EFFECT OF SOME NUTRIENTS ON THE HEALTH STATUE 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 ratsfed on standard diet and high dietary iron source as liveralong 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 groupsupplemented with vitamin A(4500 IU/kg feed).Sixth groupsupplemented 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 ferritine, 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 hemi and hemi iron absorption, which makes it different from other inhibitors that affect non hemi 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 ExperimentalDesign:

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 negativegroupfed 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:

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

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

Biochemical Analysis:

Determination of blood glucose: According to the method to (Yound, 1975). Determination of total cholesterol in serum was calorimetrically determined according toNIHP, (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 (Grodon 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)orAST and glutamic pyruvic transaminase (GPT) or (ALT) according to (Tietz, 1976). Determination ofalkaline phosphates according to (Belfield and Goidberg, 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 IMB-P-C 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≤0.01 after dietary intervention by mean 13.00±.082, 13.05±.129, 13.20±.678 and 13.17±.250 G/dl, respectively. RBCs had insignificant differ among studied groups except liver plus sucrose group that significantlyincreased by mean 20±.054 at P≤0.05. While PCV had significant changes among studied groups when compared to positive control group that significantlyincreased after dietary intervention by mean value of 39.07±.853 (P≤0.001), 37.36±1.02 (P≤0.05), 40.45±.544 (P≤0.001) and 41.00±1.25 (P≤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±1.00) while liver group, liver plus vitamin A group and liver plus sucrose group wassignificantly increased as compared to positive control group by mean of 20.00±.816 (P≤0.05), 20.25±.957 (P≤0.05), 21.00±.816 (P≤0.01) and 18.00±1.00, respectively. Regarding to MCHC and PLT, all studied groups were significantlyincreaseddue 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 hemi iron absorption is regulated by body iron status and dietary factors, that can influence hemi iron absorption to varying

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

Hematological parameters		Control negative group (ve-)	Control positeve 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**	2.12NS	-7.565***	-3.568*	-5.764**	-7.572***
RBC (N:4.5-6.5cmm/10^6)	M±SD	7.89±.061	6.88±.306	7.16±..055	7.09±.111	7.09±.147	7.20±.054
	T-test	-2.18NS	.237NS	-2.222NS	-1.558NS	-1.464NS	-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**	-.762NS	-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	.200NS	.742NS	-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	.522NS	.293NS	-3.464*	-1.732NS	-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	.522NS	.522NS	-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	-.243NS	-.255NS	-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:

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 ironnutriture, 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 (Maramag *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±2.75 ($P\leq0.001$), 108.33±4.04 ($P\leq0.05$), 109.25±5.25 ($P\leq0.05$) and 111.25±2.21 mg/dl ($P\leq0.01$), respectively. While Non significant changes were observed among studied groups in serum T.G., as in serum HDL except liver plus sucrose group at $P\leq0.05$ by mean 48.75±1.70 mg/dl as compared to positive control group (45.00±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±2.16 mg/dl) in serum LDL at $P\leq0.05$. 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 serumiron 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

in liver plus vitamin A group and liver plus sucrose group at P≤0.01 by means of 0.85±0.13 and 0.95±.13 (ug/ml) then liver group and liver plus Ca^{+2} group at P≤0.05 by means of 0.85±.13 and 0.67±.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±.58ug/ml (P≤0.01) to negative control group (119.00±1.6ug/ml), then liver plus vitamin A group by mean 120.00±.82 ug/ml (P≤0.05). While liver plus Ca^{+2} groups had insignificant differed to positive control group by mean of 118.00±1.00 and 117.66±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 weresignificantly improved at P≤0.01 after dietary intervention at P≤0.05by means of 3.30±29 and 3.35±.51(ug/ml), respectively.

Table (2): Mean ± 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±SD	112.00±1.63	102.00±2.00	117.25±2.75	108.33±4.04	109.25±5.25	111.25±2.21
	T-test	-1.134NS	2.324NS	-9.527***	-3.392*	-2.636*	-6.718**
S.TG (N:UPto200 mg/dl)	M±SD	62.00±5.29	62.00±2.00	62.00±1.70	62.66±1.52	60.75±3.30	61.75±2.21
	T-test	-1.400NS	-3.42*	-0.212NS	-0.480NS	0.678NS	0.182NS
S.HDL (N:35-100 mg/dl)	M±SD	43.50±1.29	45.00±1.00	46.00±1.82	44.33±1.52	45.00±2.16	48.75±1.70
	T-test	-.522NS	-1.406NS	-1.000NS	-1.000NS	0.000NS	-3.962*
S.LDL (N:mg/dl	M±SD	41.00±1.82	41.33±1.52	41.50±2.08	40.33±2.51	40.00±2.16	40.50±2.88
	T-test	1.00NS	.974NS	-0.206NS	0.828NS	1.000NS	0.476NS
S.VLDL (N:mg/dl)	M±SD	9.60±6.27	11.60±1.44	12.62±2.21	11.83±1.25	12.20±.588	11.92±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≤0.05, **P≤0.01, ***P≤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'ngLê*et al.*,2011) which iron sucrose may be useful for iron deficiencyanemia. (Gamble *et al.*, 2004) Although vitamin Adeficiency, iron deficiency, and inflammation may contribute to anemia, their relative contribution to anemia, Michael *et al.*, (2006) mentioned thatVitamin A deficiencyimpairs 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 Fitzerald , 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 weresignificantlyincreased among studied groupsexcept liver plus sucrose group and liver plus Ca^{+2} 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 significantlydecreased 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.

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^{NS}	0.925^{NS}	-.358^{NS}	-2.11*
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^{NS}	2.36*	1.62^{NS}	1.11^{NS}
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***	-12.19***	-9.42***	-23.91***

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

REFERENCE

Abu Syed, M. M., H. Milton and M.M. Chowdhury (2014). Study of Hematological Parameters in Children Suffering from Iron Deficiency Anemia in ChattagramMaa-o-Shishu General Hospital, Chittagong, BangladeshVolume , Article ID 503981, 10 pages

Abrishami, F., A. Golshan and K. Briand (2013). Frequency of iron deficiency anemia in girls studying in Mashhad high schools.;Iran .J.Ped Hematology Oncol., 3(4):143-148.

Ahmed, F., M.R. Khan, R. Karri, S. Taj, T. Hyderi, M.O. Faruque, B.M. Margetts and A. A. Jackson (1996). Serum retinol and biochemical measures of iron status in adolescent schoolgirls in urban Bangladesh Eur. J. Clin Nutr., 50(6):346-351..

Aktas, G., A. Alcelik, A. Yalcin, S. Karacay, S. Kurt, M. Akduman, H. Savli and T. Clin (2014). Treatment of iron deficiency anemia induces weight loss and improves metabolic parameters*Am. J. Med.,* 45;165(2):87-89.

AIN (1993). Purified diet for laboratory: Final Report . American institute of Nutrition .J. Nutrition,123: 1939_1951.

Anderson, C. and C. Fitzgerald (2010). Iron ,An Essential nutrient . final report.

Benoist, B., E. McLean and I. Egli (2005). Worldwide Prevalence of Anemia. Geneva, Switzerland: World Health Organization.

Barbosa, F.P., R.C. Luna, M.G. Monteiro, L.S. Asciutti, R.S. Oliveira, A. S. Diniz, R.T. Lima, M. J. Benigna, R.M. Moraes, M. J. Costa and M. C. Gonçalves (2013). Relationship between hemoglobin, serum retinol and habitual meat consumption in the elderly: a population-based study. Arch Gerontol Geriatr.; 57(1): 60-65.

Barton, J.C., M.E. Conrad and R.T. Parmley (1983). Calcium inhibition of inorganic iron absorption in rats.Eur.J .ClinNutr;84(1):90-101.

Belfield, H. and D.M. Goidberg (1971). Alkaline phosphates colorimetric Method. J. Enzyme 12: 561.

Bartles, J., M. Bohmer and C. Heirli (1972). Determination of Creatinine.J.Clin. Chem, Acta, 37:193.

Barham, D. and P. Trinder (1972). Determination of Uric acid Analyst, 97:142.

Gamble, M.V., N. A. Palafox, B. Dancheck and M.O. Ricks (2004). Semba RDRelationship of vitamin Adeficiency, iron deficiency, and inflammation to anemia among preschool children in the Republic of the Marshall Islands Eur .J . Clin Nutr. Oct;58(10):1396-1401.

Cook, J.D., S.A. Dassenko and P. Whittaker (1991). Calcium supplementation—effect on iron-absorption. Am. J. Clin. Nutr; 53: 106–111.

Choi, J. W., S. K. Kim and S.H. Pai (2001). Changes in serum lipid concentrations during iron depletion and after iron supplementation Ann. Clin. Lab. Sci. 31(2):151-156.

Dlouhy, A. C.; Outten, A. and Caryn, E. (2013): "Chapter 8.4 Iron Uptake, Trafficking and Storage". In Banci, Lucia (Ed.).Metallomics *and the* Cell.Metal Ions in Life Sciences 12.Springer.

Devasenapathy, N., S.B. Neogi and S. Zodpey (2013). Is intravenous iron sucrose the treatment of choice for pregnant anemic women .J .Obstet Gynaecol Res. 39(3):619-626.

Ece, M.A., M.R. Yiğitoğlu, N. Vurgun and H. Güven (2013). Serum lipid andlipoprotein profile in children with iron deficiency anemia Işcan APediatr Int.;41:168-173.

Fassati, P. and L. Prencipe (1982). Determination of triglyceride .J.Clin . Chem., 28:2077.

Fawcett, J.K. and L. Soctt (1960). Determination of triglyceride.J.Clin.Chem.,28:2077.

Gupta, A., U. Manaktala and A.M. Rathore (2014). Randomized controlled trial to compare intravenous iron sucrose and oral iron in treatment of iron deficiency anemia in pregnancy.Indian. J.Hematol Blood Transfuse. ;30:120-125.

Grodon, T. and M. Amer (1977). Determination of HDL. J.Med., 62-707.

Hallberg, L., L. Rossander-Hulthen, M. Brune and A. Gleerup (1993). Inhibition of hemi-iron absorption in man by calcium. Br .J .Nutr.;69:533–540.

Kim, J., X. Jia, P.D. Buckett, S. Liu, C.H. Lee and M. Wessling (2013). Iron loading impairs lipoprotein lipase activity and promotes hypertriglyceridemia FASEB. J.; 27(4):1657-63

Kamei, A., Y. Watanabe, K. Kondo, S. Okada, F. Shinozaki, T. Ishijima, Y. Nakai, T. Kondo, S. Arai and K. Abe (2013). Influence of a short-term iron-deficient diet on hepatic gene expression profiles in rats. PLoS One.;8(6):e65732.

Kolsteren, P., S.R. Rahman, K. Hilderbrand and A. Diniz (2013). Treatment for iron deficiency anemia with a combined supplementation of iron, vitamin A and zinc in women of Dinajpur, Bangladesh. Eur.J. Clin Nutr.; 53(2):102.

Lee, R. and D. Nieman (1966). Nutritional Assessment.2 nd Ed., Mosby, Missouri, USA.

Michael, B., R. Zimmermann, R. Fabian, D. Abdeljawad, Z. Christophe, F. Richard, C. Hurrell and C. Nourredine (2006). Vitamin A supplementation in children with poor vitamin A and iron status increases erythropoietin and hemoglobin concentrations without changing total body iron© American Society for Clinical Nutrition,75(2):77-79

Mwanri, L., A. Worsly, P. Ryan and J. Masika (2000). Supplemental vitamin A improves anemia and growth in anemic school children in Tanzania .J.Nutr ., 130:2691-2696.

Mahmud, Abd EL-Hamid, AbdEl.Latef ,KHalil (2004). Nutrionalevaluation of some Organs that Commonly Consumed and its Effect on Health, Menofiya University, pages 45-46

Maramag, C.C., J.D. Ribaya-Mercado, P. Rayco-Solon, J.A. Solon, L.W. Tengco, J.B. Blumberg and F.S. Solon (2010). Influence of carotene-rich vegetable meals on the prevalence of anaemia and iron deficiency in Filipino schoolchildren Eur. J. Clin Nutr. 64(5):468-74.

NIHP (1986). Detection , Evaluation and treatment of high cholesterol in adults . national. Institute of Health publication , 88: 292.

Titez, N.W. (1976). Fundamentals of clinical chemistry . phi lade lphia ,W.B. Saunders ,P.243.

Tanzer, F., S. Hizel and O. Cetinkaya (2001). Serum free carnitine and total triglyceride levels in children with iron deficiency anemia EInt .J.Vitam Nutr Res.;71:66-69.

Vu'o'ng, L,ê., H. Khorsi, A.S. Villegier, V. Bach and Q. Gay (2011). New rat models of iron sucrose-induced iron overload.J.ExpBiol Med (Maywood).;236:790-799.

Verma, U., N. Shankar, S.V. Madhu, O.P. Tandon, N. Madan and N. Verma (2010). Relationship between iron deficiency anemia and serum lipid levels in Indianadults..J. Indian Med Assoc.;108:555-558, 562

Yound, D.S. (1975). Determination of GOT. J. Clin. Chem., 22:1-21.

Yeh, K.Y., M. Yeh and J. Glass (2004). Hepicidin regulation of ferroportin 1 expression in liver physicol.Eur.J .Clin Nutr 286: G385-94.

Zhang, Y., D. Zhao, J. Xu, C. Xu, C. Dong, Q. Liu, S. Deng, J. Zhao, W. Zhang and X. Chen (2013). Effects of dietary factors on the pharmacokinetics of 58Fe-labeled hemi after oral administration in normal rats and the iron-deficient rats.*Am. J. Clin. Nutr,* 153(1-3): 243-250.

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

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الملخص العربى

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. أولا مجموعة الكنترول السالبة. بينما المجموعات الاخرى 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 ٤٥٠٠وحدة دولية ,المجموعة السادسة تم تدعيمها بالكالسيوم بجرعة ١.٢%.

وتوضح النتائج أن هناك ارتفاع معنوى فى سيرم الحديد بعد تدعيمه بالمجموعات المعالجة خاصة مجموعة فيتامين ا . بالنسنة للفيريتين فالتأثير المقارن أوضح أن مجموعة السكروز أعطت أقرب متوسط للمجموعة السالبة ولكن مجموعة الكالسيوم لم تظهر أى نتائج معنوية بالنسبة للمجموعة الموجبة.

وتوصى الدراسه بضرورة تناول العناصر الغذائية التى تساعد على امتصاص الحديد والاستفادة منه كمصدر هيمى 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