Zagazig J. Agric. Res., Vol. 36 No. (1) 2009 171 - 183

BIOLOGICAL EVALUATION OF HAZARDS RESULTING FROM FEEDING ON DEEP FRYING OILS

Abd El- Kader, Hend, E., M.M.A. Amer, S.S. El- Saadany, and A.M. Al- Gaby

Agric. Biochem. Dept., Fac. of Agric., Zagazig Univ.

Accepted 23/ 2/2009

ABSTRACT: The purpose of the present study was to investigate the effect of dietary deep frying oil (DFO) on the gain in body weight changes, liver and kidney body weight ratio, blood glucose level, lipids pattern, liver and kidneys functions of male Albino rats which fed on diets containing either 10% fresh oil blend consisted of a mixture (50: 35: 15, wt./wt.) of palm olein, soyabean oil and sunflower oil (PO/SYO/SO), respectively, and served as negative control or 10 % DFO. DFO prepared by frying an oil blend at 200 ± 5 °C, for 6 h each for four consecutive days. This DFO fed to another group of rats and served as positive control (G3). The other two groups were fed on an oil blend in which Tamiea was fried (G1) and potato chips and/or eggplant slices were fried (G2) using the same technique of 6 h for four consecutive days each. The duration of the experiment was prolonged for six weeks, after feeding for one week as adaptation period.

The obtained results showed that feeding on DFO slightly decreased the gain in body weight but markedly increased both liver and kidneys body weight ratios relative to the negative control. There were a highly significant increase in serum triacylglycerides, total serum cholesterol, LDL- cholesterol and blood sugar, hepatic enzymes activities (AST and ALT), blood urea, s-creatinine and s-uric acid parameters as indicators of liver and kidney functions, respectively, while there was a significant decrease in serum HDL-cholesterol displayed a significant differences between negative control, G1, G2 and G3 (positive control) of rats fed on diets containing fresh oil blend and deep frying oils (DFO).

Key words: Liver and kidneys functions, lipid profile, dietary deep frying oil blends.

INTRODUCTION

Because of their crispness and aromatic properties, fried foods are immensely popular throughout the world and contribute markedly to our total energy intake. During the deep frying process, a series of degradation reactions, including autoxidation, thermal oxidation, polymerization, cyclization and fission occur in the frying fat/oil (Chang *et al.*, 1978 and Paul and Mittal, 1997). Lipid peroxides, the primary autoxidation products, are rapidly degraded at the high temperature of frying. The nonvolatile secondary oxidation products retained in the used frying oil, comprised mainly of oxidized triglyceride (TG) monomers, dimers and polymers, are of great nutritional importance because they are absorbed into the fried foods and ingested. The oxidized TG contain at least one oxygenated function in the esterified fatty acids and may be a mixture of epoxides, ketones, alcohols, as well as polyoxygenated compounds. The dimers and polymers are complex structures in which TG monomers are covalently linked through C-C or C-O-C bonds (Paul and Mittal, 1997).

Although toxic fractions have been isolated from laboratory-abused oil (Artman , 1969), long-term rats feeding studies using fat samples oxidized under more realistic cooking practices as part of a nutritional balanced diet resulted only in mild symptoms such as less body weight gain and feed intake and enlargement of liver and kidney (Nolen *et al.*, 1967 and Poling *et al.*, 1970). Therefore, the frying oil that is ingested with the fried foods is generally regarded as safe as long as the organoleptic quality of the fried foods is acceptable (Artman, 1969 and Kubow, 1992). With substantial accumulation of oxidation products, especially dimers and polymers, the frying operation becomes difficult due to vigorous foaming and foods fried in such oil become unpalatable (Artman, 1969 and Paul and Mittal, 1997). Digestion and absorption of the dimerized and polymerized TG are decreased (Marquez- Ruiz *et al.*, 1998 and Gonz'alez- Munoz *et al.*, 1998). The effective detoxifying capability of liver microsomal enzymes, including cytochrome P450 monoxygenase and phase II conjugation enzymes, which are significantly induced by an

oxidized frying oil (OFO)-containing diet (Huang *et al.*, 1988). Interestingly, a lowered TG in plasma and liver of rats fed OFO was repeatedly observed (Huang *et al.*, 1988 and Liu and Huang 1995).

In vitro studies showed that oxidized LDL and its component hydroxy fatty acids, including hydroxy eicosatetraenoic acid and hydroxy octadecadienoic acid (HODE), were activated (Nagy *et al.*, 1998 and Delerive *et al.*, 2000). Because hydroxy fatty acids are among the various oxidation products in frying oil (Paulose and Chang, 1973) that may be absorbed after digestion.

The aim of this work is to investigate the effect of deteriorated used frying oil (DUFO) prepared from different types of oil that heated at 200 ± 5 °C for 4 days x 6 h frying time on gain in body weight, liver and kidney functions, lipid pattern and the risk ratio.

MATERIALS AND METHODS

An oil blend consisted of a mixture of palm olein, soyabean oil and sunflower oil (50: 35: 15, wt./wt.) was obtained from the local market packed in dark brown bottles and refrigerated at 5°C tell used.

Male Albino rats (95 – 110 g) were used. The animals were divided into two groups. The 1st group was the control group in which the oil blend was used as it is without any treatment and incorporated into the experimental diet which was prolonged for six weeks. The 2nd group was subdivided into three subgroups.

The 1st subgroup (G1) of rats was administrated an oil blend in which Tamiea was fried at 200 ± 5 °C for 4 days x 6 h each. The 2nd subgroup (G2) administrated an oil blend in which potato chips and/or eggplant slices were fried at 200 ± 5 °C for 4 days x 6 h each and the 3rd subgroup (G3, positive control) administrated fried oil blend at 200 ± 5 °C for 4 days x 6 h each. The duration of the experiment was prolonged for six weeks. All groups were fed on basal diet according to Ghali *et al.* (2000), vitamin mix. was prepared according to A.O.A.C.(1990) and mineral mixture was prepared according to Hegsted *et al.* (1941).

The rats were housed in cages with screen bottom in a controlled environment with 12hr light and 12hr dark cycle. Diets and water

were supplied *ad libtum* and at the end of experimental period (42 days), rats fasted for 16-18 hrs, blood samples were collected by sacrificing all groups using decapitation under chloroform anesthesia into tubes and then centrifuged at 3000 rpm for 10 min to obtain serum which was kept frozen for analysis.

Separated serum samples extracted according to Hussein *et al.* (1989) were subjected to the biochemical analysis including: total cholesterol (Naito and Kaplan 1984), HDL-cholesterol (Warnick *et al.* 1983), LDL-cholesterol (Friedwald *et al.* 1972); triglycerides (Fossati and Prencipe 1982), AST and ALT (Reitman and Frankel 1957), total protein (Doumas *et al.* 1971), blood urea (Patton and Crouch 1977) and s. creatinine (Jaffe 1986) glucose, according to the method of Trinder (1969). The risk ratio is the result of total cholesterol divided by HDL-cholesterol according to the method of Ghali *et al.* (2000).

The obtained results were statistically analyzed using analysis of variance and Least Significant Difference (L.S.D) at 0.05% level of probability as reported by Snedecor and Cochran (1980).

RESULTS AND DISCUSSION

Results

Data in Table 1 indicated the initial and final body weight and it's changes, liver weight, kidney weight, liver body weight and kidney body weight ratios of normal rats (negative control), G1, G2 and G3 (positive control) groups fed on diets containing fresh oil blend and deep frying oil (DFO). Feeding on DFO slightly decreased the gain in body weight relative to the negative control group. Liver and kidneys body weight ratios of rats fed on diets containing DFO G1, G2 and G3, (positive control) were significantly increased relative to the negative control group, but G1and G2 have no significance to positive control.

Table 2 showed the effect of administrating oil blends on blood glucose, serum triacylglycerides (TAG), total serum cholesterol, LDL -cholesterol., HDL- cholesterol (mg/dl) and risk ratio.

Table 1. Initial and final body weight changes and liver weight, kidney weight, liver body weight ratio and kidney body weight ratio of negative control, G1, G2 and G3 (positive control) groups fed on diets containing fresh oil blend* and deep fried oil (DFO)

Groups	Initial body weight (g)	Final body weight (g)	Gain in body weight (g)	Gain in body weight Change %	Liver weight (g)	Liver body weight ratio %	Kidney weight (g)	Kidney body weight ratio %
Negative control	(NS) 104.6 ± 5.7	(NS) 115.4 ± 5.9	+ 10.8	+10.33	2.46^{a} ± 0.48	2.35	0.55^{a} ± 0.12	0.48
G 1	97.6 ± 6.69	104.2 ± 5.07	+ 6.6	+ 6.76	3.36^{b} ± 0.87	3.32	0.68^{b} ± 0.19	0.65
G 2	100 ± 6.78	107 ± 5.15	+ 7.0	+ 7.0	3.67^{b} ± 0.681	3.43	0.68^{b} ± 0.19	0.64
G3 (Positive control)	97.6 ± 12.52	107 ± 11.51	+ 9.4	+ 9.63	3.79^{b} ± 0.32	3.89	0.75^{b} ± 0.91	0.70

Each value represents the mean of 6 rats in g ± S.D.

G1: Oil blend in which Tamiea was fried at 200 ± 5 °C for 4 days x 6 h each.

G2: Oil blend in which potato chips and /or eggplant slices were fried at 200 ± 5°C for 4 days x 6 h each.

G3: Oil blend in which fried oil blend at 200 ± 5 °C for 4 days x 6 h each (Positive control).

*: Oil blend / palm olein: soybean oil: sunflower oil (50: 35: 15, wt./wt.)

(NS): Non significant

a,b,c ..... etc numbers bearing different letters are significantly different.

Table 2. Blood glucose, serum triglycerides, total cholesterol, LDL, HDL - cholesterol (mg/dl) and risk ratio in control, G1; G2 and G3 rats fed on diets containing fresh oil blend* and deep fried oil (DFO)

Groups	Blood glucose		S. triglycerides		S. total cholesterol		LDL cholesterol		HDL cholesterol		Risk ratio (LDL/HDL)	
	mg/dl	Change %	mg/dl	Change %	mg/dl	Change %	mg/dl	Change %	mg/dl	Change %	Ratio	Change %
Negative control	(NS) 99.20 ± 4.97	100	85.40^{a} ± 5.73	100	72.12^{a} ± 3.38	100	(NS) 24.36 ± 9.83	100	38.60^{a} ± 2.97	100	0.63	100
G 1	104.8 ± 9.92	105.64	165.8^{b} ± 9.73	194.15	95.80^{b} ± 6.26	132.8	36.44 ± 8.77	149.59	26.20^{b} ± 1.92	67.87	1.39	220.6
G 2	98.00 ± 9.66	98.79	167.00^{b} ± 9.08	195.55	92.60^{b} ± 6.07	128.39	30.60 ± 7.23	125.62	28.60^{b} ± 2.30	74.09	1.07	169.84
G3 (Positive control)	110.60 ± 8.08	111.49	152.80^{b} ± 13.03	178.92	87.20^{b} ± 8.70	120.90	30.24 ± 8.98	124.13	36.40^{a} ± 2.07	94.3	0.83	131.7

Each value represents the mean of 6 rats in mg/ dl ± S.D.

G1: Oil blend in which Tamiea was fried at 200±5 C for 4 days x 6 h each.

G2: Oil blend in which potato chips and /or eggplant slices were fried at 200 ± 5 °C for 4 days x 6 h each.

G3: Oil blend in which fried oil blend at 200 ± 5 °C for 4 days x 6 h each (Positive control).

* : Oil blend /palm olein: soybean oil: sunflower oil (50: 35: 15, wt./wt.)

(NS): Non significant

a,b,c ..... etc numbers bearing different letters are significantly different.

There was a highly significant increase in triglycerides (TAG), total serum cholesterol and LDL - cholesterol and significant decrease of serum HDL cholesterol of rats fed on diets containing deep frying oil (DFO) relative to rats fed on diet containing the fresh oil blend. Also blood glucose level was markedly increased in G1 and G3 and decreased in G2 rats fed on diet containing deep frying oils.

Table 3 displayed significant differences in serum creatinine, blood urea, and serum uric acid. These parameters are indicators of kidney function, between negative control group, G1, G2 and G3 (positive control) rats fed on diets containing fresh oil blend and deep frying oils (DFO).

Table 4 showed the effect of feeding on oil blends on hepatic enzymes (AST and ALT), total protein, albumin and globulin. These parameters significantly affected by using DFO blends, as AST and ALT significantly increased while total protein and albumin showed an opposite trend.

Discussion

This study was conducted to investigate the effect of feeding on deteriorated used frying oil (DUFO) upon the changes in body weight; liver and kidney to body weigh ratio, blood glucose level, lipid profile, liver and kidney functions of male Albino rats. The duration of the experiment was prolonged for six weeks. Group of rats was fed on fresh oil blend consisted of palm olein, soybean oil and sunflower oil (50: 35: 15, wt./wt) and served as negative control. Group 1 (G1) fed on the same blend in which Tamiea was fried. Group 2 (G2) was fed on the same blend in which potato chips and /or eggplant slices were fried. The third group (G3) was fed on the same blend of oil which simply heated alone at the same conditions and served as positive control.

There were significant interactions between the effect of dietary fat quality on body weight gain, relative liver and kidney weight (Table 1). Dietary deep frying oil blends significantly reduced body weight gain, increased liver and kidney weight ratio relative to the negative control group. The obtained results agreed with those obtained by Nolen *et al.* (1967) and Poling *et al.* (1970) who found that less body weight gain and enlargement of liver when they used fat samples oxidized under more realistic cooking practices as part of a nutritional balanced diet.

Table 3. Serum uric acid, blood urea and serum creatinine (mg/dl) in negative control group, G1, G2 and positive control (G3) rats fed on diets containing fresh oil blend* and deep fried oil (DFO)

Groups	S. Uric acid		B. Urea		S. Creatinine	
	mg /dl	Change %	mg/dl	Change %	mg/dl	Change %
Negative control	$5.11^{a} \pm 0.53$	100	$34.00^{a} \pm 2.01$	100	$0.66^{a} \pm 0.20$	100
G 1	$5.54^{a} \pm 0.84$	108.41	$73.60^{b} \pm 4.83$	216.47	$0.96^{b} \pm 0.11$	145.45
G 2	$5.86^{b} \pm 0.22$	114.68	$77.20^{b} \pm 16.19$	227.06	$0.94^{b} \pm 0.11$	142.42
G 3 (Positive control)	$5.90^{b} \pm 0.36$	115.46	$61.00^{b} \pm 16.72$	179.41	$0.92^{b} \pm 0.13$	139.39

Each value represents the mean of 6 rats in mg/ dl ± S.D.

G1: Oil blend in which Tamiea was fried at 200 ± 5 °C for 4 days x 6 h each.

G2: Oil blend in which potato chips and /or eggplant slices were fried at 200 ± 5 °C for 4 days x 6 h each.

G3: Oil blend in which fried oil blend at 200 ± 5 °C for 4 days x 6 h each (Positive control).

* : Oil blend / palm olein: soybean oil: sunflower oil (50: 35: 15, wt./wt.)

a,b,c ..... etc numbers bearing different letters are significantly different.

Table 4. Serum total protein, albumin, globulin (g/dl) and means of AST, ALT (IU/L) and AST/ALT ratio in negative control, G1, G2 and positive control (G3) rats fed on diet containing fresh oil blend* and deep fried oil (DFO)

Groups	AST (s. GOT)		ALT (s. GPT)		AST/ ALT		Total protein		Albumin		globulin	
	(IU/L)	%	(IU/L)	%	Ratio	%	g / dl	Change %	g / dl	Change %	g / dl	Change %
Negative control	$17.00^{a} \pm 2.45$	100	$11.40^{a} \pm 1.14$	100	1.58	100	$7.01^{a} \pm 0.07$	100	$4.44^{a} \pm 0.18$	100	(NS) 2.57 ± 0.48	100
G 1	$41.40^{bc} \pm 8.2$	243.5	$21.80^{b} \pm 1.92$	191.2	1.98	188.9	$5.02^{b} \pm 0.67$	71.60	$2.40^{b} \pm 0.29$	54.05	2.62 ± 0.60	101.9
G 2	$49.80^{bd} \pm 7.19$	292.9	$20.20^{b} \pm 2.17$	177.2	2.66	137.04	$4.84^{b} \pm 0.24$	69.04	$2.38^{b} \pm 0.19$	53.60	2.46 ± 0.29	95.57
G3 (Positive control)	$45.40^{b} \pm 5.18$	267.0	$22.60^{b} \pm 3.36$	198.2	2.5	159.26	$5.28^{b} \pm 0.28$	75.32	$2.58^{b} \pm 0.31$	58.11	2.7 ± 0.52	105.05

Each value represents the mean of 6 rats in g/ dl or IU/L ± S.D.

G1: Oil blend in which Tamiea was fried at 200 ± 5 °C for 4 days x 6 h each.

G2: Oil blend in which potato chips and /or eggplant slices were fried at 200 ± 5 °C for 4 days x 6 h each.

G3: Oil blend in which fried oil blend at 200 ± 5 °C for 4 days x 6 h each (Positive control).

* : Oil blend / palm olein: soybean oil: sunflower oil (50: 35: 15, wt./wt.)

(NS) : Non significant.

a,b,c,d ..... etc numbers bearing different letters are significantly different.

Blood glucose level was slightly affected by dietary deep frying oil blends, whereas, serum triglycerides and total serum cholesterol (Table 2) were significantly increased relative to the negative control group. Also, serum LDL-cholesterol, showed a slight increase whereas, serum-HDL- cholesterol showed a highly significant decrease relative to the negative control. The risk ratio (LDL/HDL) for group of rat fed on DUFO was increased, relative to both of negative and positive control groups. The obtained results agreed with those obtained by Paulose and Chang, (1973), Nagy *et al.* (1998), Delerive *et al.*, (2000) and Ghali, *et al.*, (2000)

Most people believe that frying creates trans fats. That is not the major problem, there are more toxic chemicals produced by frying omega-6 than trans fats. Frying destroys the antioxidants in oils and as much oxidizes the oils and make cross-linking, cyclization, double-bond shifts, fragmentation and polymerization of oils that cause more damage than trans fats. This was noted from the impaired kidney and liver functions illustrated in Tables 3 and 4.

Kidney and liver functions were affected as serum uric acid level was significantly increased in G2 and G3 (positive control), while blood urea and serum creatinine, AST (s- GOT) and ALT (s-GPT) levels were significantly increased in all treated groups, relative to the negative control. Also, there was significant difference between G1 and G2 in AST (s- GOT) parameters, this may be due to material fried in the oil mixture.

Total protein and albumin levels were significantly decreased in all treated groups relative to the negative control.

Our results agreed with that obtained by Huang *et al.* (1988), who reported that feeding male Long Evants young rats on deteriorated frying oil (DUFO) caused a significant elevated serum GOT (E.C.2.6.1.1, glutamate – oxaloacetate transaminase) and GPT (E.C.2.6.1.2, glutamate – pyruvate transaminase) values.

REFERENCES

A.O.A.C. 1990. Association of Official Analytical Chemists. Official Methods of Analysis. A.O.A.C., Washington D.C.

Artman, N.R. 1969. The chemical and biological properties of heated and oxidized fats. Adv. Lipid Res., 7:245-330.

Chang, S.S., R.J. Peterson and C.T., Ho. 1978. Chemical reactions involved in the deep-fat frying of foods. J. Am. Oil Chem. Soc., 55:718-727.

Delerive, P., C. Furman, E. Teissier, J.C. Fruchart, P. Duriez and B. Staels. 2000. Oxidized phospholipids activate PPARa in a phospholipase A2-dependent manner. FEBS Lett., 471:34-38.

Doumas, B.T., W.A. Watson and H.G. Biggs. 1971. Albumin standards and the measurement of serum albumin with bromocresol green. Clin. Chem. Acta., 31: 87-96.

Fossati, P. and L. Prencipe. 1982. Serum triglycerides determined colorimetrically with an enzyme that produces hydrogen peroxide. Clin. Chem., 28: 2077 -2080.

Friedewald, T., I. William, L. Robert and S. Fredrickson. 1972. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ullracentrifuge. Clinical Chemistry, 18:499-502.

Ghali, Y., R. Laila, A.A. Moustafa, M.M. Wasef and S.N. Abdel-Rahman. 2000. Cholesterol-Lowering effect of various rice bran diets on hypercholesterolemic rats. The Egyptian J. of Biochemistry, 18: 213-228.

Gonz'alez-Munoz, M.J., S. Bastida and F.J. S'anchez-Muniz. 1998. Short-term in vivo digestibility of triglyceride polymers, dimmers, and monomers of thermoxidized palm olein used in deep-frying. J. Agric. Food Chem., 46:5188-5193.

Hegsted, D.M., R.C. Mills, C.A. Elvehjem and E.B. Hart. 1941. Choline in the nutrion of chickns. J. Bio. Chem., 138: 459-450.

Huang, C.J., N.S. Cheung and V.R. Lu. 1988. Effects of deteriorated frying oil and dietary protein levels on liver microsomal enzymes in rats. J. Am. Oil Chem. Soc., 65:1796-1803.

Hussein, Mona M., T. Thoraya, Damhougi and Y. Sahar, Al-Okbi. 1989. Effect of high dietary sucrose and fat levels on liver lipids and proteins. Egypt. J. Food Sci., 17, No 1-2,15-22.

Jaffe, M. 1986. ueber den Niederschlag, welchen Pikrinsaure in normalem Harn erzeugt und uber eine neue Reaktion des Kreatinins. Z. Physiol Chem., 10:391-406.

Kubow, S. 1992. Routes of formation and toxic consequences of lipid oxidation

products in foods. Free Radical Biol. Med. 12:63-81.

Liu, J.F. and C.J. Huang. 1995. Effect of oxidized frying oil on the lipid metabolism in rats. J. Clin. Nutr. Soc., 20:303-316.

Marquez-Ruiz, G., G. Guevel and M.C. Dobarganes. 1998. Applications of chromatographic techniques to evaluate enzymatic hydrolysis of oxidized and polymeric triglycerides by pancreatic lipase *in vitro*. J. Am. Oil Chem. Soc., 75:119-126.

Nagy, L., P.J., Tontonoz, G.A. Alvarez, H. Chen and R.M. Evans. 1998. Oxidized LDL regulates macrophage gene expression through ligand activation of PPAR7. Cell 93:229-240.

Naito, H.K. and A.Q. Kaplan. 1984. Cholesterol. Clin. Chem. The C.V. Mosby Co. St Louis. Toronto: Princeton; 437:1194-1206.

Nolen, G.A., C.J. Alexander and N.R. Artman. 1967. Long-term rat feeding study with used frying oil. J. Nutr., 93:337-348.

Paul, S. and G.S. Mittal. 1997. Regulating the use of degraded oil/fat in deep-fat/oil food frying. Crit. Rev. Food Sci. Nutr., 37:635-662.

Paulose, M.M. and S.S. Chang. 1973. Chemical reactions involved in deep fat frying of foods: VI. Characterization of nonvolatile decomposition products of trilinolein. J. Am. Oil Chem., Soc., 50:147-154.

Patton, C.G. and S.R. Crouch. 1977. Enzymatic determination of urea .Anal. Chem., 49 (82): 464 - 469.

Poling, C.E., E. Eagle, E.E. Rice, A.M.A. Durand and M. Fisher. 1970. Long-term responses of "P" rats to heat-treated dietary fats: weight gains, food and energy efficiencies, longevity and histopathology. Lipids 5:128-136.

Reitman, S. and S. Frankel. 1957. Determination of serum glutamate oxaloacetate and glutamate pyruvate trans-aminases. Am. J. Ciln. Path., 28(9):56-60.

Snedecor, G.W. and W.G. Cochran. 1980. Statistical methods. 7th ed. The Iowa state Univ. Press. Ames. Al.

Trinder, P. 1969. Determination of glucose in blood using release and oxidase with an alternative oxygen acceptor. Ann. Clin. Biochem., 6:24-27.

Warnick, G.R., V. Benderson and N. Albers. 1983. Selected methods Clin. Chem., 10: 91-99.

التقييم البيولوجى للمخاطر الناتجة عن التغذية على الزيوت السابق استخدامها فى عمليات القلى العميق

هند عزت عبد القادر- محمد مصطفى عفيفى عامر

سيد سليمان السيد السعدنى- على محمد على الجابى

قسم الكيمياء الحيوية الزراعية ــ كلية الزراعة ــ جامعة الزقازيق

تم استخدام خليط زيوت مكون من ٥٠% زيت الأولين و٣٥% زيت فول الصويا و١٥% زيت عباد الشمس لدراسة تأثير الزيوت السابق الطهى فيها بأى من المعاملات التالية:

١- المجموعة الضابطة (الكنترول السالب) تم فيها التغذية على زيت بدون تسخين.

٢- قلى الطعمية فقط (المجموعة١). ٣-قلى الباذنجان و/أو رقائق البطاطس (المجموعة ٢).

٤- تسخين الزيت بدون أى إضافات (المجموعة ٣ أو الكنترول الموجب).

ولمحاكاة ما هو حادث في المطاعم بحيث تم تسخين خليط الزيوت على درجة حرارة ٢٠٠±٥°م بمعدل٦ ساعات يومياً لمدة أربعة أيام متتالية.

تم تجهيز علائق لتغذية فئران التجارب البيضاء عليها لمدة ٦ أسابيع وذلك بعد فترة أقلمة لمدة أسبوع على عليقة قياسية جهزت من مكونات قياسية يمثل فيها الزيت نسبة ١٠% وتم استخدام الزيوت السابق الإشارة إليها وفى نهاية التجربة تم تقدير معدل التغير فى الوزن لجميع المعاملات، ثم أخذت عينات من الدم ثم فصل السيرم منها، وأجرى تقدير لنسبة الجلوكوز فى الدم ودراسة لمكونات الدهون بالدم والمتمثلة في الدهون الكلية والكولسترول الكلى والجلسريدات الثلاثية، وتمت دراسة الليبوبروتينات عالية الكثافة ومنخفضة الكثافة، كما تمت دراسة تأثير التغذية على وظائف كلا من الكبد والكلى.

وقد تبين من الدراسة حدوث تأثير حاد على كلا من وظائف الكبد والكلى وارتفاع نسبة الكولسترول الكلى والجلسريدات الثلاثية والليبوبروتينات منخفضة الكثافة وانخفاض نسبة الليبوبروتينات مرتفعة الكثافة.

ومما سبق نوصى بعدم تكرار القلى فى الزيوت لمرات متكررة فى عمليات التغذية.