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SOME BIOLOGICAL ASPECTS IN CONSEQUENCE OF STARVATION IN RATS

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ABSTRACT

Food is of greatest role on human health. One of the scientific ways to investigate this role is the starvation study. In the current investigation, 90 growing experimental rats were divided into three groups, isocaloric and two degrees hypocaloric ones. The biological parameters of these 100, 50 and 25 percentages of the used isocaloric diet, i.e., 410-calorie/100g diet, were conducted. The trial was carried out on those obese and lean animals of almost the same age. In general, the catch up growth during normal dieting was shown to be reversible except for those lean animals of starting body weight (IBW) less than 70% compared to the control. The less degree of starved rats died at 70% FBW /control FBW, within eight weeks, meanwhile, the sever one although died two weeks earlier, but at higher FBW/cFBW ratio of 80% assuming a risk to some biological factor other than loosing weight. To put . more clearly, factor other than the shift in base metabolic ratio (BMR) may posses the main reason to death. In conjugation, the total plasma cholesterol (TC) elevations were positively related to the hypocaloric degree in diet and become more remarkable with time, eg, 130 to 160% norms. These metabolic changes must be correlated with the damage of body tissues and cells out of hunger or using the protein more frequently. This sort of abnormal cholestrolemia has been shown to be associated with organs enlargement. In this regard, all types of organ weight have been extinct in range from 130% to almost 330% comparing to the normal.

The other metabolites that deviated with sever reduction in energy were the elevation of blood total protein. Similar data have been recorded for creatinine. In contrary, enzymes of liver functions have been found to go another way.

More accurate investigation is conducted to biologically explore this metabolic conjugation. It is clear that this emergence catabolic status of proteins (EMS) save the animals, but its existence for longer time may posses a reverse effect. In another words, this EMS is a sort of hormonal oxidative imbalance, which abuses the biological system at the long run.

INTRODUCTION

Although the food production has markedly increased according to the world total production, several areas in this earth are suffering from food shortage. Understanding the biology of hunger might be one step to rescue those people wherever they are. Although starvation and hypocaloric dieting are often be considered to produce a progressive reduction in base metabolic ratio (BMR) and nitrogen loss (NL), it might be vital for the long run. However, with starvation progress and lean body (LB) mass decrease, both BMR and protein oxidation decrease. The metabolic response to starvation up to several weeks between lean and obese animals were observed showing that the death in severe hypocaloric dieting is due to an increase in BMR whatever was the initial body weight (IBW) (Elia, 1991). It has been very early to state

that one of the most consistent autopsy findings of animal and human who died of pure starvation is the virtual absence of depot fat, both subcutancously and inernally (Keys *et al.*, 1950).

Most of the body weight (BW) loss during starvation in lean individuals is due to loss in body mass. A variety of autopsies study in human have shown that 25 to 50% of most lean tissues and organs are lost during starvation. However, brain, gondals and skeleton appeared to be preferentially preserved (Elia, 1991).

In subjects ingesting hypocaloric diets, the effect of initial adiposity on protein turn over (PTO) and composition of N loss in relation to other confounding variables, e.g. composition of dieting, exercise, etc. is not fully understood. For example, the man died within 63 days after refusing food, lost about 41% of his body weight. In contrast, there are several reports of successful total fasts in obese individuals who have lost more than 50% of their BW (Forbes and Drenick, 1979). If starvation had continued until death, most probably the weight loss could have been substantially more.

Terms of refeeding might be vital. Some workers favor the non-ketogenic diets since they have observed that isocaloric replacement of fat for CHO is associated with improved N balance (Vasqueez and Adibi, 1992). Diets containing more CHO, however, have been reported to be associated with greater hunger (Davies *et al.*, 1984). Attkin's diet is a suitable answer for such scientific confuse.

Biologically, starvation stress has been explored at level of enzymes, hormones and genetics most recently (Harbison, *et al.* 2005; Mackay, 2005; Lee *et al.* 1999, Lee *et al.* 2000; Schadt *et al.* 2003; Teichert *et al.* 1989; Zinke *et al.* 2002; Nelson *et al.* 1995; and Seglen and Bohley 1992). These studies involved man, animals, insects and microorganisms.

Here, this investigation try to observe the metabolic changes in conjugation of starvation in order to figure out the reality of metabolic relation between severe hunger, dieting and being alive. The information in designing this sort of studies may help the UN experts to treat such human disasters.

MATERIALS AND METHODS

Animals and Rationales:

Male Albino adult rats sorgue-dwley western strains aged from 2 to 3 months have been selected of average weight ranged from 78 +/- 5g for lean to almost 170 +/- 5g for the obese. These (15 x 6)=90 animals were obtained from biological unit of FTRI. All rats fed on basal diet consisted of 10% protein, 10% cottonseed oil, 5% cellulose, 4% salt mixture, 1% vitamin mixture and 70% corn starch of almost 410 calorie / 100g diet (Lane-Peter and Pearson, 1971). After an adaptation period of 10 days they were divided into three group as follows: the negative control (NC): 15 animals (5 subgroups) fed on complete basal diet as isocaloric diet, another same number and weight group was fed on 50% less diet. The third groups were similar animal's weights feed on 25% of the basal diet. In all cases, subgroups were organized according to most obese till the most lean body animals. Data have been collected as

mains for triplicates. The most obese group has been considered to be the control.

Methods:

Estimating body weight gain (BWG) according to Chapman *et al.* (1959) has been carried out as the main factor for the biological evaluation. The blood picture in rat's specimens was examined as described by Dacie and Lowis (1984). Total cholesterol in serum was determined enzymatically by kit based on the technique of Richmond (1973). The alanine amino transferase (ALT) and aspartate amino transferase (AST) were measured colorimetrically as described earlier (Richman and Frankely, 1957). The blood protein content was a colorimetric assay first used by Gornal *et al.* (1949). The creatinine measurement has been carried out according to Bartles, *et al.* (1972).

Data have been expressed as main of triplicates.

RESULTS AND DISCUSSION

The metabolic response to starvation up to several weeks both for lean and obese rats was observed. This experiment involved three degrees of protein-energy: normal and less than normal (PEM) dieting. Data, therefore, was divided into three parts based on the leves of calorie in diets, eg 410, 205 and 102.5 calorie / 100g diet. Those are shown in Tables 1, 2 and 3, respectively. Table 1 shows the growth of these rats of different initial body weight (IBW) that fed on isocaloric diet.

Table (1): The body weight response to isocaloric diet in rats of the same age and different initial body weight.

IBW(g)	IBW/ c IBW*	2 wk	4wk	6 wk	8 wk	10 wk	FBW/ cFBW
		% of each IBW					
171	100	108	114	122	131	135	100
148	87	109	116	122	124	128	95
130	76	104	110	112	117	121	89
112	65	112	121	134	147	152	81
79	46	119	130	152	167	188	62
Mean (g)128	100%	139	149	160	170	179	85

I BW, clBW (171) and FBW are initial, control and final body weight., respectively
Means of 6 rats

It seems that BW recovery may reestablish under this sort of nutrition within 10 weeks. The catch up growth may be, as clear in the table, reversible except for those lean animals starting BW less than 70% compared to the control. Notice that we have taken the most obese one as control here. Those animals make only 60% FBW of this control and greatly being lower than the mean growth rate of 85%, no matter was its high rate of growth. Therefore, lean animals have made the highest rate of growth under this condition but did not reach the normal FBW (cFBW). This could be attributed to the variation in requirements based on the variation in body mass. In some other wards, the

catch up growth was most likely hypertrophic growth and does not compensate the hyperplasia one in this age cycle or that physiologic condition.

In Table (2), in case of 50% less diet feeding, lean animals again exhibited higher rates of growth, meanwhile all animals, e.g. fatty or leans, have been died in two months at about 65 to 70% of their FBW to that of the control (FBW/cFBW). Less than 8 weeks was the possible duration time to stay alive when animals diet was reduced to be 25% of the basal diet. More accurately, most lean animal died few days before, as seen in Table3. However, Forbes (1987) has suggested that hypocaloric-dieting contribution to fat-free subject is greater than in obese ones to loss BW. Generally, as seen in Table 3, although lean animals may gain as seen in Table 3relatively rapid growth or use energy more efficiently, they can't still alive longer than the obese animals.

These findings have been detected earlier, Henry (1990) stated that death from starvation, or even semistarvation, frequently occurs when subject reaches a final body mass index (BMI) of 10-14. In this regard, man of BMI of 20.75, for instance, is of fat ranged from 8.1 to 10.1 Kg as calculated based on weight and height formula of Garrow and Webster (1985) and Black *et al.* (1985).

Table (2): The body weight response to a 50% hypocaloric diet in rats of same age and different initial body weight.

IBW(g)	IBW/ cIBW	2 wk	4 wk	6 wk	8 wk	10 wk	FBW/ cFBW
		% of each IBW					
173	100	90	90	84	64	-	100
150	87	90	97	92	71	-	87
127	73	89	96	86	65	-	78
113	65	91	99	90	75	-	77
84	49	94	92	92	89	-	70
Mean (g) 129	100%	116	115	112	91	-	82

I BW, cIBW (173g) and FBW are initial, control and final body weight.
Means of 6 rats

Table (3): The body weight response to a 75% hypocaloric diet in rats of same age and different initial body weight.

IBW(g)	IBW/ NIBW*	2 wk	4 wk	6 wk	8 wk	10 wk	FBW/ cFBW
		% of each IBW					
171	100	71	85	88	-	-	100
153	89	78	72	68	-	-	86
131	77	86	71	76	-	-	78
109	64	92	88	66	-	-	77
83	48	91	85	59	-	-	70
Mean (g) 129	100%	108	106	100	-	-	82

I BW, cIBW (171g) and FBW are initial, control and final body weight.
Means of 6 rats

Likewise, and in a human case of death due to starvation, the structural and storage fat left is only about 1 Kg, it can be calculated that this average daily energy expenditure (EE) was 1300 to 1600 K cal/d that makes some of 16-19% protein EE. Certainly, age, fat mass, genetic and degree of starvation influence the shape and speed of death process.

Dose this a kind of nurture or nature with up or down-regulation or all together. More recently, Lee *et al.* (1999) observed the gene expression profile of aging and its retardation by caloric restriction assuming a genetic role, even on animal brain (Lee *et al.*, 2000).

Table 4 has been depicted based on the mains of the last three tables for comparison. Since the first sort of nutrition resulted in a positive gradual catch up growth, the second type possess a negative one ended with death in 8 weeks for 50% diet level group at particular BMI of about 70%. Actually, the 25% level of diet, rats died some more weeks earlier, i.e. after 6 weeks, due to the severe less in energy in diets making BMI of more than 80% of the normal. Considering that, a risk to special biological factor other than loosing weight is to be followed. One explanation is that the animals have retained lower BMR in the beginning of starvation then an obligated relatively higher one has existed after proper level and period of starvation (Elia, 1991). Again, factor other than this shift in BMR or loosing weight may posses the main reason to death. Death, however, is not connected only with losing fat free bodies as stated before (Elia, 1991).

Table (4): The differences in body weight response between isocaloric and hypocaloric diets in rats.

Group	energy*	IBW (g)	IBW %	2 wk	4wk	7wk	8wk	10wk	FBW/ cFBW
				% to each group					
Control (100% diet)	410	129	100	110.3	117.6	126.6	134.5	141.6	100
50% diet	205	128	100	91.6	91.0	88.8	72.2	-	72
25% diet	102.5	129	100	85.6	84.0	79.4	-	-	80

I BW, cFBW and FBW are initial, control and final body weight. *cal/ 100g diet
Means of 6 rats

Learning from the nature, Escherichia coli bacterium is anything but idle when its environment shifts from nutrient rich to nutrient poor. The new findings show that a string of phosphate residues in E. coli kicks the Lon protease into action. This enzyme then chops up ribosomal proteins to release free amino acids that are used to manufacture biosynthetic enzymes, which help the bacterium to adjust to its nutrient-poor environment (Gottesman and Maurizi, 2001).

In addition to the protein and fat catabolism, some other biological consequences must be happened. However, let us figure out some of which. Hence metabolic response to starvation are studied for lean and obese animals observed in terms of BWG and organ changes, total cholesterol (TC) has been recorded for these types of feeding indicated in Table 5 as well.

Surprisingly, a TC elevation was correlated with the hypocaloric degree in diet and become more remarkable with time. This metabolic change may correlate damage of body tissues and cells out of hunger. It is a fact that changing in uncontrollable cholesterol may reflect almost pathological disorder. This sort of abnormal cholestrolemia has been associated with organs enlargement as well as an emergence metabolic status (EMS). Actually, Tables (5) and (6) show this kind of association. In this regard, all types of organ weight have been extinct in range from 130% to almost 330% compared to the normal. Hypercholesterolemia, for instance, has been found to correlate liver enlargement (Hamza *et al.*, 2003).

The other metabolites deviated with severe reduction in energy were the elevation of blood total protein. Protein catabolism is important, not only as an alternative source of fuel, but also as a source for amino acids. Teichert *et al.* (1989) mentioned that Lysosomal (vacuolar) proteinases of yeast are essential catalysts for protein degradation, differentiation, and cell survival. Similar data have been recorded for creatine. In contrary, enzymes of liver functions have been found to go another way with insignificant changing. Actually, one purpose of EMS is to secure a steadily proper secretion of these enzymes (Gottesman and Maurizi, 2001). It can be concluded however from Table (7) that the balanced diet is necessary to sustain important functions of liver, which role up the nutritional homeostatic system which may manifested by a proper hormonal secretion and balance.

In fact, the EMS connected with starvation as mentioned above involves special hormonal imbalance. For example, Nelson *et al.* (1995) suggested a neuroendocrinal involvement in aging. They recorded evidence from studies of reproductive aging and caloric restriction to establish their hyposes. However, the growth hormones and insulin would play important role in correcting this sort of metabolic deviation. Growth hormone (GH) acutely stimulates forearm muscle protein synthesis in normal humans. The short-term effects of GH on skeletal muscle protein synthesis and degradation in normal humans are unknown (Fryburg *et al.*, 1991).

Table (5): The total cholesterol response to hypocaloric dieting in rats.

Group	Total Cholesterol (mg/dl)*					
	Zero	Control %	2wk	Control%	4wk	Control%
Control (100% diet)	65.00	100	64.91	100	65.44	100
50% diet	64.90	100	83.05	130	95.01	150
25% diet	64.94	100	87.60	135	105.78	160

*Means of 6 rats.

Table (6): Organ weight as affected by hypocaloric diet in rats.

Organs* Diet	Liver (g)	Heart (g)	Spleen (g)	Kidneys (g)	Lungs (g)	Brain (g)
Control (100 diet)	2.16	0.198	0.168	0.490	0.300	0.570
50% diet	2.76	0.350	0.260	0.770	0.800	1.830
25% diet	2.94	0.561	0.315	0.892	1.236	2.429

*Means of 6 rats.

Table (7): Some biochemical parameters* associated with hypocaloric diet in rats.

Parameters / Diets	Total protein	Creatinine	ALT	AST
	mg/dl		U/dl	
Control(100%diet)				
Initial time	6.15	0.21	23.96	54.33
Final time	6.08	0.21	23.45	55.76
50% diet				
Initial time	6.96	0.27	22.59	45.57
Final time	6.72	0.25	21.77	42.62
25% diet				
Initial time	6.36	0.29	19.87	52.39
Final time	6.13	0.28	19.65	43.68

*Means of 6 rats.

Likewise, we believe that the EMS is mainly a sort of catabolism ruled up by hormones such as epinephrine. This may explore the raising up of TC. The elevation of TC for some extant has been found to associate organ enlargement and hormonal imbalance (Ahmed *et al.*, 2005). Fernandez *et al.* (1995), for instance, studied the effect of epinephrine administration on the metabolism of red and white muscle and found it inducing plasma metabolites with an overall significant glycogen depletion. Dietary therapy of muscle degradation with aging is not easy due to hormonal deviation, but starving and semistarving data may help in aging studies.

Terms of refeeding might be vital. Some workers favour the non-ketogenic diets since they have observed that isocaloric replacement of fat for CHO is associated with improved N balance (Vasqueez and Adibi, 1992). Diets containing more CHO, however, have been reported to be associated with greater hunger (Davies *et al.*, 1984). Attkin's diet is a suitable answer for such scientific confuse.

The increase in muscle strength and size, however, was not influenced by the predominant source of protein consumed by older men with adequate total protein intake (Haub, *et al.*, 2002). Meat consumption may enhance protein synthesis and muscle hypertrophy by providing creatinine. This could be one of the dietary solutions. But, to control the action of epinephrine, insulin alone might not help as a main anabolic direction to cure. The cytoplasmic serine-threonine kinase (S6K1) is critical for transitional regulation of genes that encode essential components of the protein synthesis apparatus. The protein and insulin administration regulate S6K1 activity in skeletal muscles (Bigot *et al.*, 2003).

In conclusion, this study may prove another biological parameters for death other than loosing BW. Basically, BMR is theoretically decreased in the beginning of starvation, but death may become the final event as a result of an uncontrollable increase in BMR again. According to the data revealed here, another biological consequences is more accurate. A metabolic systemic deviation must be carried out due to unusual type of hard work that temporarily takes place or carried out under this type of stress. According to these findings, a protein catabolic pathway (EMS) that runs for longer time is the real reason for death. These proteins turn over such high rates beyond the

biological capacity of both intracellular and extracellular compartments negatively affect the whole biological system. It is clear that this emergence catabolic status of proteins save the animals in short run, but its existence for longer time may posses a reverse effect. This EMS again is a sort of hormonal oxidative imbalance, which abuses the biological system at the long run

Actually, this hormonal oxidative imbalance induced by severe hungers can not be corrected without a sort of an urgent dietary therapy. For example, Rodriguez *et al.* (2002) found that olive oil induced an up regulating effect on uncoupling protein gene mRNA that was probably not mediated by systemic metabolic changes, but rather related to local effect on interscapular brown adipose tissue and skeletal muscle. Moreover, de Jonge, *et al.* (2002) studied the genetic expression effect of food component. Overexpression of arganise I in enterocytes of transgenic mice has been found to elicits a selective argenine deficiency and affects skin, muscles, and lymphoid development.

The death due to long time of starvation, therefore, involves immune dysfunction associates the hormonal imbalance and the fallen down of organ expectancy. Liver, in particular, is a real scientific adjunct to that issue due to its role in protein synthesis. Undernutrition compromises barrier function, allowing easier access by pathogens, and compromises immune function, decreasing the ability of the host to eliminate pathogens once they enter the body. Along with undernutrition, infection is becoming the primary cause of morbidity and mortality in the developing world. Complex interactions existed between these two threatening problems (Calder and Jackson, 2000). Further investigation should be designed to accurately help and secure hunger people. More accurate investigation is conducted to biologically explore this metabolic conjugation, in which, more metabolic pathways are observed, besides, new dense diet of proteins and antioxidants from animal and plant origin is tolerated.

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بعض المظاهر البيولوجية في الفئران المغذاه على عليقة تجويع منخفضة الطاقة

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قسم الأغذية الخاصة و التغذية – معهد بحوث تكنولوجيا الأغذية – مركز البحوث الزراعية

***قسم الاقتصاد المنزلى – كلية التربية النوعية بدمياط – جامعة المنصورة**

يهدف البحث لدراسة تأثير التغذية على علائق منخفضة فى محتوى الطاقة على فئتين من فئـــران التجارب (نحيف – سمين) وتم تقسيم الفئران الى ثلاث مجموعات:– المجموعة الأولى (الضابطة) وغذيت على عليقة مكتملة الطاقة (٤١٠ سعر حرارى/١٠٠ جرام عليقة)، المجموعة الثانية وغذيت على عليقة تقل فى محتوى الطاقة بمقدار ٥٠% (٢٠٥ سعر حرارى/١٠٠ جرام عليقة) ، المجموعة الثالثة وغذيت علـــى عليقة اقل فى محتوى الطاقة بقدار ٧٥% (١٠٢,٥ سعر حرارى/١٠٠ جرام عليقة) مقارنا بالكنترول.

ولقد اظهرت النتائج موت فئران المجموعة الثالثة فى الاسبوع السادس ، بينما حدث هـــذا فـــى الاسبوع الثامن فى المجموعة الثانية.

ولقد افترض انه بالرغم من انخفاض معدل التمثيل الغذائى فى بداية التغذية فى المجموعتين الثانية والثالثة الا ان حدوث موت الفئران فيهما ربما يرجع لزيادة معدله مرة اخرى.

ويلاحظ ارتفاع نسبة الكوليسترول الكلى فى المجموعتين الثانية والثالثة بمقدار ١,٣ ، ١,٦ علـــى التوالى مقارنا بالكنترول وقد ترجع هذه الزيادة الى حدوث تحطم فى بعض أنسجة الجسم والخلايا فى حالة نقص الغذاء.

كذلك وجدت بعض التغيرات البيولوجية الاخرى ، مثل ارتفاع نسب كلا من البروتينات الكلية للدم ، انزيمات الكبد ، الكرياتينين. بالاضافة لحدوث تضخم فى بعض الاعضاء الداخلية لفئـــران الجمـــوعتين الثانية والثالثة وكان بمعدل ٣٣٠ و ١٣٠% بالمقارنة بالكنترول ، بينما يحدث ضمور فى العضلات .

ومن ذلك يتضح ان توازن العليقة فى محتواها من الطاقة يلعب دورا هاما فـــى ضـــبط التـــوازن الهرمونى بالجسم ، وبالتالى مساعدة الكبد واعضاء الجسم الاخرى على القيام بوظائفها الاساسية.

و يري الباحثون ان الحاله الايضية الطارئه حالة هرمونيه لهدم البروتين و يؤجلها حجم مخـــزون الدهن وهى تنقذ حياة الكائن الحي الا ان استمرار هذة الحاله لفتره طويله تؤدي الي الوفاة. وقد يرجع هذا الى اختلال نسب النواتج الايضية النيتروجنية بين السائلين الخلوى والبين خلوى فوق طاقة اعضاء الجســـم على تعديلها. ومن المفترض أيضا نفك مخزون الجسم من مكونات توجد فى الغذاء تلعب دورا محوريا على مستوى الفعل الجينى مع هبوط حاد فى توافق الجهاز المناعى.