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Assessment of 3-Monochloro 1, 2-Propanediol in some Egyptian Foods and Its Impact on Liver and Renal Toxicity in Rats

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ABSTRACT

This study reports the results of the survey study on 3-monochloro1,2-propanediol (3-MCPD) levels in selected foods, oils and fats collected from supermarkets and restaurants in Egypt. Also; study the effect of feeding of rat on diet contained palm oil rich 3-MCPD on the biochemical parameters of rats. Twenty samples were collected from different supermarkets and restaurants in Egypt to determine 3-MCPD by GC-MS. The oil samples remained after from falafel frying showed the highest contents of 3-MCPD esters, with a mean value of 8.20 µg/kg oil, followed by palm oils and oils mixture. Forty Albino rats divided into five groups including one as a basal diet control. The other groups were fed on a a basal diet and replaced corn oil with crude palm oil heated at 200°C for 30min, heated palm oil at 200°C for 6h and palm oil fried with tortilla chips. Rats fed on either heated palm oil at 200°C for 6h and fried palm oil with tortilla chips showed elevated liver and kidney functions as well as lipid profile except HDL-cholesterol and damages in some histological liver and kidney tissue compared with the control basal diet.This would risk forming esters of 3-MCPD fatty acids.

Keywords: 3-MCPD, frying, palm oil, liver and kidney functions

INTRODUCTION

Chloro-propanols are important for high temperature processing of oils and fats.They are found in many foods ,whether free or bound (Collier *et al*., 1991). Primary chloropropanols 1,3-DCP (1,3Dichloro-2-propanol) and 3-MCPD (3-monochloro1,2-propanediol) exist in many foods; and the latter exists in foods with edible oils (especially the refined oils) at higher concentrations than the former and forms the main source of contaminants in thermally treated foods (Svejkovska,*et al*., 2004). Many 3-MCPD are in oils of different and same kinds particularly at elevated levels Weißhaar (2010).

3-MCPD esters and fatty acid esters exist in vegetable oils and formed during processing with structurally related and toxic chemicals as glycidyl esters and 2-monochloro-1, 3-propanol esters (2-MCPD esters). No index of the inverse effects of dietary exposure to these esters is available and it is difficult to determine their importance of human health. An indirect concern has been raised by the likely release of free chloropropanols (e.g., 3-MCPD, 2-MCPD) and glycidol resulting from lipase action that can hydrolyse esters in the gut (Schilter *et al*. 2011).

Toxic compounds such as chloropropanol nitrosamines and furans may be formed during food processing. Oxidation in frying oil are inhibited by proteins, starch or phenolic compounds. Mutagenic polycyclic aromatic heterocycles are done frying fats and proteins (Aznar *et al*. 2013).

Chloropropanols including genotoxics and carcinogenics were found in processed foods and food ingredients. In Brazil Arisseto *et al*., (2013) evaluated dietary exposure to 3-MCPD and 1, 3-DCP and checked whether the existence of these substances in foods may present health risks.

The aim of the study was to survey the level of 3-MCPD in Egypt caused by frying edible oils formed during the frying processed. Effect of feeding of rat on the diet containing palm oil treated to frying until formed 3MCPD in the biochemical parameters of bloods and their effects on the histological of some organs after two months

MATERIALS AND METHODS

Material:

Collection of samples:

Twenty samples of foods, oils and fats were collected from different supermarkets and restaurants of Mounofia , Qualiubia, Giza and Cairo governments, Egypt during years 2016.

Chemicals, reagents and solvents:

Sodium chloride, sodium bromide, ammonium sulphate, sodium hydrogen carbonate (purity ≥ 99%) , Sulfuric acid (purity ≥ 98%) , Isohexane, ethyl acetate, *tert*-butyl methyl, methanol for analysis and Isooctane chromatographic grade were obtained from (Merck-Darmstadt-Germany). Phenylboronic acid ,purified

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water (Fluke-Germany), Sodium sulfate (Sigma-Aldrich) , D_5 -3-MCDP-1,2-*bis*-palmitoyl ester (e.g 26.87 µgmL-1 in toluene; equivalent 5.0 µg mL-1 free 3-MCPD (Toronto Research Chemicals, Canada) were used.

Methods:

Determination of 3-MCPD ester contents:

The 3-MCPD esters were determined by DGF Standard Method C-VI 18 (10) (DGF, 2011) using GC-MS (GC-MS 2010, Shimadzu, Kyoto, Japan) system. Analysis of target compounds was performed according to the method of Chung *et al*., (2013). Chromatographic separation was by a capillary column (Restek Rxi-5ms column, 30m×0.25mm i.d. ×0.25µm film thickness) ; the injector was split less and helium at a constant flow rate of 1.18 mLmin-1, and oven temperature set as: 80°C raised to 155°C with a rate of 5°C min-1 raised to 300°C with 60°C min-1. Results were quantified by monitoring ions at m/z 150 for 3-MCPD-d5 and m/z 147 for 3-MCPD.

Gas chromatography/mass spectrometry.

Injection volume:1µL to 2µL; carrier gas: helium 5.0(φ=99.999%), const flow 1mL / min to 1.2mL / min; pTV program: e.g. 85 ° C, 300 ° C / min to 165 ° C, isothermal for 10 minutes, 300 ° C / min to 320 ° C / min, isothermal for 8 minutes, Injector: e.g. break less, purge flow 50 mL / min 0,5; to 1 minute, purge septum 3 mL / min; GC oven temperature program: e.g. 85 ° C, 0,5 min isothermal, 6 ° C / min at 150 ° C, 12 ° C / min at 180 ° C / min, 25 ° C at 280 ° C, 7 min isothermal. Mass spectrometric detector: electron- impact (EI), ion surveillance (SIM), ion traces detected,149/150/201/203 for surrogate standard,146/147/196/198 for study. Corresponding ion traces 150 and 147or 201 and 196 are for quantification while other ion traces serve as qualifiers

Animal study protocol:

To study response of albino rats to 3-monochloropropane-1, 2-diol (3-MCPD) in palm oil treated by heating five groups each is eight rats were used (El-Hadary and Hassanien, 2016).

First group fed on basal diet. Second group fed on basal diet replacing maize oil with palm oil. Third group, fed on basal diet replacing maize oil with heated palm oil at 200°C for 30 min. Fourth group, fed on basal diet replacing maize oil with palm oil heated at 200°C for 6h. Fifth group, fed on basal diet replacing maize oil with palm oil fried with tortilla chips.

Ingredients of diet

The main diet (basal diet) is composed of casein 10% as protein source, maize oil 8% as lipid source, maize starch 68.07% as the source of carbohydrate.Salts and vitamins mixture (Reeves *et al*., 1993).

Blood and tissue samples

After 8 weeks of the administration of treatments, blood samples were obtained from the retro-orbital plexus of overnight fasted rats from the individual rats. Blood was collected into a plain centrifuge tube for serum preparation to assay the biochemical parameters including liver function kidney function and lipid profile. Tissue samples from liver and kidney were fixed in 10% formalin saline for examination. Procedures were carried out (Schermer, 1967).

Blood analysis

Alanine transaminase (ALT) and aspartate transaminase (AST) activities were determined (Reitman and Frankel, 1957). ALP and total bilirubin were determined (Young *et al.*,1972 and Tietz, 1983) . Uric acid, urea and creatinine in the serum were determined (Tabacco *et al.*, 1979) whereas lipids, total cholesterol, triglycerides, HDL-cholesterol and LDL-cholesterol were determined according Fossati and Precipe (1982) and Fridewald *et al.*, (1972).

Histopathological examination

Thin paraffin sections were prepared and stained (Drury and Wallington 1986) at the Faculty of Veterinary medicine Benha University.

RESULTS AND DISCUSSION

MCPD esters concentration in samples

Data in Table 1 represent the levels of 3-monochloropropane-1, 2 diol fatty acid esters (3-MCPD esters) in the collected samples .

The oil samples of Falafel (Egyptian bean burger) after frying displayed the highest contents of 3-MCPD esters, with a mean of 7.41 µg/ kg oil varying from 4.97 to 8.30 µg/ kg oil, followed by palm oils and mixture oils, their mean values exceeded 3.80 µg /kg oil. Buffalo ghee had the lowest 3-MCPD esters(e.g 0.15 µg/ kg fat). On the other hand, 3-MCPD esters was detected. In contrast, few samples yielded 3-MCPD esters with concentrations below the detection limit when seeds were roasted for oil extraction. 3-MCPD esters were determined in margarine samples collected from Mounofia government (0.30 µg/ kg oil). The margarines were hydrogenated using palm oil. Thus they had high levels of 3-MCPD. From these results it can be concluded that refining affects formation of 3-MCPD. During refining process, most 3-MCPD esters was formed, in particular deodorisation (Li *et al*., 2015). Oil samples from FALAFEL restaurant were much higher than other samples .The wide variability in concentrations of 3-MCPD in oils, fat and food samples can be linked to the method used to process the product.Surveys conducted in the United Kingdom reported average levels of 134 µg/ kg in crackers and 16 µg/ kg in beers while pollutants were not detected in malted milk and breakfast cereals (UK FSA, 2001). It is possible to differentiate between refined and unrefined oils. 3-MCPD and 2-MCPD esters in all unrefined oils including rapeseed and coconut oil were not detectable. Results are similar results to those of Jedrkiewicz *et al*., (2016) in Poland who added that highest values were in lipid fractions of margarines and dietary supplements containing refined fish oils (7. 3 and 5.5 mg/ kg oil respectively).

The Biological Experiment

Effect of different diets containing palm oils treated on liver function marks.

Data in Table (2) show the mean value of serum transaminase activities of (ALT and AST), alkaline phosphates (ALP) and bilirubin as affected by 3-MCPD

From the above mentioned results it could be observed that the enzyme activities in serum rats fed on different diets containing palm oils treated to frying until formed 3MCPD for eight weeks caused significantly increases in serum ALT, AST and ALP activity compared with the control basal diet (group I), ALT activity was 42.33 U/L in basal diet (group I) and it increase to 121.33 U/L in rats fed on heated palm oil at 200°C for 6h (group IV) and 100.00 U/L in rats fed on fried palm oil with tortilla chips (group V). Furthermore ALT activity of serum rats fed on a diet containing crude palm oil (group II) was near to basal diet 54.00 U /L

Data in Table 2 reveal that AST activity was found to be 63.67 U/L in basal diet and it significantly increase to 145.00 and 141.67U/L in rats fed on heated palm oil at 200°C for 6h and rats fed on fried palm oil with tortilla chips comparing with control basal diet. Also, ALP activity were found to be 91.47 U/L in basal diet and it significantly increase to 208.65 and 205.29 U/L in rats fed on heated palm oil at 200°C for 6 h and rats fed on fried palm oil with tortilla chips (group IV and V) comparing with control basal diet. It can be concluded that group IV and V exposure to a high dosage of 3-MCPD and might be attributed to liver failure. These results were similar to those reported by Guan *et al*., (2017) who reported that 3-MCPD may cause carcinogenic effects in liver.

Data indicate that rats fed on heated palm oil at 200°C for 6h and rats fed on fried palm oil with tortilla chips for eight weeks caused significantly increase in total and direct bilirubin compared to normal rats (basal diet).

These results agreed with those mentioned by Wang *et al*., (2009) and Lee, *et al*., (2015) who found median or high dose exposure 1,3-DCP caused cancerous results in liver, intestine, oral epithelium, tongue and thyroid gland.

Table 1. Egyptian food samples collected to determine 3-monochloropropane-1, 2- and 3 diol fatty acid esters (3-MCPD esters).

No	Source of oil or fat	Source of samples	Concentration of 3-MCPD esters (µg/ kg)
1	*Falafel restaurants 1	Cairo	8.10
2	Falafel restaurants 2	Giza	8.30
3	Biscuit from market (salted)	Sadat	2.00
4	Corn flacks with chocolate	Qalubia	**0.98**
5	Bake Stix (Wheat snacks)	Mounofia	1.95
6	Palm oil	Cairo	3.80
7	Sunflour oil 1	Cairo	0.70
8	Sunflour oil 2	Qalubia	0.60
9	Corn oil	Qalubia	0.38
10	Rapeseed oil 1	Cairo	0.55
11	Rapeseed oil 2	Giza	0.55
12	Sesame oil 1	Cairo	0.50
13	Sesame oil 2	Cairo	0.55
14	Flax oil 1	Giza	0.00
15	Flax oil 1	Cairo	0.00
16	Mixture of oils 1	Cairo,	0.82
17	Mixture of oils 2	Qalubia	2.00
18	Margarine 1	Qalubia	0.33
19	Margarine 2	Mounofia	0.30
20	Buffalo ghee	Qalubia	0.15

***Falafel is a popular Egyptian beans-burgaer fried in oil**

Table 2. Effect of different diets containing palm oils treated on liver function marks.

G	Treatment	AST (U/ L)	ALT (U/ L)	ALP (U/ L)	Bilirubin (mg/dL)		
					Total	Direct	Indirect
I	Control (Basel diet)	63.67 ±2.73^{c}	42.33 ±1.45^{e}	91.47 ±1.96^{d}	0.70±0.03^{b}	0.29±0.01^{d}	0.41±0.01^{b}
II	Crude palm oil	72.33 ±1.45^{c}	54.00 ±2.08^{d}	117.77 ±1.47^{c}	0.88±0.04^{b}	0.47±0.04^{c}	0.42±0.02^{b}
III	Heated palm oil at 200°C for 30min	84.00 ±2.08^{b}	65.00 ±2.89^{c}	141.88 ±7.31^{b}	1.12±0.06^{b}	0.67±0.04^{b}	0.45±0.03^{b}
IV	Heated palm oil at 200°C for 6h	145.00 ±2.89^{a}	121.33 ±4.67^{a}	208.65 ±7.23^{a}	3.33±0.55^{a}	1.05±0.09^{a}	2.28±0.47^{a}
V	Palm oil fried with tortilla chips	141.67 ±6.01^{a}	100.00 ±2.89^{b}	205.29 ±12.57^{a}	3.43±0.27^{a}	1.19±0.07^{a}	2.24±0.2^{a}

a, b & c: There is no significant difference (P>0.05) between any two means, within the same column have the same superscript small letter

Effect of different diets containing palm oils treated on renal markers :

Serum creatinine, urea and uric acid are indicators of renal marker function : their increase indicate abnormality in kidney functions. Results of Table 3 indicate that theisel indicators were high in rats fed on heated palm oil at 200°C for 6h and rat fed on fried palm oil with tortilla chips (group IV and V) for eight weeks compared with the basal diet control (group I). In contrast rats that fed on a diet containing crude palm oil (group II), renal markers not affected when compared to basal diet (group I).It can be concluded that group IV and V have high amount of 3-MCPD and might be attributed to kidney failure. Rats fed on heated palm oil at 200°C for 6h showed the higest creatinine content of 5.13 followed by rats fed on fried palm oil with tortilla chips. Average values for these diets were 5.13 and 4.5 mg dL-1 respectively.

These results are in harmony with those obtained by Mahmoud *et al*., (2019), who investigated kidney

disorder induced by 3-MCPD in rodents as well as 3-MCPD was orally treated, at a dose of 60 mg/kg oil for seven days. 3-MCPD induced substantial rise of serum urea and creatinine levels along with hydropic degeneration, renal necrosis (Lynch *et al*., 1998). The results also agree with those of Lee, *et al*., (2015) observed failure of kidneys and reproductive organs function.

Table 3. Effect of different diets containing palm oils treated on renal function.

G	Treatment	Creatinine (mg/dL)	Urea (mg/dL)	Uric acid (mg/dL)
I	Control (Basel diet)	0.92±0.04^{d}	40.33±2.94^{d}	4.13±0.6^{c}
II	Crude palm oil	1.19±0.09^{d}	50.23±2.83^{c}	5.19±0.36^{c}
III	Heated palm oil at 200°C for 30min	1.60±0.06^{c}	64.71±2.41^{b}	7.10±0.15^{b}
IV	Heated palm oil at 200°C for 6h	5.13±0.13^{a}	85.25±2.88^{a}	9.68±0.30^{a}
V	Palm oil fried with tortilla chips	4.50±0.08^{b}	80.84±1.19^{a}	9.26±0.22^{a}

a, b & c: There is no significant difference (P>0.05) between any two means, within the same column have the same superscript small letter

Effect of different diets containing palm oils treated on lipid profile of rats :

Data in Table 4 reveal an increased blood lipid profile in rats fed on palm oil heated at 200°C for 6h and rats fed on fried palm oil with tortilla chips (group IV and V) for eight weeks compared with control (group I). It is evident that serum total lipid increased in rats fed on palm oil heated at 200°C for 6h (group IV) having a 29% increase ; and rats fed on fried palm oil with tortilla chips (group V) having a 33% increase as compared with the control (group I).

A similar trend is observed with serum triglycerides, with increases of 65.3 in rats fed on fried palm oil with tortilla chips (group V), 59.9% in rats fed on palm oil heated at 200°C for 6h (group IV), 26.1% in rats fed on palm oil heated at 200°C for 30 min (group III) and 14.8% in rats fed on crude palm oil (group II) as compared with the control (group I). Serum total cholesterol increased considerably in rats fed on fried palm oil with tortilla chips (group V), rats fed on palm oil heated at 200°C for 6h (group IV) and rats fed on palm oil heated at 200°C for 30min (group III) with respective increases of 71.9 , 68.7 and 37.1%. Changes in LDL-cholesterol and VLDL followed the same trend. On the other hand rats fed on fried palm oil with tortilla chips (group V) and rats fed on palm oil heated at 200°C for 6h (group IV) showed a considerable decrease in HDL cholesterol (-52%) relative to control basal diet (group I). Also rats fed on palm oil heated at 200°C for 30min (group III) reduction level of HDL cholesterol by about -35%

These results agree with Dereje *et al*., (2019), who observed that oxidized palm oil produced free radicals, promoted oxidative stress and caused abnormality in lipid profiles.. They found that reused palm oil caused harmful effects on serum lipid profile and and development of cardiovascular disorders.

Table 4. Effect of different diets containing palm oils treated on Lipid profiles.

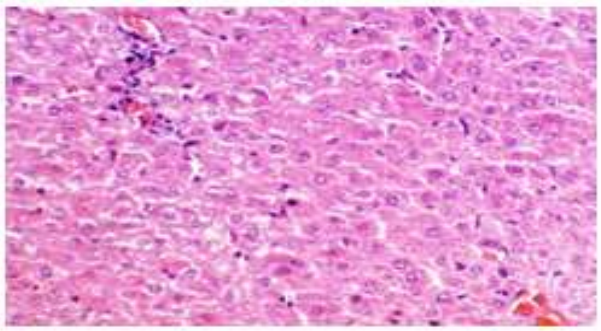
G	Treatment	Total lipids (mg/ dL)	Triglycerides (mg/ dL)	Total cholesterol (mg/ dL)	HDL-Cho (mg/ dL)	LDL-Cho (mg/ dL)	VLDL-Cho (mg/ dL)
I	Control (Basel diet)	460.35±5.32^{c}	178.77±4.33^{e}	160.34±4.78^{d}	57.73±1.63^{a}	66.86±2.38^{d}	35.75±0.87^{e}
II	Crude palm oil	488.50±4.43^{b}	205.30±2.88^{d}	188.55±4.43^{c}	47.21±1.18^{b}	100.28±2.70^{c}	41.06±0.58^{d}
III	Heated palm oil at 200°C for 30min	501.40±4.43^{b}	225.46±2.93^{c}	219.88±3.22^{b}	37.50±1.47^{c}	137.30±1.20^{b}	45.09±0.59^{c}
IV	Heated palm oil at 200°C for 6h	593.64±8.78^{a}	285.44±2.75^{b}	270.56±5.73^{a}	27.46±1.46^{d}	186.02±3.73^{a}	57.09±0.55^{b}
V	Palm oil fried with tortilla chips	610.27±5.86^{a}	295.52±2.72^{a}	275.30±2.80^{a}	27.57±1.86^{d}	188.63±0.51^{a}	59.10±0.54^{a}

a, b & c: There is no significant difference (P>0.05) between any two means, within the same column have the same superscript small letter

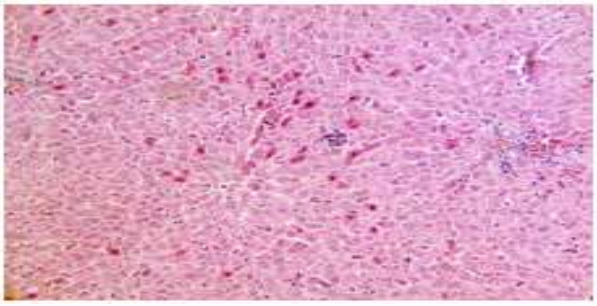
Histopathological examination

Liver histology

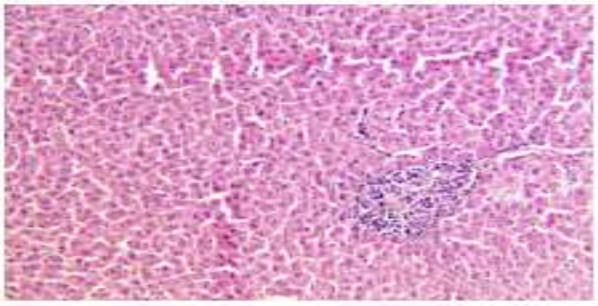
Figure 1 presents the histopathological images of liver sections of the studied five groups . Serial sections of the normal liver rat fed on basal diet (Group I) exhibited mild congestion of the central and portal veins with normal histological appearance of the hepatocytes. The portal areas were mildly expanded by small numbers of lymphocytes (Fig1, A). Examining the sections of liver rats fed on crude palm oil (Group II) revealed multifocally ; there were random foci of coagulative necrosis of individual hepatocytes, with retention of hepatic cell outline and shrunken hepatocytes with hyper eosinophilic cytoplasm and pyknotic or karyorrhectic nuclei (Fig1, B). Examining the sections of liver rats fed on heated palm oil at 200°C for 30 min. showed vacuolar degeneration of the hepatocytes and swollen pale cytoplasm. Focal coagulative necrosis of individual hepatocytes, characterized by retention of hepatic cell outline and shrunken hepatocytes with hyper eosinophilic cytoplasm and pyknotic nuclei, were also detected in few cases. Moreover, the portal areas were expanded by infiltrates of inflammatory cells mainly lymphocytes and few macrophages (Fig1, C). Examining the sections of liver rats fed on heated palm oil at 200°C for 6h revealed dilatation and congestion of the portal veins (Fig1, D). Examining the sections of liver rats fed on fried palm oil with tortilla chips showed characterized by dissociation of hepatic cord architecture, loss of hepatocytes and replacement by cellular and karyorrhectic debris, fibrin, and hemorrhage (Fig1, E).



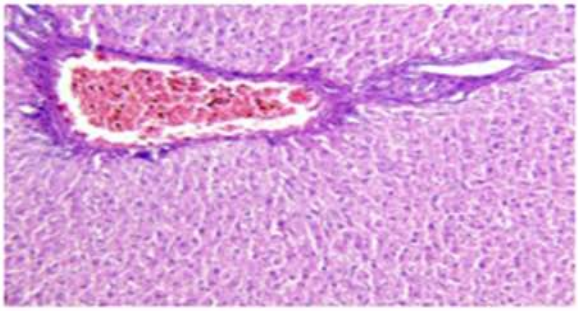
A. Liver of rat fed on basal diet control (Group I) showing mild congestion of the central and portal veins with normal histological appearance of the hepaocytes. Note also small numbers of lymphocytes in portal area. H&E stain x 200.



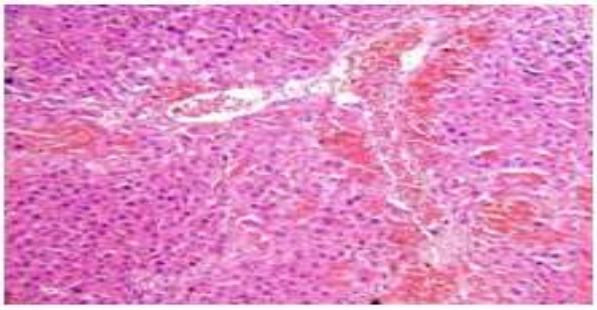
B. Liver of rat fed on crude palm oil (Group II) showing foci of coagulative necrosis of individual hepatocytes, characterized by retention of hepatic cell outline and shrunken hepatocytes with hypereosinophilic cytoplasm and pyknotic nuclei. H&E stain x 200.



C. Liver of rat fed on palm oil, heat treated for 30 minutes (Group III) showing, infiltrates of inflammatory cells in the portal area. Note also coagulative necrosis of individual hepatocytes H&E stain x 200.



D. Liver of rat fed on palm oil, heat treated for 6 hours (Group IV) showing dilatation and congestion of the portal vein. H&E stain x 200.

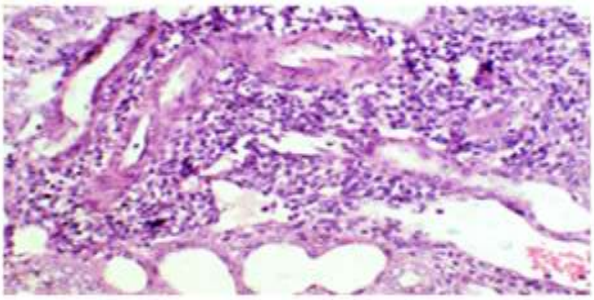


E. Liver of rat fed on palm oil fried with tortilla chips(Group V) showing areas of lytic necrosis characterized by dissociation of hepatic cord architecture, loss of hepatocytes and replacement by fibrin, and hemorrhage. H&E stain x 200.

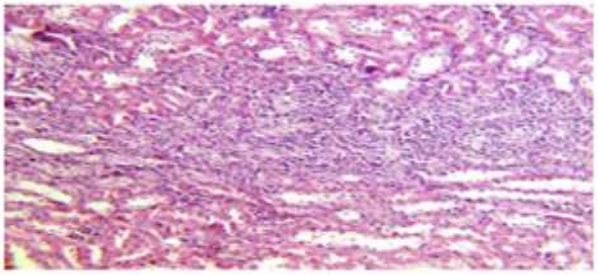
Figure 1. Histopathological photomicrographs of liver tissue (H&E X 200; A,B,C,D and E) at the end of the experimental.

Kidney histology

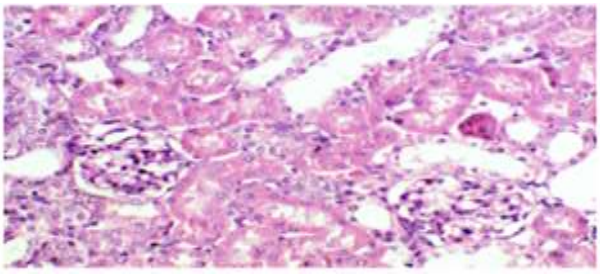
Figure 2 presents the histopathological images of kidney sections . Serial sections of the normal kidney rat fed on basal diet control (Group I). The examined kidneys revealed congestion of the renal blood vessels with perivascular and intertubular lymphocytic aggregates (Fig2, a). Sections of kidney rats fed on crude palm oil (Group II) revealed congestion of the cortical blood vessels, the interstitium was expanded by marked infiltration of inflammatory cells (Fig2, b). Sections of kidney rats fed on heated palm oil at 200°C for 30min revealed lymphocytes and macrophages (Fig2, c). Sections of kidney rats fed on heated palm oil at 200°C for 6h revealed finely granular eosinophilic cytoplasm and narrow lumen (Fig2, d). Sections of kidney rats fed on fried palm oil with tortilla chips revealed congestion of cortical blood vessels and interstitial aggregates of mononuclear inflammatory cells .



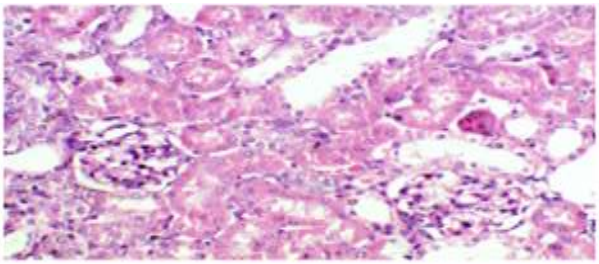
A. Kidney of rat fed on untreated corn oil (Group I) showing perivascular and intertubular lymphocytic aggregates. H&E stain x 200.



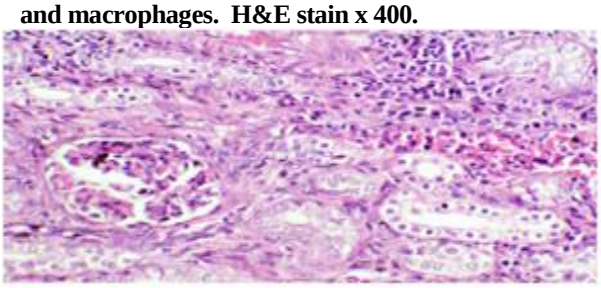
B. Kidney of rat fed on crude palm oil (Group II) showing the marked interstitial infiltration of inflammatory cells. H&E stain x 200.



C. Kidney of rat fed on palm oil, heat treated for 30 minutes (Group III) showing interstitial infiltration of inflammatory cells in renal cortex mainly lymphocytes and macrophages. H&E stain x 400.



D. Kidney of rat fed on palm oil, heat treated for 6 hours (Group IV) showing cloudy swelling of renal tubular epithelium characterized by finely granular eosinophilic cytoplasm and narrow lumen. H&E stain x 400.



E. Kidney of rat fed on fried palm oil with tortilla chips (Group V) showing congestion of the cortical blood vessels and interstitial aggregates of mononuclear inflammatory cells. H&E stain x 400.

Figure 2. Histopathological photomicrographs of kidney tissue (H&E X 200&400; A, B, C, D and E) at the end of the experimental

CONCLUSION

In conclusion, formation of 3-monochloro1, 2-propanediol during deep-oil frying in some Egyptian foods and characterized the toxicity of 3-MCPD in liver and kidney in rats is reported in the present study. The 3-MCPD formation was affected by the type of oil and foods.3-MCPD in oil samples from falafel restaurant were much higher than other samples and over the limits that determined by different regulation around the world (2 µg/kg oil). The biological experiment of rats which feed on diet containing fried palm oil revealed high significantly increasing activity in liver and kidney marker as well as lipid profile except HDL-cholesterol and damage in liver and kidney tissues..

REFERENCES

Arisseto, A. P., Vicente, E., Furlani, R. P. Z., and Toledo, M. C. D. F. (2013). Estimate of dietary intake of chloropropanols (3-MCPD and 1, 3-DCP) and health risk assessment. *Food Science and Technology*, *33*, 125-133

Aznar, M., Gomez-Estaca, J., Velez, D., Devesa, V., and Nerín, C. (2013). Migrants determination and bioaccessibility study of ethyl lauroyl arginate (LAE) from a new antimicrobial food packaging material. Food and Chemical Toxicology, 56, 363-370. https://doi.org/10.1016/j.fct.2013.02.018

Chung, C., Chan, S. W., Chung, B. T., Xiao, Y. and Ho, Y. Y. (2013). Occurrence of bound 3-monochloropropan-1, 2-diol content in commonly consumed foods in Hong Kong analysed by enzymatic hydrolysis and GC-MS detection. *Food Addit Contam A*, 30(7): 1248-1254,

Collier, P. D., Cromie, D. D. O. and Davies, A. P. (1991). Mechanism of formation of chloropropanols present in protein hydrolytes. Journal of the American Oil Chemists Society, 68(10), 785-790.

Dereje, G., Anandakumar, P., and Gizaw, M. (2019). Effect of reused palm oil on serum lipid profile in experimental rats, Adv J Pharm Life sci Res, 7;2:1-4.

DGF (2011) Fatty-acid-bound 3-chloropropane-1,2-diol (3-MCPD) and 2,3-epoxipropane-1-ol (glycidol) Determination in oils and fats by gas chromatography/mass spectrometry (GC/MS) DGF Standard Method C-VI 18(10)

Drury, R. and Wallington, E. (1986). Carlton's histological technique. 4th Ed., Oxford Univ. Press., N.Y., Toronto.

El-Hadary, A. E. & Hassanien, R. M. F. (2016) Hepatoprotective effect of cold-pressed *Syzygium aromaticum* oil against carbon tetrachloride (CCl_4)-induced hepatotoxicity in rats. Pharmaceutical Biology 54:1364-1372. https : // doi.org/ 10.3109 / 13880209.2015.1078381

Fossati, P. and Precipe, L. (1982). The determination of triglycerides using enzymatic methods. Clin. Chem. 28: 2077.

Fridewald, W. T.; Leve, R. I. and Fredrickson, D. S. (1972): Estimation of concentration of low density lipoprotein without the use of preparative ultracentrifuge. Clinical Chemistry, 18, 499–502.

Guan, S., Yu, X., Fang, B., Huang, Y., Xu, L. and Lu, J. (2017). The toxicity of 3-monochloro-1, 2-propanediol (+) to activated T cells in mice. Food and Agricultural Immunology, 28(4), 612-624 .

Jędrkiewicz, R., Głowacz, A., Gromadzka, J., and Namieśnik, J. (2016). Determination of 3-MCPD and 2-MCPD esters in edible oils, fish oils and lipid fractions of margarines available on Polish market. Food Control, 59, 487-492.

Lee, B. S., Park, S. J., Kim, Y. B., Han, J. S., Jeong, E. J., Moon, K. S., and Son, H. Y. (2015). A 28-day oral gavage toxicity study of 3-monochloropropane-1, 2-diol (3-MCPD) in CB6F1-non-Tg rasH2 mice. Food and Chemical Toxicology, 86, 95-103.

Li, C., Nie, S. P., Zhou, Y. Q., and Xie, M. Y. (2015). Exposure assessment of 3-monochloropropane-1, 2-diol esters from edible oils and fats in China. Food and Chemical Toxicology, 75, 8-13.

Lynch, B.S., Bryant, D.W., Hook, G.J., Nestmann, E.R. and Munro, I.C. (1998). Carcinogenecity of monochloro-1,2-propanediol (alpha-chlorohydrin, 3-MCPD). Int. J. Toxicol. 17, 47–76.

Mahmoud, Y. I., Abo-Zied, F. S., and Salem, S. T. (2019). Effects of subacute 3-monochloropropane-1, 2-diol treatment on the kidney of male albino rats. Biotechnic & Histochemistry, 94(3), 199-203.

Reeves, P.G., Nielsen, F.H. and Fahey, G. C.(1993) AIN-93 purified diets for laboratory rodents: final report of the American Institute of Nutrition ad hoc writing committee on the reformulation of the AIN-76A rodent diet. J. Nutr. 123: 1939-1951.

Reitman, S. and Frankel, S. (1957):A colorimetric method for the determination of serum glutamic oxaloacetic and glutamic pyruvic transaminases. Am. J. Clin. Path. 22l(5/6): 56.

Schermer, S. (1967). The Blood Morphology of Laboratory Animals, 3rd ed., Davies, F.A., Company Pheladelphia ,W.S.A.P.42.

Schilter, B., Scholz, G., and Seefelder, W. (2011). Fatty acid esters of chloropropanols and related compounds in food: Toxicological aspects. *European Journal of Lipid Science and Technology*, *113*(3), 309-313.

Svejkovska, B., Novotny, O., Divinova, V., Reblova, Z., and Dolezal, M. (2004). Esters of 3-chloropropane-1, 2-diol in foodstuffs. *Czech Journal of Food Sciences-UZPI (Czech Republic)*. 22 (5): 190–196

Tabacco, A., Meiattini, F, Moda, E. and Tarlip (1979): Simplified enzymitic colorimetric serum urea nitrogen determination. Clin. Chem. 25: 336-337.

Tietz, N.M. (1983): Textbook of clinical chemistry. W.B. sunders Co. Try. 64: 1312-1316.

UK Food Standards Agency (2001): Food Surveillance Information Sheets No. 12/01 and 14/01

Wang, J., Sawyer, J.R., Chen, L., Chen, T., Honma, M., Mei ,N. and Moore, M.M (2009). The mouse lymphoma assay detects recombination, deletion, and aneuploidy. Toxicol Sci 109:96- 105.

Weißhaar, R., Perz, R. (2010). Fatty acid esters of glycidol in refinedfats and oils. Eur. J. Lipid Sci. Technol, 112, 158–165.

Young, D.S.; Thomas, D.W.; Friedman, R.B. and Pretaner, L.C. (1972). Effect of drugs on clinical laboratory tests. Clinical Chemistry 18 (1): 1041 -1303.

تقدير 3- أحادى كلور بروبان 1،2 داى ول فى بعض الاغذية المصرية وتأثيرة على سمية الكبد والكلى فى الجرذان

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توضح هذه الدراسة نتائج حصرمركب 3-أحادى كلور بروبان 1،2 داى ول فى بعض الاغذية والزيوت والدهون التى تم جمعها من السوبر ماركت والمطاعم فى مصر وكذلك دراسة تأثير تغذية الجرذان على عليقة تحتوى على زيت النخيل الغنى بمركب 3-أحادى كلور بروبان 1،2 داى ول على المقاييس البيوكيميائية فى دم الجرذان. تم جمع عشرين عينة من محلات السوبر ماركت والمطاعم المختلفة في مصر لتقدير 3- أحادى كلور بروبان 1،2 داى ول بواسطة جهاز التحليل الكروماتوجرافى الغازى المزود بمطياف الكتلة. أظهرت عينات زيت الفلافل أعلى محتوى من استرات 3-أحادى كلور بروبان 1،2 داى ول بمتوسط 8.20 ميكروجرام / كيلوجرام ثم زيت النخيل الخام ثم مخاليط الزيوت. . تم تقسيم أربعين جرذ ألبينو إلى خمس مجموعات أحد هذه مجموعة ضابطة يتم تغذيتها على العليقة الاساسية والمجموعات الاخري تم تغذيتهاعلى العليقة الاساسية مع أستبدال زيت الذرة بزيت النخيل الخام ، وزيت النخيل المسخن عند 200 درجة مئوية لمدة 30 دقيقة ، وزيت النخيل المسخن عند 200 درجة مئوية لمدة 6 ساعات أو زيت النخيل المسخن عند 200 درجة مئوية مع رقائق التورتيلا. أظهرت النتائج أن الجرذان المغذاه على عليقة تحتوى على زيت النخيل المسخن عند 200 درجة مئوية لمدة 6 ساعات أو زيت النخيل المسخن عند 200 درجة مئوية مع رقائق التورتيلا أدت إلى أرتفاع وظائف الكبد والكلى وكذلك وبرفيل الدهون وأنخفاض تركيز HDL-Cho وتلف في بعض أنسجة الكبد والأنسجة الكلوية مقارنة بالمجموعة الضابطة وذلك ربما يرجع الى تكوين أسترات 3- أحادى كلور بروبان 1،2 داى ول اثناء عملية القلى.