±0.21 ^b	4.35±0.21ab	4.95±0.21ab	6.15±0.21a	7.35±0.21a	7.35±0.21a	7.35±0.21a		

Means with the same alphabets down the COLUMN are not significantly different at P<0.05 using Duncan Multiple Range Test (DMRT) for separation of statistically significant means. Data collected were represented as "Means \pm SD" only

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Vol 03 No 2, p 09-16/13

Table 7: Growth reactions of Absidia corymbifera to Lipotoxic treatment

			Diameter Measurement of Absidia corymbifera (cm) Day 2 Day 3 Day 4 Day 5 Day 6 Day 7							
Media	Lipid Source	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7		
Malt Extract	Coconut Oil	0.00 ± 0.00^{c}	1.80±0.14 ^d	2.05±0.21c	2.23±0.18d	2.50±0.28d	2.80±0.42d	3.10±0.85d		
	Palm Oil	0.00 ± 0.00^{c}	0.90 ± 0.00^{e}	0.93 ± 0.25 de	0.93±0.25e	0.93±0.25e	0.93±0.25e	0.93±0.25e		
Agar [MEA]	Peanut Oil	0.00 ± 0.00^{c}	2.68±0.18c	4.50±0.85b	4.50±0.85c	4.50±0.85c	4.50±0.85c	4.50±0.85c		
Commot A com	Coconut Oil	0.00 ± 0.00^{c}	0.00 ± 0.00^{f}	$0.00\pm0.00^{\rm e}$	0.00 ± 0.00^{f}	$0.00\pm0.00^{\rm e}$	$0.00\pm0.00^{\rm e}$	0.00 ± 0.00^{e}		
Carrot Agar	Palm Oil	0.00 ± 0.00^{c}	0.73 ± 0.18^{e}	0.73 ± 0.18^{de}	0.73 ± 0.18^{ef}	0.73 ± 0.18^{e}	0.73 ± 0.18^{e}	0.73 ± 0.18^{e}		
[CA]	Peanut Oil	0.75 ± 0.35^{b}	1.20 ± 0.78^{de}	1.50 ± 0.99^{cd}	1.98 ± 0.88^{d}	2.45±0.99d	2.85±1.34 ^d	3.45±1.20 ^d		
Potato Dextrose	Coconut Oil	0.00 ± 0.00^{c}	0.73±0.39e	0.73 ± 0.39^{de}	1.58 ± 0.46^{de}	3.30±0.71d	4.13±0.74c	5.33±0.11 ^c		
	Palm Oil	0.00 ± 0.00^{c}	0.73±0.11e	0.73 ± 0.11^{de}	0.73 ± 0.11^{ef}	0.73±0.11e	0.73±0.11e	0.73±0.11e		
Agar [PDA]	Peanut Oil	0.00 ± 0.00^{c}	0.00 ± 0.00^{f}	$0.00\pm0.00^{\rm e}$	0.00 ± 0.00^{f}	$0.00\pm0.00^{\rm e}$	$0.00\pm0.00^{\rm e}$	0.00 ± 0.00^{e}		
Corn Meal	Coconut Oil	0.00 ± 0.00^{c}	0.00 ± 0.00^{f}	$0.00\pm0.00^{\rm e}$	0.00 ± 0.00^{f}	0.00 ± 0.00^{e}	0.00 ± 0.00^{e}	0.00 ± 0.00^{e}		
	Palm Oil	0.00 ± 0.00^{c}	0.00 ± 0.00^{f}	$0.00\pm0.00^{\rm e}$	0.00 ± 0.00^{f}	0.00 ± 0.00^{e}	$0.00\pm0.00^{\rm e}$	0.00 ± 0.00^{e}		
Agar [CMA]	Peanut Oil	0.00 ± 0.00^{c}	0.00 ± 0.00^{f}	0.00 ± 0.00^{e}	0.00 ± 0.00^{f}	0.00 ± 0.00^{e}	0.00 ± 0.00^{e}	0.00 ± 0.00^{e}		
MEA	No Oil	2.35±0.42a	4.75±0.42a	7.15±0.42a	8.15±0.14a	8.48±0.04a	8.48±0.04a	8.48±0.04a		
CA	No Oil	2.19±0.37a	4.27±0.37b	6.35±0.37a	7.39±0.37a	8.33±0.23a	8.33±0.23a	8.33±0.23a		
PDA	No Oil	2.25±0.39a	4.45±0.39a	6.65±0.39a	7.75±0.39a	8.31±0.27a	8.31±0.27a	8.31±0.27a		
CMA	No Oil	2.09±0.28a	3.69±0.28b	5.29±0.28 ^b	6.09±0.28b	6.89±0.28 ^b	6.89±0.28b	6.89±0.28 ^b		

Means with the same alphabets down the COLUMN are not significantly different at P<0.05 using Duncan Multiple Range Test (DMRT) for separation of statistically significant means. Data collected were represented as "Means \pm SD" only

Table 8: Growth reactions of *Curvularia* spp to Lipotoxic treatment

			Diameter Measurement of Curvularia spp (cm)							
Media	Lipid Source	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7		
Malt Extract	Coconut Oil	0.00 ± 0.00^{b}	1.60±0.35d	1.60 ± 0.35^{g}	$1.60\pm0.35^{\rm fg}$	1.60±0.35g	1.60 ± 0.35 gh	1.60±0.35 ^d		
	Palm Oil	0.00 ± 0.00^{b}	1.35 ± 0.49^{de}	2.85±0.35e	4.78±0.67 ^c	7.05±1.77a-c	7.05 ± 1.77^{ab}	7.05 ± 1.77^{ab}		
Agar [MEA]	Peanut Oil	0.00 ± 0.00^{b}	2.83±0.67c	7.85±0.07a	7.85±0.07a	7.85±0.07a	7.85 ± 0.07^{a}	7.85±0.07a		
Carrot Agar	Coconut Oil	0.00 ± 0.00^{b}	0.00 ± 0.00^{f}	2.25±0.21 ^f	2.55±0.21 ^{de}	$3.10\pm0.14^{\rm ef}$	3.33±0.39ef	3.98±0.95c		
U	Palm Oil	0.00 ± 0.00^{b}	0.00 ± 0.00^{f}	0.00±0.00j	$0.93 \pm 0.46 \mathrm{gh}$	$1.60\pm0.07g$	$2.60 \pm 0.14^{\rm fg}$	3.25±0.42 ^c		
[CA]	Peanut Oil	0.00 ± 0.00^{b}	0.85 ± 0.28^{e}	3.78 ± 0.04^{cd}	5.20±0.00c	5.95±0.92 ^{cd}	5.95±0.92bc	6.33±0.39 ^b		
Potato Dextrose	Coconut Oil	0.00 ± 0.00^{b}	0.00 ± 0.00^{f}	0.00 ± 0.00^{j}	0.00 ± 0.00^{i}	0.00 ± 0.00^{h}	0.00 ± 0.00^{i}	$0.00\pm0.00^{\rm e}$		
	Palm Oil	0.00 ± 0.00^{b}	$1.05\pm0.00^{\rm f}$	1.20±0.00gh	2.13±0.53ef	4.35±0.78e	4.35 ± 0.78^{de}	4.35±0.78 ^c		
Agar [PDA]	Peanut Oil	0.00 ± 0.00^{b}	0.00 ± 0.00^{f}	0.40 ± 0.00^{ij}	0.75 ± 0.00^{h}	0.98 ± 0.04 gh	$0.98 \pm 0.04 ^{\rm hi}$	0.98 ± 0.04 de		
Corn Meal	Coconut Oil	0.00 ± 0.00^{b}	0.00 ± 0.00^{f}	0.73 ± 0.25^{hi}	1.45 ± 0.07 f-h	2.33 ± 0.39^{fg}	$2.90\pm0.85^{\rm fg}$	3.40 ± 0.64^{c}		
Agar [CMA]	Palm Oil	0.00 ± 0.00^{b}	0.00 ± 0.00^{f}	0.00±0.00j	1.13 ± 0.11^{gh}	3.53 ± 0.25 ef	3.83 ± 0.25 ef	4.03±0.46c		
Agai [CNA]	Peanut Oil	0.00 ± 0.00^{b}	0.00 ± 0.00^{f}	2.18±0.60 ^f	2.90±0.42d	5.63±0.74 ^d	5.65±0.78 ^{cd}	5.73±0.88 ^b		
MEA	No Oil	2.05±0.28a	3.65 ± 0.28 ab	5.25±0.28 ^b	6.85±0.28b	8.30±0.06a	8.50 ± 0.00^{a}	8.50±0.00a		
CA	No Oil	2.00±0.20a	3.12±0.20bc	4.24±0.20c	5.36±0.20 ^c	6.48 ± 0.20 b-d	8.30±0.14a	8.30±0.14a		
PDA	No Oil	2.00±0.33a	3.88 ± 0.33^{a}	5.76±0.33b	7.64±0.33a	7.65 ± 0.28 ab	8.50 ± 0.00^{a}	8.50±0.00a		
CMA	No Oil	1.82 ± 0.15^{a}	2.66±0.15c	3.50 ± 0.15^{d}	6.55±0.26b	8.03±0.26a	8.50 ± 0.00^{a}	8.50±0.00a		

Means with the same alphabets down the COLUMN are not significantly different at P<0.05 using Duncan Multiple Range Test (DMRT) for separation of statistically significant means. Data collected were represented as "Means \pm SD" only

Table 9: Growth reactions of *Rhizopus oryzae* to Lipotoxic treatment

	-		Diameter Measurement of Rhizopus oryzae (cm)								
Media	Lipid Source	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7			
Malt Extract	Coconut Oil	0.00 ± 0.00^{e}	3.05±0.21e	5.55±2.19b	6.70±1.13b	8.00±0.57a	8.00±0.57a	8.00±0.57a			
	Palm Oil	0.00 ± 0.00^{e}	0.00 ± 0.00^{i}	$0.00\pm0.00^{\rm e}$	$0.00\pm0.00^{\mathrm{f}}$	0.00 ± 0.00^{d}	0.00 ± 0.00^{d}	0.00 ± 0.00^{d}			
Agar [MEA]	Peanut Oil	0.00 ± 0.00^{e}	$1.53\pm0.04^{\rm f}$	2.40±0.42 ^{cd}	2.40±0.42d	2.40±0.42c	2.40±0.42 ^c	2.40±0.42 ^c			
Carrot Agar [CA]	Coconut Oil	0.00 ± 0.00^{e}	0.98 ± 0.11 g	3.30±0.42 ^c	4.68 ± 0.88^{c}	4.78 ± 1.03^{b}	5.23±1.31 ^b	5.58±1.31 ^b			
	Palm Oil	0.00 ± 0.00^{e}	0.00 ± 0.00^{i}	$0.00\pm0.00^{\rm e}$	$0.00\pm0.00^{\rm f}$	0.00 ± 0.00^{d}	0.00 ± 0.00^{d}	0.00 ± 0.00^{d}			
	Peanut Oil	0.00 ± 0.00^{e}	$0.98 \pm 0.04 \mathrm{g}$	8.43±0.04a	8.43±0.04a	8.43±0.04a	8.43±0.04a	8.43±0.04a			
Potato Dextrose	Coconut Oil	0.00 ± 0.00^{e}	0.50 ± 0.14^{h}	1.20 ± 0.57 de	1.30±0.49e	1.58±0.60c	1.78±0.32 ^c	1.80±0.35c			
	Palm Oil	0.00 ± 0.00^{e}	0.00 ± 0.00^{i}	$0.00\pm0.00^{\rm e}$	$0.00\pm0.00^{\mathrm{f}}$	0.00 ± 0.00^{d}	0.00 ± 0.00^{d}	0.00 ± 0.00^{d}			
Agar [PDA]	Peanut Oil	0.00 ± 0.00^{e}	0.00 ± 0.00^{i}	$0.00\pm0.00^{\rm e}$	$0.00\pm0.00^{\mathrm{f}}$	0.00 ± 0.00^{d}	0.00 ± 0.00^{d}	0.00 ± 0.00^{d}			
Corn Meal	Coconut Oil	0.00 ± 0.00^{e}	0.88 ± 0.32^{g}	1.18 ± 0.67^{de}	1.18 ± 0.67^{e}	1.68 ± 0.88^{c}	1.98±1.10 ^c	2.18±0.95 ^c			
	Palm Oil	0.00 ± 0.00^{e}	0.00 ± 0.00^{i}	$0.00\pm0.00^{\rm e}$	$0.00\pm0.00^{\mathrm{f}}$	0.00 ± 0.00^{d}	0.00 ± 0.00^{d}	0.00 ± 0.00^{d}			
Agar [CMA]	Peanut Oil	0.00 ± 0.00^{e}	0.95 ± 0.14 g	3.45±0.21c	4.13±0.39c	4.23±0.46b	4.63±0.60b	4.80±0.35b			
MEA	No Oil	4.45±0.28b	6.05±0.28b	8.50±0.00a	8.50±0.00a	8.50 ± 0.00^{a}	8.50 ± 0.00^{a}	8.50 ± 0.00^{a}			
CA	No Oil	3.68±0.20c	4.80±0.20c	6.00±0.14b	8.50±0.00a	8.50 ± 0.00^{a}	8.50 ± 0.00^{a}	8.50 ± 0.00^{a}			
PDA	No Oil	4.82±0.33a	6.70±0.33a	8.50±0.00a	8.50±0.00a	8.50 ± 0.00^{a}	8.50±0.00a	8.50 ± 0.00^{a}			
CMA	No Oil	3.08 ± 0.15^{d}	3.92 ± 0.15^{d}	8.10±0.42a	8.50±0.00a	8.50±0.00a	8.50±0.00a	8.50±0.00a			

Means with the same alphabets down the COLUMN are not significantly different at P<0.05 using Duncan Multiple Range Test (DMRT) for separation of statistically significant means. Data collected were represented as "Means \pm SD" only

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Vol 03 No 2, p 09-16/14

At the end of the experiment, all the oils used as treatment had significant effects in the reduction of the growth and mycelia production of *Curvularia* spp (P<0.05) but coconut oil suspended in PDA was the most effective for this pathogen, producing 100% mycelia inhibition at the end of the Lipotoxicity test compared to the blanks used as control for this experiment [Radial mycelia growth of *Curvularia* spp on all the substrate media was 8.50cm] at day 7 (Table 8).

The Lipotoxic response of *Rhizopus oryzae* to lipids from different oil sources was very high after treatment (100% eradication of mycelia growth) at the beginning of the research compared to some of the test pathogens (P<0.05). The microbial activities and growth of Rhizopus oryzae was totally (100%) inhibited by lipid(s) from Palm oil suspended in MEA (0.00cm), CA (0.00cm), PDA (0.00cm) and CMA (0.00cm) from the beginning (Day 1) of the experiment to the end i.e. Day 7 (Table 9) compared to the control setup (P<0.05) for this experiment. Mycelia diameter of Rhizopus oryzae on MEA, CA, PDA and CMA only was 8.50cm, respectively for each] at Day 7 of the Lipotoxicity test. Also, peanut oil dissolved in PDA gave the same impressive result as palm oil (Table 9).

The Lipotoxicity test conducted showed that all the fungal pathogens were susceptible at varying degrees to the presence of saturated and unsaturated lipids extant at different levels in the test oil sources. This was in agreement with the research of Wise et al. [Wise et al., 2014] who came to the conclusion that there was a strong correlation between the sensitivity microorganisms (especially fungal pathogens) to the presence of lipids or lipid based drugs used as biocontrol agents. Also, Avis and Belanger [Avis and Bélanger, 2001] found out that the fatty acid composition and degree of unsaturation in some polar lipids can alter membrane fluidity as an adaptive response to temperature fluctuations or other stressors, which could be the major reason for the success of the Lipotoxicity test conducted.

Coconut oil was the most effective lipid source for the treatment of fungal pathogens from stored melon seeds as it totally inhibited the growth of six (6) out of the nine (9) pathogens treated. Palm oil and Peanut oil totally inhibited the microbial activities of four (4) out of the (9) fungal pathogens treated. The rationale behind this observation was given by Wise *et al.* (2014)

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who noted that palmitic, oleic, and linoleic acids present in varying levels in most polar lipids can antagonize fungal pathogens. Also, he noted that there was lack of a dose-dependent response in the inhibitory activities of lipids or lipid based drugs used as treatment of most plant based fungal pathogens.

Seven (7) out of nine (9) pathogens used for this experiment were killed by the lipids from the oil sources during the Lipotoxicity test. Although, 83.33% success rate was recorded [Seven (7) killed and two (2) biologically deactivated fungal strains], there is need to ascertain why Aspergillus niger and Cladosporium species could not be totally eradicated by these treatments. This could be due to their resilience to lipids or lipid based substances. This was explained by Avis and Belanger (2001), Eeman et al. (2009). They stated that although sterols (cholesterol in particular) may fluidize highly rigid membranes, lipids present in less rigid and more aqueous membranes such as those of actively growing fungal cells would have positive effects on the treated pathogen, which may further explain the tolerance of such pathogen to treatment. Therefore, microorganisms containing little or no sterols would be more susceptible to lipidmembrane interactions, leading to a decrease in viability, whereas those with a larger proportion of sterols would be more capable of buffering stress induced changes.

Conclusion

It was concluded that the fungal pathogens extrapolated from stored melon seed samples within local markets in Lagos State, Nigeria were susceptible at varying degrees to the presence of saturated and unsaturated lipids extant at different levels in the test oil sources. Seven (7) out of nine (9) pathogens used for this experiment were killed and two (2) were biologically deactivated by the lipids during the Lipotoxicity test. Therefore, the use of lipids in the pre-treatment of melon seeds before storage can help limit postharvest deterioration, improve shelf life, eradicate toxin contamination, sustain important melon seed nutrient and increase seed viability of the stored melon seeds in other to ensure availability of disease free melon seeds for human consumption and safeguard life.

Acknowledgement

Author would like to acknowledge the efforts of Chief John O. and Mrs. Esther Etaware for their moral and financial support towards the completion of this research.

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