

## EFFECT OF SOME NUTRIENTS ON THE HEALTH STATUS OF IRON ANEMIC RATS

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**ABSTRACT:** *The main objective of this study is to evaluate the interaction effects of some nutritional elements in healthy status of rats induced by iron deficiency anemia. A total of 60 male albino rats weighing (120±10g) were used in this study. Rats were divided into 6 groups, the first control negative group, other groups was induced by iron deficiency anemia as fed on 20 mg of tannic acid for 21 frequency days, these anemic rats fed on standard diet and high dietary iron source as liver along studying period and divided into follow groups, the second control positive group. Third group fed on high dietary iron source as liver only. Fourth group supplemented with sucrose (13.8% of the diet). Fifth group supplemented with vitamin A (4500 IU/kg feed). Sixth group supplemented with calcium (1.2% of diet) for 28 days. The results showed that there was a significant increase in serum iron among treated groups after dietary intervention, especial in liver plus vitamin A group. For serum ferritin, comparative effect stated that liver plus sucrose group had the nearest mean to negative control group. While liver plus calcium group had insignificant differed to positive control group.*

**Key words:** Rats- Calcium- Liver-Vitamin A-Sucrose – Hemoglobin.

### INTRODUCTION

Iron deficiency is one of the most common of the nutritional deficiencies. Iron is present in all cells in the human body, and has several vital functions (Dlouhy *et al.*, 2013). More than 1.6 billion peoples, almost a quarter of the world's population, are anemic. Despite considerable economic and scientific advancement during recent decades, there has been, at best, only marginal reduction in the global prevalence of anemia (Benoist *et al.*, 2005). Calcium has been shown to have negative effects on non-heme and heme iron absorption, which makes it different from other inhibitors that affect non-heme iron absorption only (Cook, *et al.*, 1991; Hallberg *et al.*, 1993). The maintenance of adequate vitamin A intake should be encouraged which appears to be an effective intervention strategy to achieve appropriate serum retinol and hemoglobin values, and hence caused a lower iron deficiency anemia rates (Barbosa *et al.*, 2013). (Devasenapathy *et al.*, 2013) reported that the current evidence on the safety and efficacy of intravenous iron sucrose on hematological and clinical outcomes was confirmed. Though the evidence on its efficacy in

improving hemoglobin and serum ferritin is convincing. Thus, the present study aimed to the effects of some some nutrients on healthy status of rats induced iron deficiency anemia.

### MATERIALS AND METHODS

#### Materials:

Fresh liver and sucrose were obtained from the local market of Shibin El-Kom City, Minufiya governorate, Egypt.

Vitamin A, calcium capsules and Tannic acid were obtained from Morgan Co., Cairo, Egypt.

**Rats:** A total of 60 adult normal male white albino rats Sprague Dawley strain weighing 120±10g were obtained from Vaccine and Immunity Organization, Ministry of Health, Helwan Farm, Cairo, Egypt.

#### Animals and Experimental Design:

Sixty adult male white albino rats, Sprague Dawley Strain, 8 weeks age, weighting (120±10g) were used in this experiment. All rats were fed on basal diet prepared according to American Institute of Nutrition (AIN, 1993) for 7 consecutive days. Rats were randomly divided into six

groups (10 rats per each). The first control negative group fed on normal basal diet for 28 days, while the second Control positive group anemic rats were fed on the standard diet during the study period. Third group fed on standard diet and high dietary iron source as liver. Fourth group fed on standard diet and high dietary iron source as liver with given sucrose (13.8% of the diet). Fifth group fed on standard diet and high dietary iron source as liver and also were given vitamin A (4500 IU/kg feed). Sixth group fed on standard diet and high dietary iron source as liver and as well as calcium (1.2% of diet).

### **Biological evaluation**

During the experimental period (28 days), all rats were weighted once weekly. At the end of the experiment, biological evaluation of the different diets was carried out by determination of body weight gain % (BWG %), food efficiency ratio (FER) according to (Chapman *et al.*, 1959). Using the following formulas:

$$\text{BWG}\% = \frac{\text{Final weight} - \text{initial weight}}{\text{initial weight}} \times 100$$

$$\text{FER} = \frac{\text{Gain in body (g)}}{\text{Food intake}} \times 100$$

### **Biochemical Analysis:**

Determination of blood glucose: According to the method to (Yound, 1975). Determination of total cholesterol in serum was calorimetrically determined according to NIH, (1987). Triglycerides in serum were determined according to the method of (Fassati and Prencipe, 1982). Determination of HDL was carried out according to the method of (Gordon and Amer, 1977). The determination of VLDL and LDL were carried out according to (Lee and Nieman, 1966). Determination of liver function Alkaline phosphates (ALP), glutamic oxalocetic transaminase (GOT) or AST and glutamic pyruvic transaminase (GPT) or (ALT) according to (Tietz, 1976). Determination of alkaline phosphates according to (Belfield and Goldberg, 1971). Determination of kidney functions Serum creatinine according to (Bartles *et al.*, 1972). Serum uric acid according to Barham and Trinder,

(1972). serum urea according to (Fawcett and Soctt, 1960).

### **Statistical analysis:**

Statistical analysis were performed by using Computer Program Statistical Package for social (SPSS,), and compared with each other using the suite tests. All obtained results were tabulated. Statistical analysis has been achieved using IBM-PC computer by SPSS, program (SPSS, 1998).

## **RESULTS AND DISCUSSION**

For hematological profiles, results presented in Table (1) showed that there was insignificant differences in Hgb level among control negative group, liver group and liver plus Vitamin A group, moreover liver plus sucrose group had significant change at  $P \leq 0.01$  after dietary intervention by mean  $13.00 \pm 0.82$ ,  $13.05 \pm 1.29$ ,  $13.20 \pm 0.678$  and  $13.17 \pm 0.250$  G/dl, respectively. RBCs had insignificant differences among studied groups except liver plus sucrose group that significantly increased by mean  $20 \pm 0.54$  at  $P \leq 0.05$ . While PCV had significant changes among studied groups when compared to positive control group that significantly increased after dietary intervention by mean value of  $39.07 \pm 0.853$  ( $P \leq 0.001$ ),  $37.36 \pm 1.02$  ( $P \leq 0.05$ ),  $40.45 \pm 0.544$  ( $P \leq 0.001$ ) and  $41.00 \pm 1.25$  ( $P \leq 0.001$ ) for liver group, liver plus Ca group, liver plus Vitamin A group and liver plus sucrose group, respectively. The same line was observed in MCV. Otherwise in MCH, Ca plus liver group had insignificant differences ( $19.00 \pm 1.00$ ) while liver group, liver plus vitamin A group and liver plus sucrose group was significantly increased as compared to positive control group by mean of  $20.00 \pm 0.816$  ( $P \leq 0.05$ ),  $20.25 \pm 0.957$  ( $P \leq 0.05$ ),  $21.00 \pm 0.816$  ( $P \leq 0.01$ ) and  $18.00 \pm 1.00$ , respectively. Regarding to MCHC and PLT, all studied groups were significantly increased due to dietary intervention when compared to positive control group. The Iron deficiency is one of the most prevalent anemia as mentioned by (Abrishami *et al.*, 2013), also (Zhang *et al.*, 2013) showed that the mechanism of heme iron absorption is regulated by body iron status and dietary factors, that can influence heme iron absorption to varying

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**Table (1): Mean ± SD of hematological parameters in anemic rats after administered dietary intervention**

Hematological parameters		Control negative group (ve-)	Control positive group (ve+)	Dietary intervention groups			
				Liver group	Liver plus Ca group	Liver plus V.A group	Liver plus sucrose group
Hgb (N:13-18G/dl)	M±SD	13.00±.082	10.43±.832	13.05±.129	12.10±.788	13.20±.678	13.17±.250
	T-test	-7.77**	2.12 <sup>NS</sup>	-7.565***	-3.568*	-5.764**	-7.572***
RBC (N:4.5-6.5cmm/10 <sup>6</sup> )	M±SD	7.89±.061	6.88±.306	7.16±.055	7.09±.111	7.09±.147	7.20±.054
	T-test	-2.18 <sup>NS</sup>	.237 <sup>NS</sup>	-2.222 <sup>NS</sup>	-1.558 <sup>NS</sup>	-1.464 <sup>NS</sup>	-2.478*
PCV (38-48%)	M±SD	39.97±1.32	34.70±1.13	39.57±.853	37.36±1.02	40.45±.544	41.00±1.25
	T-test	-6.32**	-.762 <sup>NS</sup>	-7.734***	-4.273*	-10.693***	-8.066***
MCV ( N:76-96FL)	M±SD	50.25±1.25	49.66±.577	53.00±1.63	52.66±1.52	54.00±1.63b	55.00±1.15
	T-test	.200 <sup>NS</sup>	.742 <sup>NS</sup>	-3.941*	-4.497**	-5.118**	-8.576***
MCH (N:27-32pg)	M±SD	17.75±.957	18.00±1.00	20.00±.816	19.00±1.00	20.25±.957	21.00±.816
	T-test	.522 <sup>NS</sup>	.293 <sup>NS</sup>	-3.464*	-1.732 <sup>NS</sup>	-3.576*	-5.196**
MCHC ( N:31-35gm%)	M±SD	29.25±.957	29.00±1.00	33.75±.957	32.66±.677	33.50±1.29	34.00±1.82
	T-test	.522 <sup>NS</sup>	.522 <sup>NS</sup>	-7.550***	-7.738***	-5.892**	-5.000**
PLT ( N:150-450Thousands /cmm)	M±SD	287.00±2.44	288.33±1.52	333.25±3.09	331.33±4.16	333.75±2.75	334.50±2.64
	T-test	-.243 <sup>NS</sup>	-.255 <sup>NS</sup>	-26.921***	-23.750***	-30.052***	-31.572***

Hgb: Hemoglobin; RBCs: Red cell count;;Pcv: packed cell volume; Mcv: mean corpuscular volume ; Mch : mean corpuscular hemoglobin ; Mchc: : mean corpuscular hemoglobin concentration; PLT: Platelet count.

Significant values between periods analyzed by paired sample T-test.

Significant values between control positive group and other studied groups were analyzed by Independent T-test.

\*P≤0.05, \*\*P≤0.01, \*\*\*P≤0.001 and NS: Not significant

degrees. (Ahmed *et al.*, 1996) found that anemic patients have been significantly lowered serum retinol as well as lower packed cell volume (PCV), mean corpuscular hemoglobin concentration (MCHC), serum iron, TS, and higher serum total iron binding capacity (TIBC) as compared with those with normal hemoglobin levels. Moreover, the data of (Kamei *et al.*, 2013) indicated that there is an interaction between serum retinol and biochemical indices of iron nutrition, these data suggested that although the blood hemoglobin level itself remains unchanged during non-anemic iron deficiency, a variety of metabolic processes involved in the maintenance of the energy balance are altered. The results of (Kolsteren *et al.*, 2013) suggested that the addition of vitamin A to the treatment for anemia can be increased hemoglobin levels more than with iron alone. Moreover, (Barbosa *et al.*, 2013) suggested that the maintenance of adequate vitamin A intake should be encouraged which appears to be an effective intervention strategy to achieve appropriate serum retinol and hemoglobin values, and hence caused a lower iron deficiency anemia rates. The rate of hemoglobin rise is faster with intravenous iron sucrose therapy as compared to oral iron therapy (Gupta *et al.*, 2014). Moreover, ingestion of carotene-rich yellow improved the total-body vitamin A and Hb concentration and decreased anemia (Maramba *et al.*, 2010), also (Barbosa *et al.*, 2013) concluded that the maintenance of adequate vitamin A and meat intake in their population should be encouraged, which appears to be an effective intervention strategy to achieve appropriate serum retinol and hemoglobin values, and hence lower iron deficiency anemia rates. The effect of dietary intervention with liver and other nutrients on lipid profiles are presented in Table (2). Serum T.C. was decreased significantly among studied groups after dietary intervention when compared to positive control group. The mean value (mg/dl) was  $117.25 \pm 2.75$  ( $P \leq 0.001$ ),  $108.33 \pm 4.04$  ( $P \leq 0.05$ ),  $109.25 \pm 5.25$  ( $P \leq 0.05$ ) and  $111.25 \pm 2.21$  mg/dl ( $P \leq 0.01$ ), respectively. While Non significant changes were observed among studied groups in

serum T.G., as in serum HDL except liver plus sucrose group at  $P \leq 0.05$  by mean  $48.75 \pm 1.70$  mg/dl as compared to positive control group ( $45.00 \pm 1.00$  mg/dl). Furthermore, serum LDL and VLDL had insignificant differences in all over dietary intervention among studied groups, except liver plus vitamin A group was decreased significantly ( $40.00 \pm 2.16$  mg/dl) in serum LDL at  $P \leq 0.05$ . The findings of (Choi *et al.*, 2001) indicated that severe iron deficiency anemia is attended by decreased of serum total cholesterol and triglyceride concentrations. (Ece *et al.*, 2013) mentioned that high stored body iron, high serum iron concentrations and low iron binding capacity were found to be a risk factors for coronary heart disease. Iron-deficient diets have caused contradictory lipid changes in rats. Also, Iron loading is associated with altered lipid metabolism, but underlying mechanisms remain unknown. Lowering serum iron in rats reduced TG levels, this study explained the relationship between iron status and lipid metabolism and provides mechanistic support for interventions that reduce serum iron levels in individuals at risk for hyper-triglyceridemia (Aktas *et al.*, 2014). In parallel, iron deficiency anemia and hyper-lipidemia are common public health problems indicated a higher serum total triglyceride, total cholesterol and VLDL levels in iron deficient patients than the healthy controls. Hyperlipidemia appears to be a risk factor for premature cardiovascular diseases according to (Verma *et al.*, 2010), these findings indicated that iron deficiency anemia is attended by abnormal serum lipid profile, which responds significantly to iron therapy. In children deficient in vitamin A and iron, vitamin A supplementation mobilizes iron from existing stores to support increased erythropoiesis, an effect likely mediated by increases in circulating EPO. Also, HDL was increased and triglyceride was decreased significantly after treatment comparing to pre-treatment period (Tanzer *et al.*, 2001). The comparative effect of some nutrients on cured anemic rats was shown in Table (3). There was significant increase in serum iron among treated groups after dietary intervention, especially

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in liver plus vitamin A group and liver plus sucrose group at  $P \leq 0.01$  by means of  $0.85 \pm 0.13$  and  $0.95 \pm 0.13$  (ug/ml) then liver group and liver plus  $Ca^{+2}$  group at  $P \leq 0.05$  by means of  $0.85 \pm 0.13$  and  $0.67 \pm 0.057$  (ug/ml). Otherwise, non significant changes were observed among studied groups. For serum ferritine, non significant differ was illustrated on iron absorption from liver and source group in rats diet after dietary intervention. Moreover, comparative effect stated that liver plus sucrose group had a close mean of  $120.50 \pm 58$  ug/ml ( $P \leq 0.01$ ) to negative control group ( $119.00 \pm 1.6$  ug/ml),

then liver plus vitamin A group by mean  $120.00 \pm 82$  ug/ml ( $P \leq 0.05$ ). While liver plus  $Ca^{+2}$  groups had insignificant differed to positive control group by mean of  $118.00 \pm 1.00$  and  $117.66 \pm 1.15$  ug/ml, respectively. For TIBC, there was insignificant differed among treated groups that significantly changed when compared to positive control group. While liver plus vitamin A group and liver plus sucrose group were significantly improved at  $P \leq 0.01$  after dietary intervention at  $P \leq 0.05$  by means of  $3.30 \pm 29$  and  $3.35 \pm 51$  (ug/ml), respectively.

**Table (2): Mean  $\pm$  SD of lipid profiles in anemic rats after administered dietary intervention**

Lipids profile		Control Negative group (Ve-)	Control positive group (Ve+)	Dietary intervention groups			
				Liver group	Liver plus Ca group	Liver plus V.A group	Liver plus Sucrose group
S.TC (N:UPto200 mg/dl)	M $\pm$ SD	112.00 $\pm$ 1.63	102.00 $\pm$ 2.00	117.25 $\pm$ 2.75	108.33 $\pm$ 4.04	109.25 $\pm$ 5.25	111.25 $\pm$ 2.21
	T-test	-1.134NS	2.324NS	-9.527***	-3.392*	-2.636*	-6.718**
S.TG (N:UPto200 mg/dl)	M $\pm$ SD	62.00 $\pm$ 5.29	62.00 $\pm$ 2.00	62.00 $\pm$ 1.70	62.66 $\pm$ 1.52	60.75 $\pm$ 3.30	61.75 $\pm$ 2.21
	T-test	-1.400NS	-3.42*	-0.212NS	-0.480NS	0.678NS	0.182NS
S.HDL (N:35-100 mg/dl)	M $\pm$ SD	43.50 $\pm$ 1.29	45.00 $\pm$ 1.00	46.00 $\pm$ 1.82	44.33 $\pm$ 1.52	45.00 $\pm$ 2.16	48.75 $\pm$ 1.70
	T-test	-.522NS	-1.406NS	-1.000NS	-1.000NS	0.000NS	-3.962*
S.LDL (N:mg/dl)	M $\pm$ SD	41.00 $\pm$ 1.82	41.33 $\pm$ 1.52	41.50 $\pm$ 2.08	40.33 $\pm$ 2.51	40.00 $\pm$ 2.16	40.50 $\pm$ 2.88
	T-test	1.00NS	.974NS	-0.206NS	0.828NS	1.000NS	0.476NS
S.VLDL (N:mg/dl)	M $\pm$ SD	9.60 $\pm$ 6.27	11.60 $\pm$ 1.44	12.62 $\pm$ 2.21	11.83 $\pm$ 1.25	12.20 $\pm$ 5.88	11.92 $\pm$ 1.48
	T-test	.753NS	3.300*	-0.818NS	-0.288NS	-0.911NS	-0.343NS

S.cholesterol: Serum total Cholesterol; S. Triglycerides: Serum triglycerides; S.HDL: Serum HDL- Cholesterol; S. LDL: Serum LDL- cholesterol ;S. VLDL: Serum VLDL- cholesterol

Significant values between periods analyzed by paired sample T-test.

A significant value between control positive group and other studied groups was analyzed by Independent T-test.

\* $P \leq 0.05$ , \*\* $P \leq 0.01$ , \*\*\* $P \leq 0.001$  and NS: Not significant

**Table (3): Mean ± SD of anemic profiles in anemic rats after administered dietary intervention**

Anemic profiles		Control Negative group (Ve-)	Control positive group (Ve+)	Anemic groups			
				Liver group	Liver plus Ca group	Liver plus V.A group	Liver plus Sucrose group
S. Iron (N:0.4-1.3ug/ml)	M±SD	.450±.129	.633±.152	.850±.129	.666±.057	.850±.129	.950±.129
	T-test	-1.411NS	-1.300NS	0.837NS	-0.013NS	0.837NS	0.762NS
S.ferritin (N:20-280ug/ml)	M±SD	119.00±1.63	117.66±1.15	119.75±1.50	118.00±1.00	120.00±.816	120.50±.577
	T-test	.577NS	.878NS	-2.496*	-0.775NS	-3.873*	-5.196**
S. TIBC (N:1.6-5.0 ug/ml)	M±SD	1.42±.419	.933±.152	3.10±.496	2.86±.321	3.30±.294	3.35±.506
	T-test	.071NS	.652NS	-8.465***	-13.111***	-14.810***	-9.267***

S. iron :serum iron; s. ferritin: serum ferritin; s. TIBC: total iron binding capacity

Significant values between periods analyzed by paired sample T-test.

Significant values between control positive group and other studied groups were analyzed by Independent T-test.

\*P≤0.05, \*\*P≤0.01, \*\*\*P≤0.001 and NS: Not significant

(Devasenapathy *et al.*, 2013) reported that the current evidence on the safety and efficacy of intravenous iron sucrose on hematological and clinical outcomes. Though the evidence on its efficacy in improving hemoglobin and serum ferritin is convincing, (Vu'o'ng Lê *et al.*, 2011) which iron sucrose may be useful for iron deficiency anemia. (Gamble *et al.*, 2004) Although vitamin A deficiency, iron deficiency, and inflammation may contribute to anemia, their relative contribution to anemia, Michael *et al.*, (2006) mentioned that Vitamin A deficiency impairs iron metabolism; and vitamin A supplementation of vitamin A-deficient populations may reduce anemia (Kim *et al.*, 2013). On the other hand, (Barton *et al.*, 1983) showed that calcium significantly diminishes the absorption of ferrous and ferric iron in a dose related manner, whether the calcium is administered orally or introduced into isolated intestinal segments; these data suggested that individuals consuming a high-calcium diet contain marginal amounts of iron could develop iron deficiency anemia. (Mwanri *et al.*, 2000) reported that vitamin A may be needed for erythropoiesis, including the incorporation of iron for mobilization of iron from the spleen or liver

stores. Similar results were obtained by (Yeh *et al.*, 2004) who found that there was communication link between liver Fe stores and intestine Fe absorption. As suggested by (Anderson and Fitzgerald, 2010) who reported that vitamin A helps to release iron from stores and makes it more available for the body to use. The effect of dietary intervention with liver and other nutrients on biological value of studied rats were presented in Table (4). BWG and FER were significantly increased among studied groups except liver plus sucrose group and liver plus Ca<sup>2+</sup> group at P≤0.05 by mean 129.63± 20.43g and 87.01±26.96g, respectively when compared to positive control group. FI was significantly decreased among studied groups after dietary intervention when compared to positive control group, the mean values were 89.49±4.19 (P≤0.001), 86.52±3.54 (P≤0.001), 90.27±6.27 (P≤0.001), and 102.27±1.79g (P≤0.001), respectively. In the same line, the weight loss by 73.33% was found among IDA patients as reported by (Abu Syed *et al.*, 2014). According to the results of (Mahmud, 2004) showed an increase in FI in group of rats fed on liver organ.

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**Table (4): Effect of administered liver and some nutrients on anemic rats**

parameters	Positive control group (PCG)	Negative control group (NCG)	Liver group (LG)	Liver plus ca group (LCG)	Liver plus V.A group (LAG)	Liver plus sucrose group (LSG)
BWG (g)	84.87±25.11	91.00±30.47	106.75±32.40	74.25±19.56	98.25±26.66	129.63±20.43
			-0.708 <sup>NS</sup>	0.925 <sup>NS</sup>	-.358 <sup>NS</sup>	-2.11 <sup>*</sup>
FER	125.16±36.12	161.76±57.31	120.43±39.37	87.01±26.96	109.02±30.30	128.03±21.28
			1.18 <sup>NS</sup>	2.36 <sup>*</sup>	1.62 <sup>NS</sup>	1.11 <sup>NS</sup>
FI (g)	67.68±5.35	56.72±3.36	89.49±4.19	86.52±3.54	90.27±6.27	102.27±1.79
			-12.18 <sup>***</sup>	-12.19 <sup>***</sup>	-9.42 <sup>***</sup>	-23.91 <sup>***</sup>

B.W.G: body weight gain ;FER: Feed efficiency ratio; FI: feed intake

Significant values between periods analyzed by paired sample T-test.

Significant values between control positive group and other studied groups were analyzed by Independent T-test.

\*P≤0.05, \*\*P≤0.01, \*\*\*P≤0.001 and NS: Not significant

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## **تأثير بعض العناصر الغذائية على الحالة الصحية للفئران المصابة بأنيميا نقص الحديد**

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### **الملخص العربي**

الهدف الرئيسي من هذه الدراسة هو تقييم تأثير بعض العناصر الغذائية على الحالة الصحية للفئران المصابة بأنيميا نقص الحديد وتم إجراء التجربة على ذكور فئران الالبينو وعددهم ٦٠ وتتراوح أوزانهم بين ١٢٠±١٠جم) وتم تقسيمهم إلى ٦ مجموعات. أولاً مجموعة الكنترول السالبة. بينما المجموعات الأخرى تم إصابتها بأنيميا نقص الحديد بالتغذية على ٢٠ ملج تانيك أسيد لمدة ٢١ يوم متتالية. تم تقسيم الفئران المصابة بالانيميا لخمس مجموعات و تغذيتها على الوجبة القياسية بجانب مصدر عالي للحديد كالكبد. مجموعة الكنترول الموجبة، المجموعة الثالثة تم تغذيتها على مصدر عالي للحديد كالكبد فقط. المجموعة الرابعة تم تدعيمها بالسكروز بجرعة %13.8، المجموعة الخامسة تم تدعيمها بفيتامين أ بجرعة ٤٥٠٠ وحدة دولية، المجموعة السادسة تم تدعيمها بالكالسيوم بجرعة ١,٢%. وتوضح النتائج أن هناك ارتفاع معنوي في سيرم الحديد بعد تدعيمه بالمجموعات المعالجة خاصة مجموعة فيتامين أ . بالنسبة للفيريتين فالتأثير المقارن أوضح أن مجموعة السكروز أعطت أقرب متوسط للمجموعة السالبة ولكن مجموعة الكالسيوم لم تظهر أى نتائج معنوية بالنسبة للمجموعة الموجبة.

وتوصى الدراسة بضرورة تناول العناصر الغذائية التي تساعد على امتصاص الحديد والاستفادة منه كمصدر هيمي للحديد وهو الكبد وتناول فيتامين أ الذي يساعد على امتصاص الحديد بينما السكروز يعمل على زيادة الاستفادة من الحديد. أما الكالسيوم فيعمل على تقليل امتصاص الحديد.

الكلمات المفتاحية : الفئران – الكالسيوم –الكبد-فيتامين أ – السكروز –الهيموجلوبين-الحديد

