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Liquid Chicken Oil Could Be a Healthy Dietary Oil

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Abstract: Liquid chicken oil is similar to the human lipid ratio, and is similar to the ideal fatty acids ratio suggested by Hayes, but its benefits remain unclear (Hwang, K.N.; Tung, H.P.; Shaw, H.M. J. Oleo. Sci. 69, 199-206 (2020)). Using soybean oil as a control, liquid chicken oil, coconut oil, lard oil, and olive oil, were tested on SD rats with the rodent diet 5001 plus 1% of high cholesterol addition and moderate 10 % of test oils. Positive results showed that a 10% liquid chicken oil diet reduced LDL and triglycerides, atherogenic index while increasing superoxide dismutase more than the soybean oil control ($0.05 \leqq p < 0.10$). Moreover, increment of hepatic endogenous glutathione peroxidase was found to be significantly different from the soybean oil control ($p < 0.05$). In this study, liquid chicken oil had more benefits than vegetable soybean dietary oil, with little evidence of hyperlipidemia. Comparison of the test oils with categories of fatty acids to the idea ratio SFA : MUFA : PUFA = 1 : 1.5 : 1, scored by its average weight implied a parallel trend of lipidemia and hepatic antioxidant activity to its score. It is difficult to use the test of rat to reflect human physiology, it remain 19% different of the fatty acids ratio from human ratio, however, this study reveal that the healthiness of a dietary oil seems relate well to its compatibility to the idea ratio or the host oil ratio, in this case, it is the human ratio.

Key words: liquid chicken oil, ideal oil ratio, glutathione peroxidase, rats

1 Introduction

Dietary lipids regulate plasma cholesterol levels in animals and humans, and hypercholesterolemia is associated with the risk of CVD. Saturated fats withstand higher heat and last longer than most vegetable oils, but have adverse effects on human lipoprotein patterns[1-4], animal fats with large proportions of saturated fat, such as lard and tallow, are generally disfavored as dietary oils for health reasons. Ironically, the most popular oils, sunflower and olive oil, are known to have few n-3 essential fatty acids, and the saturated fatty acids in coconut oil show little or no difference with the unsaturated fatty acids in olive oil, in trial[5].

Knobbe and Chapman recently showed that modern commercial products, including vegetable oil for dietary use, are one of the primary and proximate causes of age-related macular degeneration, and although our ancestors digested mostly animal oils, few modern diseases appeared until the modern 20th century[6, 7]. It seems that not all saturated fatty acids rich oils or animal oils are detrimental to human health.

Chicken oil, which as an animal fat has been largely commercially abandoned, is usually used only as forage rather than as dietary oil for cooking or baking. Ahmad showed the most dominant fatty acid extracted from chicken adipose tissue is oleic acid (43.9%)[8], yet the most abundant oleic acid in olive oil, effectively lowers LDL-C levels in normal triglyceridemic patients[9]. Moreover, a systematic review showed that a high amount (>12%) of MUFA in dietary regimens has the effect of reducing body fat and regulating blood pressure, which helps prevention of cardiovascular disease[10].

We have previously reported that liquid chicken oil (LK) (SFA : MUFA : PUFA = 1 : 1.6 : 0.9), harvested from cold recrystallization of crude chicken oil (SFA : MUFA : PUFA = 1: 1.4 : 0.5), enriched with MUFA (43.5% C18:1) and PUFA (21.1% C18:2, 0.5% C18:3(n-3), 0.2% C18:3(n-6)), has a unique property of conforming to Hayes' suggested ideal oil ratio (SFA : MUFA : PUFA = 1 : 1.5 : 1) which believed to alleviate cardiovascular disease[11]. This LK rich in MUFA and PUFA n-3 and n-6 but contained less saturated fatty acid then general chicken oil. It not only mimicked

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the ideal fatty acid ratio but was also very compatible with human lipids (SFA : MUFA : PUFA = 1 : 1.8 : 0.6), and was found to be the highest scored oil beyond camellia or olive oil on the base of the ideal oil ratio[12]. However, there has been little research on physiological activities of chicken oil and its relationship to lipid metabolism. In order to interpret the benefits of this liquid chicken oil, tests to investigate the effect of plasma lipids metabolism compared with other oils, and the hepatic antioxidant activities in SD rats were carried out.

2 Materials and Methods

2.1 Animals and diets

Male Sprague Dawley (SD) rats purchased from BioLASCO Co. Taipei, Taiwan, were housed individually in stainless-steel wire cages at 23 ± 2℃, with a controlled 12-hr light/dark cycle, and given free access to water and food. Protocols for animal care and handling were in accordance with guidelines of the Institutional Animal Care and Use Committee of Chia-Nan University of Pharmacy and Science.

The liquid chicken oil used was provided by Find Health Co. Ltd., Taiwan. The soybean oil from the President Co. Ltd., Taiwan. The lard from the I Mei food Co. Ltd., Taiwan. The olive oil from the self-brand of Carrefour Co. Ltd., Taiwan, and the coconut oil from the Kirkland Co. Ltd., Philippine. All test diets were prepared by blending the oils with the basic diet Laboratory Rodent diet 5001 (Lab Supply Co. Ltd., Ford Worth, Texas, USA), which contained 5% fat, and an additional 1% cholesterol added. The rats were randomly divided into six groups: a control group fed soybean oil (SC, 10% soybean oil), a liquid chicken oil group (QK, 10% liquid chicken oil), a lard oil group (LD, 10% lard oil), an olive oil group (OL, 10% olive oil), and a coconut oil group (CO, 10% coconut oil), each group with at least six rats, respectively. Body weight and food intake were routinely monitored and recorded during the 13-week test period.

2.2 Tissue preparation

On the last day of the 13-week feeding program, the rats were fasted overnight, and then sacrificed by carbon dioxide asphyxiation. Blood was collected from the abdominal vena cava, the liver, heart, kidney, spleen and testis, and the epididymis adipose tissue and peritoneal adipose tissue were collected and weighed. The EDTA-spiked blood was centrifuged with 1,000 x g to collect the plasma, and stored at −20℃ until analysis. One lobe of each liver was stored at −70℃ for the following analysis.

2.3 Plasma biochemical analysis

Plasma biochemical markers were assayed by an automated chemical analyzer, AU5800, Beckman Coulter Inc., U.S.A. in brief; Plasma cholesterol was assayed by testing saponified cholesterol extracts using dehydrogenase[13], with a CHOL cholesterol kit from Beckman Coulter, Inc., La Brea, CA. Triglycerides were assayed by a coupled enzymatic reactions method[14], using a TRIG triglyceride kit, from Beckman Coulter, Inc., La Brea, CA. High density lipoprotein HDL was assayed in two distinct phases, measured bichromatically at 600/700 nm[15], by an HDL-cholesterol kit from Beckman Coulter, Inc., La Brea, CA. Low density lipoprotein-LDL was also assayed in two phases, measured bichromatically at 540/660 nm[16], using a low density lipoprotein kit from Beckman Coulter, Inc., La Brea, CA. The atherogenic index (AI) was derived by AI = (Chol-HDL)/HDL as Amani suggests[17]. AST was assayed by aminotransferase catalyzation of the transamination of aspartate and α-oxoglutarate, and then measuring the NADH consumption at 340 nm directly[18], using an AST-kit from Beckman Coulter, Inc., La Brea, CA. ALT was assayed by ALT transference of the amino group from alanine to α-oxoglutarate to form pyruvate and glutamate, and then measuring the consumption of NADH at 340 nm and calculate[19, 20], using an ALT-kit from Beckman Coulter, Inc., La Brea, CA.

2.4 Hepatic antioxidant analysis

Glutathione peroxidase was assayed by glutathione peroxidase (GPX) catalyzing the oxidation of glutathione by cumene hydroperoxide[21], using a Glutathione Peroxidase kit from RANSEL RANDOX Laboratories Ltd., Antrim, UK. Superoxide dismutase (SOD) activity in liver was measured by employing xanthine and xanthine oxidase to generate superoxide radicals, which react with 2-(4-iodophenyl) ω-(4-nitrophenol)-5-phenyltetrazolium chloride to form the red dye formazan[22]. This used a SD-25 kit, Superoxide Dismutase kit, from RANSOD RANDOX Laboratories Ltd., Antrim, UK.

The catalase activity was evaluated by enzyme catalase reaction with H_2O_2 in methanol to aldehyde that reacts with a chromogen 4-amino-3-hydrazino-5-mercapto-1,2,4 triazole, forming a purple color that was measured colorimetrically[23], using a catalase assay kit from Cayman Chemical Company, Ann Arbor, MI. Malondialdehyde (MDA) in the liver was measured by reacting malondialdehyde with thiobarbituruc acid (TBA), forming a colorimetric MDA-TBA adduct (530-540 nm) and measured[24], using a commercial TBARS–assay kit from Cayman Chemical Company, Ann Arbor, MI.

2.5 Lipid analysis

The rats' peritoneal adipose was collected, stewed at 125℃ for 15 min using Imarflex electronic pressure cooker, Imarflex Co. Ltd., Taiwan, removed of debris, washed with water, and vacuum dried as a rat oil. Fatty acids of the oil

were analyzed according to the Taiwan FDA method, where Glycerolipids (40 mg) were mixed with 2 mL of hexane and 0.2 mL of 2 M methanolic KOH at room temperature for 2 min. The fatty acid methyl esters in the hexane layer were analyzed by gas chromatography, using an HP-88 column (Agilent, 100 m × 0.25 mm I.D., 0.2 μm film thickness) with a FID detector, and a Shimatzu GC Model-2010[25].

2.6 Statistical analysis

All statistical calculations were done with SPSS ver. 12.0 (SPSS Inc., Chicago, IL, USA) and results are shown as mean ± SD. One-way analysis of variance used $p<0.05$ as statistically significant and Tukey-Kramer was used as post-hoc test.

3 Results

3.1 Animal body weight, food intake and organs

The rats' body weight, food intake, body weight gain, and feeding efficiency in Table 1 show insignificant differences among groups in these categories ($p>0.05$). Relative organ weights are shown in Table 2, with differences between groups not reaching significance ($p>0.05$). These results indicate that the efficiency of energy or nutrition usage of LK was similar to soybean and other commercial oils used.

3.2 Plasma lipids

Table 3 show no significant difference between groups was found for plasma glucose, cholesterol and triglyceride. Glucose is slightly elevated higher but still within the normal range[26]. ALT and LDL are also slightly elevated, but not substantially above the normal range[27–29]. CO had a significant negative effect on TG regulation ($p<0.05$), and the 14% reduction of TG level in QK group was insignificant ($p=0.095$), showing that the QK had some positive effect on TG regulation. Neither HDL-C nor LDL-C showed significant difference from SC, but QK reduced 21% of the LDL compared to SC ($p=0.077$). In addition, the atherogenic index (AI) of QK and OL were 23% and 14% ($p=0.091$) lower than SC, respectively. Thus QK showed a potentially significant effect in reducing atherogenic risk or obesity-related syndromes.

3.3 Antioxidant activities of enzymes in the liver

Table 4 shows there was no significant difference in SOD activity among groups, but the SOD activity of QK was 19% higher than that of SC ($p=0.079$). The activity of GPX in QK was significantly 29% higher than SC ($p<0.05$). Histologic examination of non-alcoholic fatty liver disease activity by Balloon Score found a few rat livers with slight lymphocyte infiltration in some lobular parenchyma, especially in LD. But the average scored steatosis on each group was zero (data not shown)[30].

Table 1 Initial body weight, final body weight and feeding efficiency of rats fed with the test oils for 13 weeks show no significant differences between test oils ($p>0.05$).

	SC	LD	QK	OL	CO
Initial body weight (*g*)	122 ± 13	125 ± 16	126 ± 15	122 ± 15	126 ± 12
Final body weight (*g*)	539 ± 46	544 ± 36	561 ± 68	536 ± 28	517 ± 59
Daily weight gain (*g/day*)	7.21 ± 0.74	7.65 ± 0.63	8.12 ± 2.14	7.45 ± 0.77	6.86 ± 1.09
Daily food intake (*g/day*)	28.01 ± 4.87	29.02 ± 3.81	30.48 ± 2.04	29.14 ± 2.88	28.62 ± 4.51
Feed efficiency (*%*)	26.66 ± 7.62	26.78 ± 4.49	26.52 ± 5.29	25.94 ± 3.04	24.04 ± 2.49

Table 2 Relative organ weight in rats fed with different test diet for 13 weeks, showing no significant differences between test oils ($p>0.05$).

	SC	LD	QK	OL	CO
			(%)		
Liver	5.32 ± 0.92	5.09 ± 0.84	5.07 ± 0.58	5.27 ± 0.43	5.34 ± 0.56
Heart	0.272 ± 0.016	0.281 ± 0.028	0.280 ± 0.040	0.279 ± 0.014	0.276 ± 0.024
Spleen	0.208 ± 0.046	0.196 ± 0.071	0.173 ± 0.035	0.195 ± 0.040	0.180 ± 0.040
Kidney	0.689 ± 0.067	0.702 ± 0.043	0.703 ± 0.034	0.707 ± 0.150	0.703 ± 0.016
Testis	0.598 ± 0.050	0.636 ± 0.073	0.592 ± 0.096	0.618 ± 0.077	0.637 ± 0.095
Epidydimal adipose	1.62 ± 0.52	1.68 ± 0.19	2.06 ± 0.39	2.00 ± 0.40	1.80 ± 0.20
Peritoneal adipose	1.68 ± 0.35	1.72 ± 0.34	2.04 ± 0.41	2.11 ± 0.52	1.74 ± 0.46

Table 3 Plasma enzyme activities and glucose, lipids levels in rats fed with different test diets. The CO was worst on TG regulation ($p < 0.05$), reductions of TG in the QK group are very positive although still insignificant, but AI was significantly reduced in QK.

	SC	LD	QK	OL	CO
AST (*U/L*)	67 ± 27^{a}	81 ± 35^{a}	72 ± 39^{a}	66 ± 19^{a}	64 ± 29^{a}
ALT (*U/L*)	87 ± 32^{a}	91 ± 33^{a}	88 ± 27^{a}	84 ± 35^{a}	94 ± 34^{a}
Glucose (*mg/dl*)	117 ± 41^{a}	129 ± 42^{a}	101 ± 37^{a}	95 ± 49^{a}	113 ± 59^{a}
Cholesterol (*mg/dl*)	68 ± 15^{a}	81 ± 26^{a}	71 ± 15^{a}	67 ± 11^{a}	80 ± 22^{a}
Triglyceride (*mg/dl*)	29 ± 7^{b}	33 ± 7^{ab}	25 ± 6^{b}	29 ± 3^{b}	42 ± 11^{a}
HDL-C (*mg/dL*)	20.2 ± 6.9^{a}	24.8 ± 7.1^{a}	27.1 ± 7.2^{a}	23.5 ± 4.6^{a}	20.0 ± 6.7^{a}
LDL-C (*mg/dL*)	19.7 ± 9.1^{a}	19.0 ± 6.9^{a}	13.8 ± 6.4^{a}	17.5 ± 7.4^{a}	18.2 ± 8.7^{a}
AI	2.3 ± 0.6^{a}	2.3 ± 0.8^{a}	1.7 ± 0.5^{a}	1.9 ± 0.7^{a}	2.8 ± 0.8^{a}

Each value is the mean ± SD (n=6). Values not sharing common superscript in the same row are significantly different from one another among the five groups by one-way ANOVA and Tukey-Kramer test ($p < 0.05$).

Table 4 Activities of antioxidant enzyme and levels of thiobarbituric acid-reacting substances (TBARS) in livers of rats fed with various dietary oils.

	SC	LD	QK	OL	CO
SOD (U/mg protein)	34.1 ± 2.8^{a}	34.0 ± 8.7^{a}	40.6 ± 5.6^{a}	36.6 ± 7.0^{a}	30.6 ± 5.4^{a}
GPX (U/mg protein)	5.62 ± 0.50^{b}	5.28 ± 0.69^{b}	7.27 ± 0.49^{a}	5.26 ± 0.61^{b}	5.12 ± 0.36^{b}
Catalase (U/mg protein)	53.3 ± 22.4^{a}	36.1 ± 24.6^{a}	46.8 ± 19.9^{a}	59.9 ± 27.2^{a}	39.4 ± 27.7^{a}
TBARS (umol/g protein)	2.40 ± 0.07^{a}	2.56 ± 0.98^{a}	2.17 ± 0.84^{a}	2.40 ± 0.54^{a}	2.38 ± 0.52^{a}

Each value is the mean ± SD (n=6). Values not sharing common superscript in the same row are significantly different from one another among the five groups by one-way ANOVA and Tukey-Kramer test ($p < 0.05$).

3.4 Fatty acids analysis

Composition of the SD rats' fatty acids is shown in Table 5. The rats' fatty acid ratio and their weighting scores are compared with the Hayes ratio and the average ideal ratio of Hayes, Monica, and human ratios[12]. The results of Table 6 show that the rats' fatty acid ratio differed from the ideal fatty acid ratio by 18% (SFA : MUFA : PUFA = 1 : 1.5 : 1), and was 19% different from the human oil ratio of 1 : 1.8 : 0.6.

4 Discussion

As a macronutrient, fat is the densest source of food energy. Though the use of liquid chicken oil for human consumption is no longer common, Marx reported that the digestibility of poultry fat could be as high as 100% in dogs[31]. But we found no reports on the effects of chicken oil on plasma lipids, and its other possible health benefits remain unknown.

All rats fed with liquid chicken oil diet remained healthy during the study, with no significant differences in body weight or feeding efficiency did, compared to rats on a soybean oil diet. Table 3 shows that most of the tested values remain normal, there were insignificant differences between groups, and the ALT and Glu are higher among groups, indicating the possibility of hepatic inflammation or insulin resistance but within the normal range[26, 31, 32].

Due to the wide variation in the normal range between reports, we surveyed all rats and noticed an occasional small lymphocyte infiltration on a few liver lobes, especially in LD. But the average score for fatty liver on the balloon test remained 0, indicating that under this moderately higher fat and cholesterol diet the tested fat seemed reach a threshold to trigger the non-alcoholic fatty liver syndrome, but values within this moderately high fat and cholesterol remained safe.

On the other hand, when rats were fed with the diet for 13 weeks the LDL was higher, perhaps due to the additional 1% cholesterol in the diet, but an insignificant difference remained between groups[28]. Although insignificant, QK showed a reduction of 21% in LDL over SC, consistent with many reports on benefits of oleic acid in LK (41%) to reduce the level of LDL[33–35]. Moreover, if the index of ath-

Table 5 Average of fatty acid composition of SD rats peritoneal adipose (n=4). Comparison of the SD rat's fatty acids ratio to human was as previously reported (S : M : P = 1 : 1.8 : 0.6)[12, 38].

Fatty acids	SD rat
	(%)
Saturated fatty acid	30.9
Lauric a.	0.1
Myristic a.	1.2
Palmitic a.	24.6
Heptadecanoic acid	0.2
Stearic a.	4.6
Arachidic a.	0.1
tetrasaenoic acid	0.1
Monounsaturated fatty acid	43.1
Myristoleic a.	0
Palmotoleic a.	4.8
Heptadecenoic acid	0.2
Oleic a.	37.3
Palunic acid	0.4
Ecosaenoic a.	0.4
Erucic acid	0.1
Polyunsaturated fatty acid	23.7
Linoleic a.	22.6
γ-linolenic a.	0.2
α-linolenic a.	0.9
Ecosadienoic a.	0
Arachidonic a.	0
Ecosatetraenoic acid	0
Adrenic acid	0
Docosapetaenoic acid	0
Docosahexaenoic acid	0
S:M:P	1 : 1.4 : 0.7

erogenicity (IA) was evaluated by its composition of fatty acids, as Ulbritcht and Southgate proposed; IA = [C12:0 + (C14:0 x 4) + C16:0]/Σ UFA, the IA of SC is 0.14, then the LD is 0.46, the QK is 0.35, the OL is 0.13, and the CO is 8.11[36], showing that olive oil is the best oil, with the lowest IA. However, if the differences was evaluated by its atherogenic index (AI) as Amani suggested; [AI = (Chol-HDL)/HDL][17], then QK lowered AI by 26% over SC ($p > 0.05$) and lowered by 40% over CO ($p > 0.05$). Further exploration of the relationship between IA and AI requires further research, but QK performed significantly better AI than SC or better than even OL.

Soybean oil as the control in this study, has high levels of polyunsaturated fatty acids (PUFA), which reduce the plasma cholesterol and reduce the incidence of CVD[37]. This study shows that the liquid chicken oil enriched with n-3 and oleic acid has a substantial benefit on lipid profile and AI, reducing levels to those of vegetable soybean oil better.

It is known that dietary lipid determines the structure and fatty acid composition of cell membranes, and the cellular susceptibility to peroxidation is dependent on its polyunsaturated fatty acid (PUFA) content and antioxidant status[38]. In addition, monounsaturated fatty acid is more stable against oxidation than PUFA. In this study, the liver MDA or the TBARS of soybean oil diet were not elevated as expected. This may be because previous tests were done with an abnormal AIN-76 diet, or high chemical carcinogen stress and, as such, may not reflect the normal nutrition physiology of commercial oils[38, 39]. On the other hand, GPx, SOD, and catalase are well-known as cellular antioxidant enzymes. The changes in enzyme activity show its adaptation to oxidative stress[40–42]. This study finds a 12% decrease in catalase activity in normal rats fed with liquid chicken oil, while there was no increase in TBARS. This indicates that MUFA-rich liquid chicken oil may be less susceptible to peroxidation. The GPx and SOD activity were observed to be both elevated on QK (see Table 4). Moreover, the hepatic GPx activity was significant and 30% higher in QK than in SC. This may be due to the photosensitive yellow color of chicken oil, since chicken oil has high levels of vitamin A or beta-carotene antioxidant[43, 44]. However, the antioxidant activity shown in this study was an endogenous enzyme, so our cold-harvested LK had some factors in addition to vitamin E or carotene that regulated hepatic antioxidant activity.

The effects of olive oil in the well-known Mediterranean diet are controversial. Abenavoli considered the Mediterranean diet as characterized by mainly plant-based foods and fish, with reduced consumption of meat and dairy products[45]. However, Khaw found little difference between olive oil, butter or coconut oil in trials[5]. Although olive oil is often considered a healthy oil, it has fewer n-3 fatty acids, and our test found the differences in activity of olive oil from soy bean oil to be insignificant, or with only a few positive effects.

On the other hand, Kang has stated that the endogenic hepatic antioxidant enzyme was regulated by a better SFA/PUFA fatty acids ratio 1～1.5[46], and the ratio of 0.9 for LK is similar, supporting LK's positive effect on endogenous hepatic antioxidant and reinforcing our hypothesis that the ideal fatty acid ratio for humans is SFA : MUFA : PUFA = 1 : 1.5 : 1[12]. This may also reinforce the significance of AI and the insignificant but positive effects of SOD of QK in this study. Moreover, because the rat's fatty acid ratio showed

Table 6 Comparison of the scored test oils. Coconut oil has the lowest score, and QK has the highest score among the test oils. Implying a sequential-like relationship between the oil scores for the benefits of hyperlipidemia and hepatic antioxidant activities.

		Hayes ratio 1 : 1.5 : 1		Avg ratio 1 : 1.5 : 0.8		Rat ratio 1 : 1.4 : 0.7	
	S : M : P	3 weights	2 weights	3 weights	2 weights	3 weights	2 weights
SC	1 : 2.9 : 2.5	64	37	61	37	58	33
LD	1 : 1.2 : 0.5	76	61	80	61	85	67
QK	1 : 1.6 : 0.9	94	83	94	83	88	70
OL	1 : 4.1 : 0.5	62	39	66	39	68	41
CO	1 : 0.1 : 0.0	35	3	35	3	36	4

S: saturated fatty acid, M: mono-unsaturated fatty acids, P: poly-unsaturated fatty acids, Avg ratio: the average of Hayes, Monnica, and human ratio. Relative scores were tested by the weighted method[12].

an 18% difference from the ideal oil ratio, and a 19% difference from the human ratio, the results may become better with humans. Table 6 shows that for all ideal ratios evaluated, liquid chicken oil always had the best score[12, 47]. It seems that if there are health defects brought about by the dietary use of animal fat, then liquid chicken oil, should be regarded as an exception.

In conclusion, in this high cholesterol and moderate high fat condition, liquid chicken oil showed benefits similar to vegetable soybean dietary oil in both energy metabolism and feeding efficiency. In addition, QK showed a significant increase of the endogenous hepatic antioxidant GPx, and reduced AI.

This first evaluation of lipid by the host fatty acid composition, together with the relationship of oils scored, implying a sequential-like relationship between the oil scores for the benefits of hyperlipidemia and hepatic antioxidant activities, finds that, being the most similar oil to human lipids. It seems that the best oil for the soy bean plant is the soy oil, the best oil for the olive tree is the olive oil, and the best oil to us, *homo sapiens*, should be the human oil. However, as the dietary use of human oil is impossible, then the most human mimic or compatible lipid, liquid chicken oil could be a good choice for dietary consumption. Our test remain small, further studies are expected. (A part of this study was presented at The Annual Symposium of The Nutrition Society of Taiwan, 2020).

Authors' Contributions

K.-N. Hwang: material preparation, biochemical markers analysis, intelligent properties, logical analysis and manuscript processing; H.P. Tung: chicken oil preparation, anatomy and analysis; Y.H. Lu: fat analysis, antioxidant and data analysis. H.M. Shaw: experimental design, biochemical markers analysis, data processing and manuscript processing.

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