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Effect of Avocado (Persea Americana), Cabbage (Brassica Oleracea) and Ginger (Zingiber Officinale) on Rat Liver and Thyroid Injuries Induced by CCl4 (Carbon Tetrachloride)

Asmaa F. Hamouda[1, 2], Manal Y. Sameeh[2] and Randa M. Shrourou[2, 3]

1. Department of biochemistry, University of Alexandria, Alexandria 21111, Egypt

2. Department of chemistry, University of Al Leith- Umm Al-Qura, Mecca, 21955, Saudi Arabia

3. Department of Food Nutrition, University of King Abdul-Aziz, Jeddah 21589 Saudi Arabia

Abstract: Avocado, Cabbage, and Ginger are a part of a regular human diet and have antioxidant, and antitumor effects. The effect of AVOE (avocado), GE (Ginger) and CE (Cabbage) extracts separately on liver NO (nitric oxide), MDA (malondialdehyde), as well as serum AST (aspartate aminotransferase), ALT (alanine aminotransferase), total bilirubin, TC (total cholesterol), T.G (triglyceride), HDL cholesterol (high-density lipoprotein), LDL cholesterol (low-density lipoprotein), TSH (thyroid-stimulating hormone), T3 (Triiodothyronine), T4 (Thyroxine) in rats treated and untreated with CCl4 (carbon tetrachloride) was studied. The levels of NO, MDA, as well as serum AST, ALT, total bilirubin, TC, T.G, LDL, and TSH, showed an elevation while, HDL, T3 and T4 showed the decline in rats treated with CCl4 as compared to control. Treatment of rats with AVOE and GE pre, during, and post CCl4 administration improve NO, MDA, as well as serum AST, ALT, total bilirubin, TC, T.G, HDL, LDL, TSH, T3, T4 as compared to CCl4. Treatment of rats with CE pre, during, and post CCl4 administration did not improve in the thyroid hormones and lipid profile levels as compared to CCl4. These findings suggest that avocado and ginger treatment exerts a protective effect on metabolic disorders by decreasing oxidative stress.

Key words: Liver injuries, CCl4 (carbon tetrachloride), avocado (Persea Americana), cabbage (Brassica Oleracea) and ginger (Zingiber Officinale), thyroid function.

1. Introduction

The liver has a useful role in the metabolism of thyroid hormone including conjugation, excretion, and peripheral deiodination and in the synthesis of TBG (thyroxine-binding globulin) [1, 2]. The level of thyroid hormones is also important to normal hepatic function and bilirubin metabolism, therefore, defect any role led to many serious diseases including obesity [1-3]. Carbon tetrachloride is colorless liquid used as a dry-cleaning agent, a solvent in chemical synthesis, chlorofluorocarbon production, and causes many diseases including liver damage with an indirect effect on serum levels of thyroid hormones [4-8]. CCl4 (carbon tetrachloride) metabolized in cytochrome P450 and induced reactive free radicals and initiates cell damage through oxidative stress [3-5].

Natural products are more effective treatment strategies for drug discovery and clinical therapy. Avocado (Persea Americana) has health benefits effects in wound healing, psoriasis, wrinkles, stretch marks, scleroderma, hepatic injuries, stroke prevention, obesity, and cancer. The health benefits effect of fatty avocado fruit may be due to its content. It included essential nutrients, protein source, fiber source monounsaturated (oleic acid) and polyunsaturated fats

Corresponding author: Asmaa F. Hamouda, Ph.D., assistant professor in the Umm Al-Qura University in Mecca in Saudi Arabia, research field: biochemistry.

linoleic and linolenic acids, polyphenols, proanthocyanidins, tocopherols, carotenoids, β-carotene, lecithin, minerals, and vitamins A, C, D, and E) [9-11]. Previous studies have showed that it reduces the risk of diabetes, control weight, normalizes blood cholesterol levels, and involved in liver metabolism [12]. Zingiber officinale Roscoe, ginger (rhizome), is used worldwide as a spice in cooking, and its chemical contents including zingerone, shogaols, and gingerols, that have medical importance in treating nausea and others [13-16]. Cabbage (Brassica oleracea) of the family Brassicaceae or Cruciferacea that contain goitrogenic is a part of the regular human diet, used as a green leafy vegetable. Cabbage vegetable use for controlling many metabolic diseases such as diabetes [17, 18].

The target of this study stresses to 1- Illustrated the effect of CCl4 (carbon tetrachloride) and on normal liver and thyroid. 2- Study the chemoprevention of avocado (AVOE), GE (Ginger) and CE (Cabbage) extracts separately against CCl4. 3- Study the effect of (AVOE), (GE) and (CE) extracts separately on normal liver and thyroid. The studies included the changes in liver NO (nitric oxide), MDA (malondialdehyde), as well as serum AST (aspartate aminotransferase), ALT (alanine aminotransferase), total bilirubin, TC (total cholesterol), T.G (triglyceride), HDL cholesterol (high-density lipoprotein), LDL cholesterol (low-density lipoprotein), TSH (thyroid-stimulating hormone), T3 (Triiodothyronine), T4 (Thyroxine).

2. Experimental Procedures

Sulphanilamide, N-1-Napthyl ethylene diamine, Standard sodium nitrite, and SDS (sodium dodecyle sulfate), CCl4 (carbon tetrachloride), TBA (thiobarbituric acid), TMP (tetramethoxypropan) and Diethylene triaminopentaacetic acid purchased from Sigma-Aldrich, USA, (DTPA).

2.1 AVOE (Avocado Extract)

The cured oil obtained from avocado purchased from a local market (Saudi Arabia).When edible maturity ripe had been reached [19], the avocados were washed and peeled and the seed removed. Subsequently, the pulp was homogenized using the electric blender with water. The obtained viscous slurry-like pulp was Stored at 4 ℃until used as orally administered to the rats [20].

2.2 Ginger (Zingiber Officinale)

The ginger plant material (GE) purchased local market (Saudi Arabia). The plant peeled, and ground with a mechanical grinder. The ground plant (500 g) extracted with absolute ethanol and concentrated using a rotary evaporator at 40 -50°C. The dried yield of the extract was 4 g [21].

2.3 The Cabbage (Brassica oleracea)

The CE (cabbage) purchased market, dried and extracted with absolute ethanol. The extract filtrated and dried (yield: 12%) [22].

2.4 Animals

80 adult male Sprague-Dawely rats weighing 100-110 g used in this study. All rats were examined for health status at 25 ^{o}C, given standard diet and water daily for two weeks before handling. After acclimatization, rats were divided into eight groups of ten rats. All animal experiments approved by the Ethics Committee of the Experimental Animal Care Society.

The control Group (C): untreated rats.

(CCl4) Group: Injected rat subcutaneous, (SC) with 3 ml of CCl4 /kg/week for three weeks, [23].

(AVOE) Group: Treated rat orally with a daily dose of AVOE 1 ml/kg bm (body mass) for 35 days [20].

(AVOE- CCl4) Group: Treated rat orally with a daily dose of AVOE 1 ml/kg bm (body mass). Rats were treated with CCl4 (as described before) at the beginning of the second week in addition to a daily dose of AVOE until whole 35 days [20, 23].

(GE) Group: Treated rat orally with a daily dose of GE 30 mg/kg (bm) for 35 days [22].

(GE- CCl4) Group: Treated rat orally with a daily dose of GE 30 mg/kg (bm). Rats treated with CCl4 (as described before) at the beginning of the second week in addition to a daily dose of GE until whole 35 days [14, 23].

(CE) Group: Treated rat orally with a daily dose of CE 500 mg/kg (bm) for 35 days [22].

(CE- CCl4) Group: Treated rat orally with a daily dose of CE 500 mg/kg (bm). Rats treated with CCl4 (as described before), at the beginning of the second week, in addition to a daily dose of CE until whole 35 days [15, 22].

After the experimental term, before rats anesthetized with diethyl ether and sacrificing, feeding stopped 12 hours. The liver tissues washed with a cold saline solution (0.9% NaCl), weighed, and kept at -80 ^{o}C until used for biochemical analysis.

Unheparinized blood samples were collected, maintained at room temperature for 15 min and then sera were separated by centrifugation at 3000 rpm at 2°C for 20 min. Sera stored at -30 C^{0} until used.

2.5 Biochemical Assay

2.4.1. NO Level

It was determined spectrophotometrically [24]. The liver tissues homogenized in four volumes of cell lysate buffer (pH 7.5). The homogenate centrifuged at 10,000 g for one min at 4 ^{0}C, and the supernatant stored at -30 C^{0} until used. 100 µl of sample (liver or standard sodium nitrite 100 µM) added to 1 ml of Sulphanilamide, and mixed. 50 µl N-1-Napthyl ethylene diamine added, and then incubated at room Temp for 20 min. After that the absorbance recorded at 540 nm against blank (since the buffer was added instead of the sample).

2.4.2. Lipid Peroxidation

We used the calorimetric method for MDA level, the end product of lipid peroxidation, determination [25]. Fifty microliters of the liver tissues homogenate or the buffer that used in homogenization process (blank) incubated with assay mixture that included (100 lL of 8.1% of SDS, 750lL of 20% acetic acid containing HCl, 750 lL of 0.8% TBA, and 300 lL of distilled water)pH 3.5. Then the mixture put in boiling water bath for 45min. At room temperature, after cooling, add 500 lL of distilled water and 2.5mL of n-butanol/pyridine mixture (15:1 v/v), mixed well, and centrifuged for 10 min at 1780 g. Then we measured the absorbance of the pink color at 532 nm and determined the concentration of MDA as nmol/g liver. Then we used different concentrations of TMP (20-300 nmol) as standard and assay in a similar way as the sample.

2.4.3. Liver Function Test

AST and ALT activities and Total bilirubin concentration determined according to the methods of and Frankel, and Reitman (1957) and Jendrassik and Grof (1938), respectively [26, 27].

2.4.4. Lipid Profile

Total cholesterol, LDL-cholesterol, HDL-cholesterol and TG were determined according to the method of Burstein et al., (1970) [28].

2.4.5. Thyroid Functions

Serum TSH (µ IU/ml), T3 (ng/ml), and T4 (µg/dl) were estimated using the standard protocol for commercially available kits by Roche/Hitachi Cobas e 601 analyzers (Roche Diagnostics, Mannheim, Germany) utilizing electrochemiluminescence immunoassay TSH, T3, and T4.

2.4.6. Statistical Analysis

All results expressed as mean ± SD. Differences considered significant at $p < 0.05$. All analyzes were performed using the statistical software SPSS, version 16.

3. Results

3.1 Lipid Peroxidation

MDA levels in control (C) were 1.99 ± 1.12nmol/g lower than that in CCL4 group 19.23 ± 1.06 nmol/g; $p < 0.05$. MDA levels in AVOE group were 2.00 ± 0.16 nmol/g compared to C; $p < 0.05$. MDA levels in AVOE-CCL4 were 2.10 ± 0.17 nmol/g compared to CCL4; $p < 0.05$ (Table.1). MDA levels in CE group

were 1.94 ± 0.08 nmol/g compared to C; $p < 0.05$. MDA levels in CE-CCl4 were 12.10 ± 0.26 nmol/g compared to CCL4; $p < 0.05$. Otherwise, MDA levels in GE group were 1.94 ± 0.98 nmol/g compared to C; $p < 0.05$. MDA levels in GE-CCl4 were 6.00 ± 0.32 nmol/g compared to CCl4 (Table.1).

3.2 NO Level

NO levels in control(C) were 34.03 ±1.89 µm lower than that in CCL4 group 72.66 ± 1.81 µm; $p < 0.05$. NO levels in AVOE group were 33.99 ± 2.01 µm compared to C; $p < 0.05$. NO levels AVOE-CCL4 were 39.22 ± 2.22 µm compared to CCL4; $p < 0.05$. (Table.1) NO levels in CE group were 33.98 ± 2.00 µm compared to C; $p < 0.05$. NO levels CE-CCl4 were 40.22 ± 0.34 µm compared to CCL4; $p < 0.05$. NO levels in GE group were 34.24 ± 1.01µm compared to C; $p < 0.05$. NO levels GE-CCl4 were 30.01 ± 1.00 µm compared to CCL4; $p < 0.05$ (Table.1).

3.3 Liver Function

ALT, AST and bilirubin levels were (0.70 ± 0.06 U/L, 13.41 ± 1.56 U/L , 0.66a ± 0.82 mg/dl) lower than that in CCL4 group (6.55 ± 0.96 U/L, 25.2 ± 2.23 U/L, 1.59 ± 0.06 mg/dl); $p < 0.05$ (Table 1). ALT, AST and bilirubin levels in AVOE-CCL4 were (1.27 ± 0.88 U/L, 15.24 ± 1.82 U/L, 0.99 ± 0.07 mg/dl) compared to CCL4 group $p < 0.05$. ALT, AST and bilirubin levels in AVOE were (0.71 ± 0.17 U/L, 13.44 ± 1.81 U/L, 0.67 ± 0.09 mg/dl) compared to control group $p < 0.05$. (Table 1). ALT, AST and bilirubin levels in CE-CCL4 were (2.34 ± 0.67 U/L, 18.24 ± 0.46 U/L, 1.85 ± 0.22 mg/dl) compared to CCL4 group $p < 0.05$. ALT, AST and bilirubin levels in CE were (0.70 a ± 0.89 U/L, 13.42 a ± 1.21 U/L, 0.67 a ± 1.45 mg/dl) compared to control group $p < 0.05$. (Table 1) ALT, AST and bilirubin levels in GE- CCL4 were (0.78 ± 0.48 U/L, 17.42 ± 0.86 U/L, 1.31± 1.34 mg/dl) compared to CCL4 group $p < 0.05$. ALT, AST and bilirubin levels in GE were (0.71 ± 0.66 U/L, 13.42 ± 1.47 U/L, 0.66 ± 0.86 mg/dl) compared to control group $p < 0.05$.

3.4 Thyroid function

TSH levels in control were (0.13 ± 0.08 µIU/ml) lower than that in CCL4 group (0.19 ± 0.09 µIU/ml) while T3, T4 in control(0.70 ± 1, 24 ng/ml, 29.96 ± 3.41 µg/dl) higher than that in CCL4 group (0.40 ± 2, 25 ng/ml, 12.96 ± 4.45 µ g/dl); $p < 0.05$ (Table 1). T3, T4 and TSH levels in AVOE- CCL4 were (0.66 ± 3, 35 ng/ml, 28.68 ± 3.33 µg/dl, 0.13 ± 0.06 µIU/ml) compared to CCL4 group $p < 0.05$. T3, T4 and TSH levels in AVOE were (0.71 ± 3, 22 ng/ml, 30.06 ± 2.21 µg/dl, 0.12 ± 0.07 µIU/ml) compared to control group $p < 0.05$. (Table 1). T3, T4 and TSH levels in CE- CCL4 were (0.30 ± 1.23 ng/ml, 8.34 ± 1.01 µg/dl, 0.17 ± 0.45 µIU/ml) compared to CCL4 group $p < 0.05$. T3, T4 and TSH levels in CE were (0.70 ± 1.02 ng/ml, 29.19 ± 1.11 µg/dl, 0.13 ± 0.29 µIU/ml) compared to control group $p < 0.05$. (Table 1). T3, T4 and TSH levels in GE-CCL4 were (0.61 ± 0.04 ng/ml, 19.86 ± 1.33 µg/dl, 0.14 ± 0.23 µIU/ml) compared to CCL4 group $p < 0.05$. T3, T4 and TSH levels in GE were (0.70 ± 1.21 ng/ml, 29.98 ± 0.86 µg/dl, 0.13 ± 0.09 µIU/ml) compared to control group $p < 0.05$. (Table 1)

3.5 Lipid Profile

TC, TG and LDL-C levels were (76.22 ± 2.23 mg/dl, 82.23 ± 0.99 mg/dl , 37.14 ± 0.44 mg/dl) lower than that in CCL4 group (250.99 ± 4.02 mg/dl, 176.62 ± 3.23 mg/dl, 171.80 ± 0.92 mg/dl) while HDL-C in C (24.92 ± 1.67 mg/dl) higher than that in CCl4 (13.19 ± 2.46 mg/dl) ; $p < 0.05$ (Table 1). TC, TG, HDL-C and LDL-C levels in AVOE- CCL4 were (99.25 ± 2.27 mg/dl, 90.65 ± 3.01mg/dl, 23.83 ± 2.03 mg/dl, 38.91 ± 0.67 mg/dl) compared to CCL4 group $p < 0.05$. TC, TG, HDL-C and LDL-C levels in AVOE were (75.97 ± 2.76mg/dl, 81.99 ± 2.82 mg/dl, 25.01 ± 2.00 mg/dl, 36.99 ± 0.11 mg/dl) compared to control group $p < 0.05$ (Table 1). TC, TG, HDL-C and LDL-C levels in CE- CCL4 were (155.25 ± 0.65 mg/dl, 194.21 ± 2.15 mg/dl, 11.02 ± 1.02 mg/dl, 123.12 ± 0.23 mg/dl) compared to CCL4 group $p < 0.05$. TC, TG, HDL-C and LDL-C levels in CE were (76.21 ± 1.09mg/dl,

Table 1 Different biochemical analysis.

Particulars	C	CCl4	AVOE-CCl4	AVOE	CE-CCl4	CE	GE-CCl4	GE
Serum								
Total T3 (ng/ml)	0.70 a ± 1,24	0.40 b ± 2,25	0.66 c ± 3,35	0.71 a ± 3,22	0.30 d ± 1.23	0.70 a ± 1.02	0.61 e ± 0.04	0.70 a ± 1.21
Total T4(µ g/dl))	29.96 a ± 3.41	12.96 b ± 4.45	28.68 c ± 3.33	30.06 a ± 2.21	8.34 d ± 1.01	29.19 a ± 1.11	19.86 e ± 1.33	29.98 a ± 0.86
TSH(µ IU/ml)	0.13 a ± 0.08	0.19 b ± 0.09	0.13 c ± 0.06	0.12 a ± 0.07	0.17 d ± 0.45	0.13 a ± 0.29	0.14 e ± 0.23	0.13 a ± 0.09
ALT (U/L)	0.70a ± 0.06	6.55 b ± 0.96	1.27 c± 0.88	0.71 a ± 0.17	2.34 d± 0.67	0.70 a ± 0.89	0.78 e± 0.48	0.71a ± 0.66
AST (U/L)	13.41 a ± 1.56	25.2 b ± 2.23	15.24 c ± 1.82	13.44 a ± 1.81	18.24 d ± 0.46	13.42 a ± 1.21	17.42 e ± 0.86	13.42 a ± 1.47
Bilirubin (mg/dl)	0.66 a± 0.82	1.59 b ± 0.06	0.99 c ± 0.07	0.67 a ± 0.09	1.85 d ± 0.22	0.67 a ± 1.45	1.31e± 1.34	0.66a± 0.86
Total cholesterol (TC) concentration mg/dl)	76.22 a±2.23	250.99 b ±4.02	99.25 c ± 2.27	75.97 a ±2.76	155.25 d ± 0.65	76.21 a ± 1.09	98.53 e ± 2.68	76.22 a ± 1.43
TG mg/dl	82.23 a ± 0.99	176.62 b ± 3.23	90.65 c ± 3.01	81.99 a ±2.82	194.21 d ±2.15	81.99 a ±2.12	99.75 e ±0.27	82.65 a±0.11
HDL-C (mg/dl)	24.92 a ± 1.67	13.19 b ± 2.46	23.83 a ± 2.03	25.01 a ± 2.00	11.02 c ± 1.02	24.45 a ± 1.05	18.22 d ± 2.34	24.45 a ±2.03
LDL-C (mg/dl)	37.14 a ± 0.44	171.80 b ± 0.92	38.91 c ± 0.67	36.99 a ± 0.11	123.12 d ± 0.23	37.15 a ± 0.23	55.23 e ± 0.46	37.14 a ± 0.76
Tissue								
Malondialdehyde (MDA) levels (nmol/g tissue	1.99 a ± 1.12	19.23 b ± 1.06	2.10 a ± 0.17	2.00 a ± 0.16	12.10 c ± 0.26	1.94 a ± 0.08	6.00 d ± 0.32	1.94 a ± 0.98
NO concentration (µm) in LIVER tissues	34.03 a ± 1.89	72.66 b ± 1.81	39.22 c ± 2.22	33.99a ± 2.01	40.22 d ± 0.34	33.98a ± 2.00	30.01 e ± 1.00	34.24a ± 1.01

Results express as mean ± S.D. Values with different letter in each row are significantly different at $p < 0.05$.

81.99 ± 2.12mg/dl, 24.45 ± 1.05mg/dl, 37.15 ± 0.23mg/dl) compared to control group $p < 0.05$(Table 1). TC, TG, HDL-C and LDL-C levels in GE- CCL4 were (98.53 ± 2.68 mg/dl, 99.75 ± 0.27 mg/dl, 18.22 ± 2.34mg/dl, 55.23 ± 0.46 mg/dl) compared to CCL4 group $p < 0.05$. TC, TG, HDL-C and LDL-C levels in GE were (76.22 ± 1.43mg/dl, 82.65 ± 0.11 mg/dl, 24.45 ± 2.03mg/dl, 37.14 ± 0.76mg/dl) compared to control group $p < 0.05$ (Table 1).

4. Discussion

Fatty liver and injuries are a consequence of different causative agents, as alcohol, viral hepatitis, and many other drugs. In an attempt to model this process, chemical induction of fatty liver injuries was initiated by CCl4 administration in different experimental models. The results related to the deleterious effect of CCl4 itself and its metabolites; CCl4 is metabolized by the cytochrome P-450. CCl4 metabolite trichloromethyl free radicals reacted with protein and lipid within the cell; this reaction accelerated with oxygen and yielded trichloromethyl peroxyl radicals, which attack lipids on the membrane of endoplasmic reticulum faster than trichloromethyl free radical disturbed calcium homeostasis and finally cell death [29-31].

In the present study, CCl4 administration significantly increased liver NO (nitric oxide), MDA (malondialdehyde), as well as serum AST (aspartate aminotransferase), ALT (alanine aminotransferase), total bilirubin, TC (total cholesterol), T.G (triglyceride), low density lipoprotein (LDL cholesterol), TSH (thyroid-stimulating hormone) as well as decreased in HDL cholesterol (high density lipoprotein), T3 (Triiodothyronine), T4 (Thyroxine) as compared to control group. This may mean that CCl4 induced lipid peroxidation and liver injuries in turn affected thyroid function.

In current work increase in AST, ALT, TC, MDA and NO level after administration with CCl4 indicated that CCl4 is induced oxidative stress that characterized by an imbalance between pro-oxidant and antioxidant in the favor of the pro-oxidants that led to tissue injuries including liver [32]. Administration of CCl4 is induced free radical formation resulted in hepatocytes injuries and leakage of cytosolic contents, and this led to an elevation in the activities of serum AST, ALT, and bilirubin level compared to the controls.

The present results showed that CCl4 administration significantly increased serum TC, TG, and LDL as well as decreased HDL levels. These findings agree with previous results that found that CCl4 treatment significantly increased liver tissue weight, total lipid, TC, TG where CCl4 is induced fatty liver where lipogenesis is produced [30].

Increased in TSH as well as decreased in T3 and T4 after administration with CCl4 confirmed by previous work where found that there are several abnormalities of thyroid function tests in patients with chronic liver diseases [33]. The liver play vital role in thyroid hormone metabolism consequences, the defect in liver function led to a defect in thyroid function [34]. Other investigator found that Treatment with CCl4 significantly reduced the levels of T3 and T4 and increased TSH levels as well as induced oxidative stress in the thyroid tissue of rats [3, 34].

Some investigators found that avocado has selective roles in their effects [9, 35, 36]. Our result where, treatment with AVOE before during and after CCl4 administration showed that, decreased in liver NO, MDA, as well as serum AST, ALT, total bilirubin, total TC, T.G, LDL cholesterol, TSH and increased in HDL cholesterol, T3, T4 as compared to CCl4 group, while there are no significant change in the levels of MDA, NO as well as thyroid and liver function and lipid profile levels in AVOE group as compared with control group. These results suggest that AVOE can protect liver and thyroid tissue against oxidative damage, possibly through the antioxidant effects of its bioactive compounds.

T4 (Thyroxine), T3 (triiodothyronine) hormones and calcitonin secreted by the thyroid gland into blood, and

regulation of the body's metabolism, regulate heart rate, and body temperature and control the energy that used by the body from food. Calcitonin helps monitor the level of calcium in the blood [37, 38]. Avocado contains selenium, zinc, copper, iron, antioxidants, vitamin A, C, D, E, K and the B vitamins as well as tyrosine and iodine [39-42] which can boost thyroid function [43]. Selenium converts T4 (thyroxine) into the most accessible form of thyroid hormone, T3 (triiodothyronine) and maintains the correct amount of thyroid hormones in body tissues and blood, including the liver and thyroid gland as well as recycles its iodine stores. Zinc supplementing can reverse hypothyroidism, and Copper is needed to produce T4 to keeps the body's cholesterol synthesis on track. Iron-rich foods such as avocado can change iron deficiency anemia that is common in hypothyroidism. Vitamins A, C, and E, along with selenium and iodine, are important antioxidants to neutralize the oxidative stress in the thyroid gland. The B vitamins such as B2, B3, and B6 are essential for thyroid function because they involved in manufacturing T4. Tyrosine, an amino acid, is a building block of protein that required producing thyroid hormones from iodine [44-48]. T3 and T4, the thyroid hormones, enter all cells and bind to a nuclear T3 receptor such as retinoic acid, retinol and, vitamin D receptors, and this may be the role of vitamins in thyroid activation [38].

The treatment with GE before during and after CCl4 administration showed that, decreased in liver NO, MDA, as well as serum AST, ALT, total bilirubin, total TC, T.G, LDL cholesterol, TSH and increased in HDL cholesterol, T3, T4 as compared to CCl4 group, while there are no significant changes in the levels of MDA, NO as well as thyroid and liver function and lipid profile levels in GE group as compared with control group. These results suggest that GE can protect liver and thyroid tissue against oxidative damage, possibly through the antioxidant effects of its bioactive compounds. That included gingerols, shogaols, phenolic ketone derivatives, monoterpens, and sesquiterpens. Ginger extract has antioxidative properties and scavenges superoxide anion and hydroxyl radicals [49-51]. Previous studies demonstrate that ginger possesses hypoglycemic, hypocholesterolemic and hypolipidemic, causes a decrease in lipid peroxidation and an increase in plasma antioxidant capacity [49].

Ginger extracts have many pharmacological effects such as anti-platelet, anti-oxidant, anti-tumour, anti-rhinoviral, antihepatotoxicity and anti-arthritic effect [52-54]. It can relieve any problem such as cataract, rheumatism, neurological disorder, gingivitis, toothache, asthma, stroke, constipation, and diabetes [55]and affect serum alkaline phosphatase in adult male rats [55, 56]. Other observation illustrated that ginger has autoimmune effect as an antigen known as subacute thyroiditis, which changes antigenic properties of thyroid follicular cells, there are other allergic reactions to ginger are reported as skin rash [57].

Based on these observations, our result illustrates that protective effect of avocado on thyroid function more than ginger, this may be the chemical competent of avocado especially monounsaturated fats, tyrosine, and iodine. Many reporters study the effects of an avocado diet as antioxidant, anti-inflammatory, anti-lipid effect through effect in total cholesterol LDL and HDL as well as thyroid function [37, 58-62]. We are not surprising because there are many encouraging and dynamic effects of MUFA on blood pressure, insulin resistance, coagulation systems, fat metabolism and body fat distribution, that makes the monounsaturated fats, the most adequate for nutritional recommendations.

Otherwise, the treatment with CE before, during and after CCl4 administration showed that, decreased in liver NO, MDA, as well as serum AST, ALT, total bilirubin, as compared to total CCl4. The treatment with CE also before, during and after CCl4 administration showed that an increases in TC, T.G, LDL cholesterol, and TSH and decrease in HDL

cholesterol, T3, T4 as compared to CCl4, while there are no significant differences in the levels of MDA, NO as well as thyroid and liver function and lipid profile levels in CE group as compared with control group. These result indicated that Cabbage (Brassica oleracea) increase thyroid injuries in the presence of CCl4 more than the CCl4 group; this may be due to an effect of its contents that reflect a significant drop in both T3 and T4 concentrations as compared to CCl4. Other studies agree with our results that reported that thioglucosides are the most common goitrogens, which found in Brassica families, such as cabbage, cauliflower, broccoli, and turnip. On hydrolysis, thioglucosides yield thiocyanates and isothiocyanates [63] which inhibit the selective concentration of iodine by the thyroid [64]. The most studied factors relating to iodine bioavailability are the goitrogens, but these only have a significant impact on IDD (Iodine deficiency disorders) when the usual dietary intake of iodine is low. Goitrogens can reduce the levels of iodine uptake by the thyroid, or impair its metabolism [63, 64]. Other investigators reported that goitrogens in foodstuffs might contribute to great significance in explaining the high incidence of goiter in many areas, in the world. A goitrogenic potency of some vegetables and their effect on thyroid function and an appreciable increase in thyroid weight to body weight ratio may be due to of present thiocyanate in the blood of rats [65]. Our study showed that cabbage showed a beneficial effect on lipid peroxidation and liver function that agree with previous results, which reported that many beneficial roles in the human health of cabbage vegetable including a lipid metabolism. This may be due to bioactive contents that included vitamin C, phenolic, fatty acids and flavonoid and depended on included season and several other environmental. Our results agree with the previous study that found a hepatoprotective effect from the leaf extracts of Brassica juncea in CCl4 induced rat model but the bad effect on thyroid function and a drop in both T3, and T4 concentrations may be due to the presence of the glucosinolate that interference with the conjugation process. The glucosinolate interferes with iodine uptake (thiocyanate ion) and the synthesis of thyroid hormones T3 (triiodothyronine) and plasma thyroxine (T4) (5- vinyloxazolidine-2 thione), leading eventually to hypothyroidism and enlargement of the thyroid gland (goiter) [66].

5. Conclusion

Finally, the studies mentioned above, we have been able to conclude a positive association between avocado (Persea Americana), Ginger (Zingiber Officinale) feeding and the decrease thyroid disease. Also, there is a positive relationship between the ingest of Cabbage (Brassica Oleracea) and the increase thyroid disease; these observations will require further experiments for validation.

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