



## **The Effect of Arachidonic acid and calcium deposition on Bony mass in experimental animal**

**Sahar Othman El Shafei, Khaled Ali Abdel Rahman Shaheen,  
and Souad Hashem Mustafa**

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### **Abstract:**

Bones are one of the most important parts of the human body, as weak bones may lead to general weakness as it shields for all body system.

Arachidonic acid is a polyunsaturated fatty acid present in the phospholipids (especially phosphatidylethanolamine, phosphatidylcholine, and phosphatidylinositides) of membranes of the body's cells, and is abundant in the brain, muscles, and liver. Skeletal muscle is an especially active site of arachidonic acid retention, accounting for roughly 10-20% of the phospholipid fatty acid content on average. It still plays a fundamental role in reducing pathogenicity.

This study aims to investigate the effect of Arachidonic acid (deficiency & treatment) on calcium deposition and bones density. Compare the results of those groups with the group affected by decrease Arachidonic acid and increase calcium deposition.

### **Experimental design:**

Thirty six healthy adult male albino rats were fed on standard diet for one week for adaptation. After this week, they were divided into six groups, The first group (6 rats) fed on basal diet (as a negative control group).

The second group (6 rats) fed on standard diet without arachidonic acid and double dose of calcium (as a positive control group).

The third group (6 rats) fed on standard diet with arachidonic acid (1000 mg) and the double dose of calcium

The fourth group (6 rats) fed on standard diet with arachidonic acid (1150mg) and the double dose of calcium

The five group (6 rats) fed on standard diet with aracidonic acid (1300 mg) and the double dose of calcium

The last group (6 rats) fed on standard diet with aracidonic acid (1450 mg) and the double dose of calcium

**Conclusion:**

Results obtained for this study led to several conclusions:

it is important for The Relation ship between Arachidonic acid and first time walking in children bone mass increase and the best Results in 1450 mg.

**Keywords:** arachidonic acid-like omega-3- fatty acids (DHA)- calcium deposition

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**Introduction**

Arachidonicacid (AA) sometimes ARA) is a polyunsaturated omega-6fatty acid 20:4( $\omega$ -6), or 20:4(5,8,11,14). It is structurally related to the saturated arachidic acid found in cupuaçu butter. (**Dorland's Medical Dictionary ,2007**)

In chemical structure, Arachidonic acid is a carboxylic acid with a 20-carbon chain and four cis-double bonds; the first double bond is located at the sixth carbon from the omega end.

Some chemistry sources define 'arachidonic acid' to designate any of the eicosatetraenoic acids. However, almost all writings in biology, medicine, and nutrition limit the term to all cis-5,8,11,14-eicosatetraenoic acid. Sources (meat, eggs) or is synthesized from linoleic acid. .(**MacDonald, etal. 1984**) and(**Rivers, etal. 1975**).

Arachidonic acid is not one of the essential fatty acids. However, it does become essential if a deficiency in linoleic acid exists or if an inability to convert linoleic acid to arachidonic acid occurs. Some mammals lack the ability or have a very limited capacity to convert linoleic acid to arachidonic acid, making it an essential part of their diets. Since little or no arachidonic acid is found in common plants, such animals are obligate carnivores; the cat is a common example having inability to desaturate essential fatty acids .(**MacDonald, etal. 1984**) and(**Rivers, etal. 1975**).

In the body: muscle growth Arachidonic acid promotes the repair and growth of skeletal muscle tissue via conversion to prostaglandin PGF<sub>2</sub>alpha during and following physical exercise. (

**Trappe, et al. 2013).** Arachidonic acid does still play a central role in inflammation related to injury and many diseased states. How it is metabolized in the body dictates its inflammatory or anti-inflammatory activity. Individuals suffering from joint pains or active inflammatory disease may find that increased arachidonic acid consumption exacerbates symptoms, presumably because it is being more readily converted to inflammatory compounds. Likewise, high arachidonic acid consumption is not advised for individuals with a history of inflammatory disease, or who are in compromised health. (Birdwell, et al.1994).

**Material And Methods.**

**Materials:**

Skimmed milk and corn starch were purchased from local market, Cairo, Egypt.

**Chemicals:**

DL methionine, choline chloride, vitamins, minerals, omega3,6,9 and kits required were obtained from El- Gomhorya Company for chemicals and Drugs, Cairo, Egypt Omega 6 the unit contain Of 296 mg of linoleic acid and gama linolenic acid. Induction of 2000mg calcium.

**Animals:**

Thirty sex healthy adult male albino rats "Sprague Dawley strain" weighing (150±10g.) were obtained from vaccine and immunity organization Helwan Farm, Cairo, Egypt.

**Diets:** Standard diet was prepared as previously described by (Reeves ,et al.,1993 ).

**Methods:**

**Biological Experimental.**

Thirty sex healthy adult male albino rats "Sprague Dawley strain" weighing (150±10g.) were kept in wire cages . the diet was introduce to the rats in special food cups to avoid scattering of food . also water was provided to the rats . food and water were provided ad-libitum and checked daily.

Induction of Omega 3,6,9 Omega 6 the unit contain Of 296 mg of linoleic acid and gama linolenic acid. Induction of 2000mg calcium.

**Experimental design:**

Thirty sex healthy adult male albino rats were fed on standard diet for one week for adaptation. After this week, they were divided into

six groups, each group with similar total body weight and were housed individually in wire cages.

The first group (6 rats) fed on basal diet fed on standard diet (as a negative control group).

The second group (6 rats) fed on standard diet without aracidonic acid and double dose of calcium (as a positive control group).

The third group (6rats) fed on standard diet with aracidonic acid( 1000 mg) and the double dose of calcium

The fourth group (6 rats) fed on standard diet with aracidonic acid(1150mg ) and the double dose of calcium

The five group (6 rats) fed on standard diet with aracidonic acid (1300 mg) and the double dose of calcium

The last group (6 rats) fed on standard diet with aracidonic acid (1450 mg) and the double dose of calcium At the end experiment (4 weeks),

Animals sacrificed under anesthesia .Blood samples were taken in dry centrifuge tubes from the hepatic portal vein . Serum was separated and kept in plastic vial at -20c until analysis.

#### **Biological evaluation:**

- **Calculation of body weight gain (BWG) and relative organs' weight:-**

All animals were individually weighed once a week during the experiment. The difference between the initial and final body weight was calculated as **follows:**

$$\text{BWG (g)} = \text{final weight (g)} - \text{initial weight (g)}$$

- **Calculation of feed intake (FI) and feed efficiency ratio (FER):-**

The total food consumed was calculated by subtracting the remaining food for each animal at the end of each week from that allocated to it at the start of the week. Food wastage was weighed and subtracted.

The feed efficiency ratio was calculated according to the following equation as mentioned by (**Hosoya, 1980**).

$$\text{FER} = \text{Body weight gain (g)} / \text{Feed intake (g)}$$

#### **Biochemical analysis:**

Determination of lipid profiles:

Determination of Triglycerides:

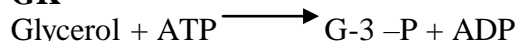
The quantitative enzymatic colorimetric determination of triglycerides in serum using kits Stanbio laboratory according to (Wahlefeld , 1974).

**Principle:**

1- Glycerol and fatty acids first formed by lipase action on the triglycerides.

2- Glycerol is then phosphorylated by adenosine-5-triphosphate (ATP) to produce glycerol -3- phosphate (G-3-P) and adenosine-5-diphosphate (ADP) in a reaction catalyzed by glycerol kinase (GK):

**GK**



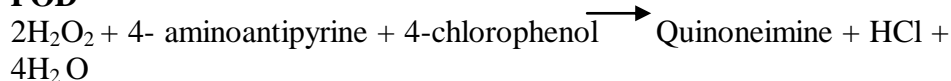
3- The G-3-P oxidized by glycerylphosphate oxidase (GPO) producing dihydroxyacetone phosphate (DAP) and hydrogen peroxide:

**GPO**



4- Peroxide reacts with a 4-aminoantipyrine and 4-chlorophenol under the catalytic influence of peroxidase (POD) to form quinoneimine

**POD**



Lipid Clearing Factor (LCF): a mixture of special additives developed by Stanbio integrated into the triglyceride reagent to help minimize interference due to lipemia.

Read the absorbance at 500nm by (Spectronic Unicam No. 082329 analytical Systems).( Wahlefeld , 1974).

**Determination of Cholesterol:**

Quantitative enzymatic colorimetric determination of total cholesterol in serum using kits Stanbio laboratory according to (Stein, 1986).

**Determination of High-Density Lipoprotein (HDL):**

Stanbio HDL cholesterol kits use for in the determination of HDL cholesterol in serum according to( Stein, 1986).

**Determination of Low-density lipoprotein (LDL):**

Low-density lipoprotein (LDL) cholesterol according to (Friedewald, *et al.*, 1972)

**Determination of Aminotransferase (ALT and AST):-**

Aminotransferase were determined using kits supplied from Setinel CH according to the method of (Bergmeyer and Horder, 1980).

**Determination of Gamma –GT:**

Gamma – GT was determined using kits supplied Quimica clinical Aplicada S.A. according to( Szasz, 1969).

**Determination of Blood Urea Nitrogen (BUN):**

Urea nitrogen was determined in the serum according to (Tabacco, et al., 1979).

**Determination of Uric Acid:**

Enzymatic colorimetric method to determination of uric acid by using Sentinel CH kits according to (Fossati, et al., 1980).

**Determination of Creatinine:**

Kinetic determination of Creatinine using BioMerieux kits according to( Houot, 1985).

**Statistical analysis:**

Statistical analysis was carried out using the programme of Statistical Package for the Social Sciences (SPSS), PC statistical software (Version22; Untitled–SPSS Data Editor). (SPSS), PC statistical software (Version 22; Untitled–SPSS Data Editor).

The results were expressed as mean  $\pm$  standard error (mean  $\pm$  S.E.). Data were analyzed using one way classification, analysis of variance (ANOVA). The differences between means were tested for significance using least significant difference (LSD) test at  $p < 0.05$ . Independent T test was also used to determine the statistical difference between two means(Sendcor and Cochran, 1979).

**Histopathological examination:**

Hearts ,livers and kidneysaccordingto( Lambergton and Rothstein,1988).

**Results:**

Data in table (1) showed the effect of Aracidonic and double dose of Calcium( feed intak ,Body weight again and feed efficiency ratio) on of bony massin rats The observation from table (1) to illustrate the rate of increas perecentages between the six groups . it was found that thefeed intak( FI), observed negative control group( 1)was $14.8^a \pm 0.76$  , which was significantlly lower than G2 group, also positive control group was significantlly higher than G1 group while G5 group

was significantly higher than G3 & G4 groups. There were no significant changes among G2, G3 and G4 groups. The observation from table (1) to illustrate the rate of increase percentages between the six groups. It was found that the Body weight again (BWA) weight of the positive control group was greater than the negative control group  $47.1 \pm 15.1$  so it was weight again equal between the (4 and 5) groups  $52.1 \pm 17.1$  and  $52.1 \pm 17.2$  but group (6) recorded the largest noticable increase  $56.3 \pm 19$  and equal rats increase were seen for both (4,5) groups of the two ( $52.1 \pm 17.1$  and  $52.1 \pm 17.2$ ).

The obtained results indicated feed efficiency ratio (FER) observed negative control group was  $0.025 \pm 0.12$ , which was significantly lower than G6  $0.037 \pm 0.027$  also positive control group was significantly higher than G1 group. There were no significant changes among G4, G5. Data in table (2) showed the effect of Aracidonic and double dose of Calcium on liver function (ALT, AST and ALP) of bony massin rats. The obtained results indicated ALT, observed negative control group was  $58.7 \pm 7.64$ , which was significantly higher than G5 group  $38.0 \pm 2.00$ , also positive control group  $53.7 \pm 2.89$  was significantly higher than G5 group  $38.0 \pm 2.00$ , but G5 group  $38.0 \pm 2.00$  was significantly lower than G3  $58.0 \pm 4.00$  & G4  $55.7 \pm 3.51$ . There were no significant changes among G3, G4 and G6 groups.

The obtained results indicated AST, it could be observed negative control group was  $188.7 \pm 8.15$ , which was no significant with all tested groups, also positive control group was no significant with all tested groups, but G3 group  $194 \pm 15.52$  was significantly higher than G5  $135.7 \pm 19.86$  & G6  $135.7 \pm 27.47$ . There were no changes among G6, G5 the obtained results indicated ALP, it could be observed negative control group was  $191.0 \pm 29.82$ , which was significantly higher than G5 group, while positive control group was significantly lower than G6 group  $238.0 \pm 18.08$ , but G6 group was significantly higher than G3, G4 and G5 groups. Data in table (3) showed the effect of Aracidonic and double dose of Calcium on Protein of bony massin rats (Total Protein, Albumin and Globulin). The obtained results indicated Total Protein, observed negative control group was  $7.4 \pm 0.46$ , which was no significant with all tested groups, also positive control group was no significant with all tested groups. There were no significant changes

among  $G36.9^a \pm 0.06$ ,  $G46.7^a \pm 0.35$ , There were no significant changes among  $G27.0^a \pm 1.01$  and  $G67.0^a \pm 0.40$ .

**The obtained results indicated Albumin**, observed negative control group was  $3.7 \pm 0.06$ , which was not significant with all tested groups, also positive control group was not significant with all tested groups. There were no significant changes among G1, G2, G3 and G4 groups. But G5 group  $3.9^a \pm 0.10$  which was significantly higher than G6 group  $3.8^a \pm 0.36$ .

**The obtained results indicated Globulin**, observed negative control group was  $3.7 \pm 0.40$ , was significantly higher with all tested groups, also positive control group was not significant with G3  $3.3^a \pm 0.21$ . There were no significant changes among G5  $3.2^a \pm 0.26$  and G6  $3.2^a \pm 0.76$ . **Data in table (4) showed the effect of Aracidonic and double dose of Calcium on Renal function of bony massin rats (Creatinine, Uric acid and Urea). the obtained results indicated Creatinine** observed negative control group  $0.8 \pm 0.00$ , which was not significant with all tested groups, while positive control group was significantly Lower than G3  $0.8^{ab} \pm 0.06$ . There were no significant changes among, G4, G5  $0.9^a \pm 0.06$  and There were no significant changes among G6, G3  $0.8^{ab} \pm 0.06$  groups.

**The obtained results indicated Uric acid**, it could be observed negative control group was  $3.4 \pm 0.36$ , which was significantly higher with G3, G4, G5, G2 groups, while positive control group was significantly Lower than G6  $3.6^a \pm 0.42$ . There were no significant changes among G3  $2.2^{ab} \pm 0.26$ , G4  $2.3^{ab} \pm 0.78$ .

**The obtained results indicated Urea**, it could be observed negative control group was  $35.6 \pm 8.80$ , which was significantly higher with all tested groups, also positive control group  $23.1^a \pm 2.78$  was not significant with G3  $23.6^a \pm 0.64$ . **Data in table (5) showed the effect of Aracidonic && double dose of Calcium on Lipid function of bony massin rats (Total Cholesterol, Triglycerides Cholesterol, HDL Cholesterol, LDL Cholesterol and VLDL Cholesterol). the obtained results indicated Total Cholesterol**, observed negative control group was  $54.8 \pm 6.09$ , which was significantly lower than with all tested groups without G3, also positive control group  $57.5^a \pm 8.61$  was not significant with G3, G5 groups.



**The obtained results indicated Triglycerides**, observed negative control group was  $88.6 \pm 11.43$ , which was significantly higher with all tested groups with out  $G2111.8 \pm 59.06$ , There were no significant changes among G3, G4, G5 and G6 groups.

**The obtained results indicated HDL Cholesterol**, observed negative control group was  $19.2 \pm 3.03$ , which was no significant with all tested groups, also positive control group was no significant with all tested groups. There were no significant changes among G3, G4, G5 and G6 groups.

**The obtained results indicated LDL Cholesterol**, observed negative control group was  $17.9 \pm 6.03$ , which was no significant with G6, also positive control group  $15.8 \pm 4.79$  was significantly lower than  $G323.4 \pm 6.73$ .

**The obtained results indicated VLDL Cholesterol**, observed negative control group was  $17.7 \pm 2.29$ , which was significantly higher than G3, G4, G5 groups, also positive control group  $22.4 \pm 11.81$  was significantly higher than all tested groups

#### **Discussion:**

Arachidonic acid is marketed as an anabolic bodybuilding supplement in a variety of products. Supplementation of arachidonic acid (1,500 mg/day for 8 weeks) has been shown to increase lean body mass, strength, and anaerobic power in experienced resistance-trained men. This was demonstrated in a placebo-controlled study at the University of Tampa. Thirty men (aged  $20.4 \pm 2.1$  years) took arachidonic acid or a placebo for 8 weeks, and participated in a controlled resistance-training program. After 8 weeks, lean body mass (LBM) had increased significantly, and to a greater extent, in the ARA group (1.62 kg) vs. placebo (0.09 kg) ( $p < 0.05$ ). The change in muscle thickness was also greater in the ARA group (.47 cm) than placebo (.25 cm) ( $p < 0.05$ ). Wingate anaerobic power increased to a greater extent in ARA group as well (723.01 to 800.66 W) vs. placebo (738.75 to 766.51 W). Lastly, the change in total strength was significantly greater in the ARA group (109.92 lbs.) compared to placebo (75.78 lbs.). These results suggest that ARA supplementation can positively augment adaptations in strength and skeletal muscle hypertrophy in resistance-trained men. (Ormes, Jacob.2007)

An earlier clinical study examining the effects of 1,000 mg/day of arachidonic acid for 50 days found supplementation to enhance anaerobic capacity and performance in exercising men. During this study, a significant group–time interaction effect was observed in Wingate relative peak power (AA:  $1.2 \pm 0.5$ ; P:  $-0.2 \pm 0.2$  W•kg<sup>-1</sup>,  $p=0.015$ ). Statistical trends were also seen in bench press 1RM (AA:  $11.0 \pm 6.2$ ; P:  $8.0 \pm 8.0$  kg,  $p=0.20$ ), Wingate average power (AA:  $37.9 \pm 10.0$ ; P:  $17.0 \pm 24.0$  W,  $p=0.16$ ), and Wingate total work (AA:  $1292 \pm 1206$ ; P:  $510 \pm 1249$  J,  $p=0.087$ ). AA supplementation during resistance training promoted significant increases in relative peak power with other performance-related variables approaching significance. These findings support the use of AA as an ergogenic. ( Roberts, ,etal.2011) .

**Table ( 1) Effect of Aracidonic and double dose of Calcium on( feed intak ,Body weight again and feed efficiency ratio).**

Groups	FI(gm/day)	BWG(gm)	FER%
Negative control (G1)	14.8 <sup>a</sup> ±0.76	37.5 <sup>b</sup> ±12.1	.025 <sup>b</sup> ±0.12
Positive control (G2) + Calcium	14.9 <sup>a</sup> ±0.77	47 <sup>b</sup> ±15.1	.031 <sup>b</sup> ±0.32
Aracidonic (1000mg) + Calcium (G3)	14.9 <sup>a</sup> ±0.77	49 <sup>b</sup> ±16.1	.032 <sup>b</sup> ±0.04
Aracidonic (1150mg) + Calcium (G4)	14.9 <sup>a</sup> ±0.77	52 <sup>b</sup> ±17.1	.034 <sup>b</sup> ±0.016
Aracidonic (1300mg) + Calcium (G5)	15.01 <sup>a</sup> ±0.79	52.1±17.2	.034 <sup>b</sup> ±0.015
Aracidonic (1450mg) + Calcium (G6)	15.08 <sup>a</sup> ±0.80	56.3 <sup>b</sup> ±19	.037 <sup>b</sup> ±0.027

**Table (2) Effect of Aracidonic & double dose of Calcium on liver function of bony massin rats**

Groups	ALT (u/l)	AST (u/l)	ALP (u/l)
Negative control (G1)	58.7 <sup>a</sup> ±7.64	188.7 <sup>ab</sup> ±8.15	191.0 <sup>ab</sup> ±29.82
Positive control (G2) + Calcium	53.7 <sup>a</sup> ±2.89	176.0 <sup>ab</sup> ±29.10	131.7 <sup>bc</sup> ±25.17
Aracidonic (1000mg) + Calcium (G3)	58.0 <sup>a</sup> ±4.00	194 <sup>a</sup> ±15.52	135.3 <sup>bc</sup> ±20.82
Aracidonic (1150mg) + Calcium (G4)	55.7 <sup>a</sup> ±3.51	169.3 <sup>ab</sup> ±10.79	176 <sup>b</sup> ±14.00
Aracidonic (1300mg) + Calcium (G5)	38.0 <sup>b</sup> ±2.00	135.7 <sup>b</sup> ±19.86	110.3 <sup>c</sup> ±22.68
Aracidonic (1450mg) + Calcium (G6)	47.3 <sup>ab</sup> ±2.31	135.7 <sup>b</sup> ±27.47	238.0 <sup>a</sup> ±18.08

**Table (3) Effect of Aracidonic and double dose of Calcium on Protein of bony massin rats**

Groups	Total Protein(u/l)	Albumin(u/l)	Globulin(u/l)
Negative control (G1)	7.4 <sup>a</sup> ±0.46	3.7 <sup>a</sup> ±0.06	3.7 <sup>a</sup> ±0.40
Positive control (G2)+ Calcium	7.0 <sup>a</sup> ±1.01	3.7 <sup>a</sup> ±0.15	3.3 <sup>a</sup> ±0.85
Aracidonic (1000mg) + Calcium (G3)	6.9 <sup>a</sup> ±0.06	3.7 <sup>a</sup> ±0.15	3.3 <sup>a</sup> ±0.21
Aracidonic (1150mg) + Calcium (G4)	6.7 <sup>a</sup> ±0.35	3.7 <sup>a</sup> ±0.06	3.0 <sup>a</sup> ±0.29
Aracidonic (1300mg) + Calcium (G5)	7.1 <sup>a</sup> ±0.17	3.9 <sup>a</sup> ±0.10	3.2 <sup>a</sup> ±0.26
Aracidonic (1450mg) + Calcium (G6)	7.0 <sup>a</sup> ±0.40	3.8 <sup>a</sup> ±0.36	3.2 <sup>a</sup> ±0.76

**Table (4) Effect of Aracidonic & double dose of Calcium on Renal function of bony massin rats**

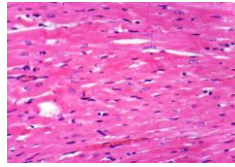
Groups	Creatinine (u/l)	Uric acid(u/l)	Urea(u/l)
Negative control (G1)	0.8 <sup>ab</sup> ±0.00	3.4 <sup>ab</sup> ±0.36	35.6 <sup>a</sup> ±8.80
Positive control (G2) + Calcium	0.7 <sup>b</sup> ±0.06	2.0 <sup>b</sup> ±0.46	23.1 <sup>a</sup> ±2.78
Aracidonic (1000mg) + Calcium (G3)	0.8 <sup>ab</sup> ±0.06	2.2 <sup>ab</sup> ±0.26	23.6 <sup>a</sup> ±0.64
Aracidonic (1150mg) + Calcium (G4)	0.9 <sup>a</sup> ±0.06	2.3 <sup>ab</sup> ±0.78	31.3 <sup>a</sup> ±3.38
Aracidonic (1300mg) + Calcium (G5)	0.9 <sup>a</sup> ±0.06	3.2 <sup>ab</sup> ±0.71	29.1 <sup>a</sup> ±5.16
Aracidonic (1450mg) + Calcium (G6)	0.8 <sup>ab</sup> ±0.00	3.6 <sup>a</sup> ±0.42	26.1 <sup>a</sup> ±2.50

**Table (5) Effect of Aracidonic and double dose of Calcium on lipid function of bony massin rats**

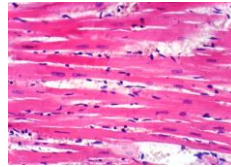
Groups	Total Cholesterol	Triglycerides	HDL Cholesterol	LDL Cholesterol	VLDL Cholesterol
Negative control (G1)	54.8 a±6.09	88.6 a±11.43	19.2 a±3.03	17.9 a±6.03	17.7 a±2.29
Positive control (G2)+ Calcium	57.5 a ±8.61	111.8 a ±59.06	19.3 a±1.03	15.8 a±4.79	22.4 a±11.81
Aracidonic (1000mg) + Calcium (G3)	57.9 a±4.67	68.0 a±23.01	20.9 a ±3.38	23.4 a ±6.73	13.6 a ±4.60
Aracidonic (1150mg) + Calcium (G4)	49.2 a±7.79	79.1 a±31.36	18.7 a ±4.00	14.6 a ±9.78	15.8 a ±6.27
Aracidonic (1300mg) + Calcium (G5)	57.0 a±6.92	59.0 a±15.45	19.9 a ±2.05	25.4 a±5.33	11.7 a±3.14
Aracidonic (1450mg) + Calcium (G6)	53.7 a ±9.70	94.7 a±28.29	17.2 a ±2.78	17.6 a±11.85	18.9 a±5.66

#### **Histopathological Examination Of Heart:**

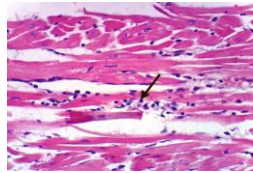
Microscopically, heart of rat from group 1 revealed the normal histological structure of cardiac myocytes (Figs. 1 and 2). Meanwhile, heart of rats from group 2 showed intermyocardial oedema associated with intermyocardial infiltration with mononuclear inflammatory cells (Figs. 3 and 4). However, heart of rats from group 3 revealed no histopathological alterations except slight intermyocardial oedema in some sections (Figs. 5 and 6). Some sections from group 4 showed intermyocardial infiltration with mononuclear inflammatory cells (Fig. 7), whereas, other sections from this group revealed no histopathological alterations (Fig. 8). Moreover, heart of rats from group 5 exhibited no histopathological alterations (Figs. 9 and 10). Furthermore, the only histopathological change observed in heart of rats from group 6 was slight intermyocardial oedema (Figs. 11 and 12).



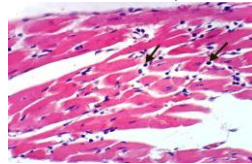
**Fig. (1):** Heart of rat from group 1 showing the normal histological structure of cardiac myocytes (H & E X 400).



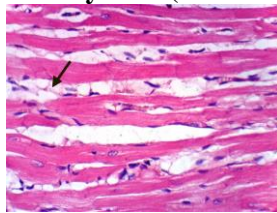
**Fig. (2):** Heart of rat from group 1 showing the normal histological structure of cardiac myocytes (H & E X 400).



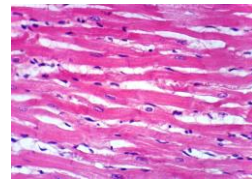
**Fig. (3):** Heart of rat from group 2 showing intermyocardial infiltration with mononuclear inflammatory cells (H & E X 400).



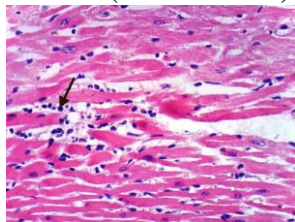
**Fig. (4):** Heart of rat from group 2 showing intermyocardial infiltration with mononuclear inflammatory cells (H & E X 400).



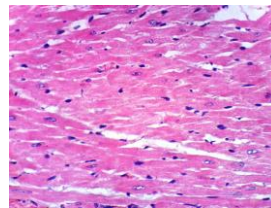
**Fig. (5):** Heart of rat from group 3 showing slight intermyocardial oedema (H & E X 400).



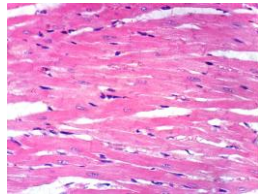
**Fig. (6):** Heart of rat from group 3 showing no histopathological alterations (H & E X 400).



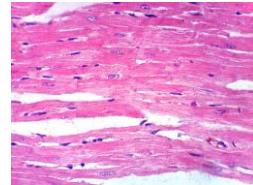
**Fig. (7):** Heart of rat from group 4 showing intermyocardial infiltration with mononuclear inflammatory cells (H & E X 400).



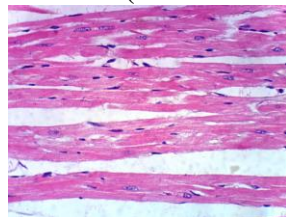
**Fig. (8):** Heart of rat from group 4 showing no histopathological alterations (H & E X 400).



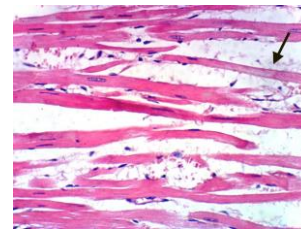
**Fig. (9): Heart of rat from group 5 showing no histopathological alterations (H & E X 400).**



**Fig. (10): Heart of rat from group 5 showing no histopathological alterations (H & E X 400).**



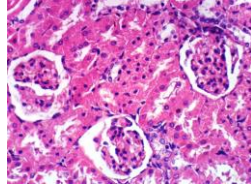
**Fig. (11): Heart of rat from group 6 showing no histopathological alterations (H & E X 400).**



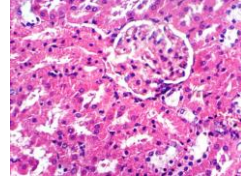
**Fig. (12): Heart of rat from group 6 showing slight intermyocardial oedema (H & E X 400).**

#### **Histopathological examination of kidneys:**

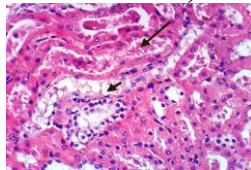
Microscopically, kidneys of rats from group 1 revealed the normal histological structure of renal parenchyma (Figs. 1 and 2). On contrary, kidneys of rats from group 2 showed cytoplasmic vacuolization of epithelial lining renal tubules, proteinaceous material in the lumen of renal tubules (Fig. 3) and focal necrosis of renal tubules associated with inflammatory cells infiltration (Fig. 4). However, kidneys of rats from group 3 revealed no histopathological changes (Fig. 5) except proteinaceous material in the lumen of renal tubules (Fig. 6). On the other hand, kidneys of rats from group 4 showed no histopathological changes (Fig. 7) except focal interstitial few inflammatory cells infiltration (Fig. 8) in some sections. Examined sections from group 5 showed cytoplasmic vacuolization of epithelial lining renal tubules, congestion of glomerular tuft (Fig. 9) and focal necrosis of renal tubules associated with calcification (Fig. 10). Meanwhile, kidneys from group 6 revealed no histopathological changes (Figs. 11 and 12).



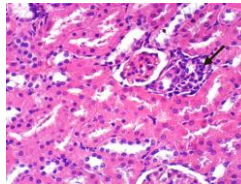
**Fig. (1):** kidney of rat from group 1 showing the normal histological structure of renal parenchyma (H & E X 400).



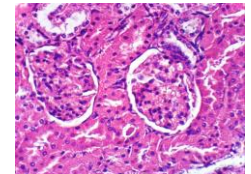
**Fig. (2):** kidney of rat from group 1 showing the normal histological structure of renal parenchyma (H & E X 400).



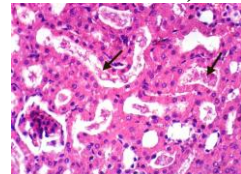
**Fig. (3):** kidney of rat from group 2 showing cytoplasmic vacuolization of epithelial lining renal tubules and proteinaceous material in the lumen of renal tubules (H & E X 400).



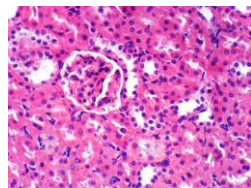
**Fig. (4):** Kidney of rat from group 2 showing focal necrosis of renal tubules associated with inflammatory cells infiltration (H & E X 400).



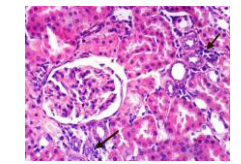
**Fig. (5):** kidney of rat from group 3 showing no histopathological changes (H & E X 400).



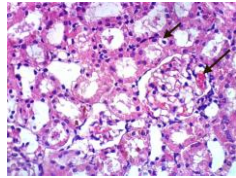
**Fig. (6):** kidney of rat from group 3 showing proteinaceous material in the lumen of renal tubules (H & E X 400).



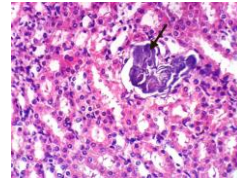
**Fig. (7):** kidney of rat from group 4 showing no histopathological changes (H & E X 400).



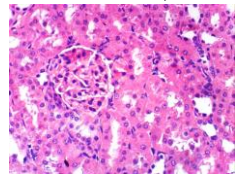
**Fig. (8):** kidney of rat from group 4 showing focal interstitial few inflammatory cells infiltration (H & E X 400).



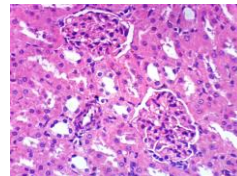
**Fig. (9):** kidney of rat from group 5 showing cytoplasmic vacuolization of epithelial lining renal tubules and congestion of glomerular tuft (H & E X 400).



**Fig. (10):** kidney of rat from group 5 showing focal necrosis of renal tubules associated with calcification (H & E X 400).



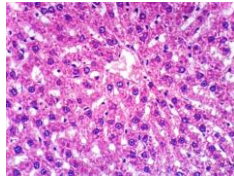
**Fig. (11):** kidney of rat from group 6 showing no histopathological changes (H & E X 400).



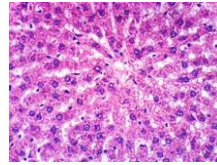
**Fig. (12):** kidney of rat from group 6 showing no histopathological changes (H & E X 400).

#### **Histopathological examination of liver:**

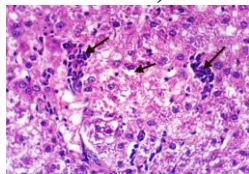
Microscopically, liver of rats from group 1 revealed the normal histological structure of hepatic lobules (Fig. 1). On contrary, liver of rats from group 2 hydroptic degeneration of hepatocytes (Fig. 3 and 4), focal hepatic necrosis associated with inflammatory cells infiltration (Fig. 3) and portal infiltration with inflammatory cells (Fig. 4). However, liver of rats from group 3 showed slight hydroptic degeneration of hepatocytes (Fig. 5) and vacuolar degeneration of some hepatocytes (Fig. 6). Meanwhile, some examined sections from group 4 revealed slight hydroptic degeneration of hepatocytes (Fig. 7), whereas, other sections showed focal hepatic necrosis associated with inflammatory cells infiltration (Fig. 8). Furthermore, liver of rats from group 5 revealed slight Kupffer cells activation (Figs. 9 and 10) and portal infiltration with few inflammatory cells (Fig.10). Moreover, liver of rats from group 6 showed slight hydroptic degeneration of some hepatocytes (Fig. 11) and sinusoidal leukocytosis (Fig.12).



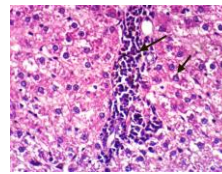
**Fig. (1):** Liver of rat from group 1 showing the normal histological structure of hepatic lobule (H & E X 400).



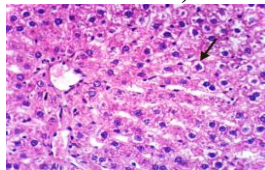
**Fig. (2):** Liver of rat from group 1 showing the normal histological structure of hepatic lobule (H & E X 400).



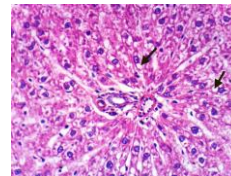
**Fig. (3):** Liver of rat from group 2 showing hydropic degeneration of hepatocytes and focal hepatic necrosis associated with inflammatory cells infiltration (H & E X 400)



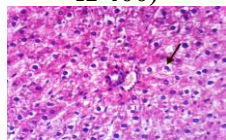
**Fig. (4):** Liver of rat from group 2 showing hydropic degeneration of hepatocytes and portal infiltration with inflammatory cells (H & E X 400)



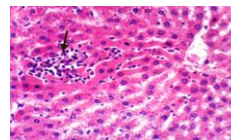
**Fig. (5):** Liver of rat from group 3 showing slight hydropic degeneration of hepatocytes (H & E X 400)



**Fig. (6):** Liver of rat from group 3 showing vacuolar degeneration of some hepatocytes (H & E X 400)

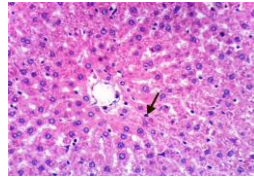


**Fig. (7):** Liver of rat from group 4 showing slight hydropic degeneration of hepatocytes (H & E X 400)

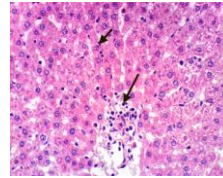


**Fig. (8):** Liver of rat from group 4 showing focal hepatic necrosis associated with inflammatory cells infiltration (H & E X 400)

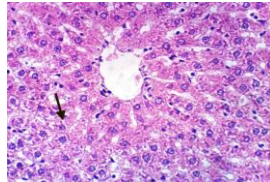




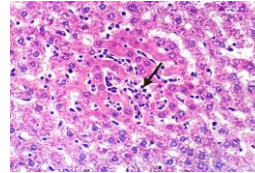
**Fig. (9):** Liver of rat from group 5 showing slight Kupffer cells activation (H & E X 400).



**Fig. (10):** Liver of rat from group 5 showing slight Kupffer cells activation and portal infiltration with few inflammatory cells (H & E X 400).



**Fig. (11):** Liver of rat from group 6 showing slight hydropic degeneration of some hepatocytes (H & E X 400).



**Fig. (12):** Liver of rat from group 6 showing sinusoidal leukocytosis (H & E X 400).

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## تأثير حامض الأراكيدونيك وتكثيف الكالسيوم علي الهيكل العظمي في حيوانات التجارب

سحر عثمان الشافعي، خالد على عبد الرحمن شاهين، سعاد هاشم مصطفى

### الملخص العربي:

العظام من اهم اجزاء جسم الانسان فقد يؤدي ضعف العظام للضعف العام والاصابة بالامراض فهي دروع لجميع اجهزة الجسم وهنا تبرز اهمية تناول حامض الاراكيدونيك حيث حمض الاراكيدونيك من الاحماض الكربوكسيلية مع السلسلة 20 واربع روابط مستقلة ويقع اول رابطة مزدوجة فى الكربون السادس مع نهاية اوميغا ومن اهم مصادر اللحم والبيض و تخليق حمض اللينوليك ويمكن اسخلاصه من فطر فطر مورتاريللا البيتا حامض الاراكيدونيك هو من الاحماض الدهنية فى الدهون الفوسفاتية خاصة فوسفاتيديل ايثانولامين من اغشية الخلايا فى الجسم ويوجد بوفرة فى الدماغ والعضلات والكبد والهيكل العظمى وهو موقع نشط بشكل خاص من الاحتفاظ بحمض الاراكيدونيك وهو ما يمثل من 10-20% من محتوى الدهون الفسفورية فى المتوسط كما انها الى كونها تشارك فى الاشارات الخلوية كما انها رسول ثانى فى تنظيم اشارات الانزيمات

أهمية حامض الاراكيدونيك فى الجسم: نمو العضلات حماية خلايا المخ من التلف

### الهدف من الدراسة:

- 1- التحقق من تأثير نقص وعلاج حامض الاراكيدونيك على تركيز وتكثيف الكالسيوم وكثافة العظام
- 2- مقارنة نتائج المجموعات مع المجموعة التى تم نقص حامض الاراكيدونيك وزيادة تركيز الكالسيوم
- 3- علاقة حامض الاراكيدونيك باول نقطة بداية المشى للاطفال
- 4- علاقة حامض الاراكيدونيك بالجسم النحيف وزيادة كتلة العظام.