

Journal of Chemical, Biological and Physical Sciences



An International Peer Review E-3 Journal of Sciences

Available online at www.jcbpsc.org

Section B: Biological Sciences

CODEN (USA): JCBPAT

Research Article

Obesity outcome in mothers independent of dietary fat type after birth but during pregnancy period

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Received: 26 July 2014; Revised: 08 August 2014; Accepted: 16 August 2014

Abstract: The maternal obesity have adverse effect on mother and neonatal health also developed serious metabolic risks in mother life related to change in adipose metabolic process. The aim is to study the effect of dietary fat types on obesity development during pregnancy and the role of Leptin to modulate energy balance after delivery and correlation with metabolic problems. Female Wister rat feeding during the pregnancy period (3 weeks) on one of the following diets: control diet (10%fat: tallow), high fat diet (45%fat: tallow), moderate saturated fat diet (22.5%fat: tallow) and moderate unsaturated fat diet (22.5%fat: olive oil). After delivery, dams exposure to control diet for 4 weeks, gestational weight gain and postpartum body weight were recorded, plasma analysis were measured pre and postpartum periods.

Feeding dams during pregnancy with saturated fat diets (45%, 22.5%) increased gestational weight gain as higher energy consumed and elevated glucose, lipoprotein, Leptin levels and reduced Insulin sensitivity by reducing HOMI even when exposure postpartum to control diet, the previous parameters still high (W: 57.871±5.422 gm, 47.923±6.110 gm; Glu: 16.033±0.472, 14.433±0.399; T.ch: 2.276±0.022, 1.790±0.024; TG: 1.263±0.020, 1.135±0.010 mmol/L; Lep: 6.876±0.020 ng/ml, 4.832± 0.010 ng/ml; HOMI: 26.702 ± 0.200, 23.590±0.219) compared to control group, while feeding dams with unsaturated fat diet (22.5% olive oil) showed favorable effect on gestational weight and weight gain, plasma analysis including lipoproteins, Leptin, Insulin and HOMI levels pre and postpartum periods (Pre: W: 315.66±9.127gm, 36.855±5.299 gm; Glu:

13.233±0.100; T.ch: 1.141±0.011; TG: 0.745±0.017 mmol/L; Lep: 2.759±0.009 ng/ml; Insu: 1.379±0.004 ng/ml; HOMI:17.659±0.122) (Post: W: 319.397±6.835 gm; Glu: 13.033±0.457; T.ch:1.270±0.031; TG: 0.889±0.011 mmol/L; Lep: 3.184±0.009 ng/ml, Insu: 0.803±0.011 ng/ml, HOMI:10.127±0.311). The nutrition type during pregnancy influence on dams' metabolic process, Leptin and Insulin sensitivity that effect on energy expenditure and body weight even when change dietary fat type and proportion after delivery, referring that the feeding through prepartum period is important in development of maternal obesity.

Keywords: Maternal obesity, pre and postpartum Leptin and Insulin levels, metabolic risks.

INTRODUCTION

Maternal obesity is one of the most public health problems that threat women during gestation period¹, it associated with serious metabolic syndromes for both mothers and developing fetus that included gestational diabetes, delivery outcomes, preeclampsia, hypertension² which reflected in childhood obesity and diabetes^{3,4}. The excess weight gain during pregnancy increased in mothers the risk of venous thrombosis⁵, coronary heart disease⁶ high incidence of macrosoma^{7,4} and developed type 2 diabetes within following ten years⁸. The distinct metabolic state of adipose tissue is responsible for maternal health problems during pregnancy^{3,9}.

The adipose tissue (visceral fat) had endocrine and immunological function in addition to adiposity¹⁰, one of the secretion hormone is Leptin which interact with Insulin to modulate energy homeostasis that regulate body weight¹¹. The saturated fatty acids had deleterious effect on body weight by increasing fat deposition¹² because have low oxidative rate¹³ that correlated positively with plasma lepin and caused Leptin resistance a characterization of human obesity¹¹ therefore the obesity during gestation mediated by differences in metabolic process arising from body fat deposition in addition to distribution¹⁴. The control of body weight through pregnancy is an important step for pregnancy safe, maternal and neonatal health. The few recent studies either reported to the metabolic abnormalities of maternal weight gain or to the recommendations for weight management, there is no study referred to the metabolic syndromes resulted from weight gain after pregnancy or the activity of Leptin and Insulin on postpartum body weight or the role of these hormones to manage weight after delivery associated with appropriate diet, therefore the aim of the research is to study the effect of dietary fat consumption on body weight pre and postpartum periods and the role of adiposity hormone in weight gain and metabolic abnormalities.

MATERIALS AND METHODS

Diet: Diet induced obesity in rodents (HF 45 % fat) and it's control (LF 10 % fat) was formulated according to the research diet INC USA¹⁵. Moderate diet M.d with saturated or unsaturated fat (M.d-SFA, M.d-UFA) was modifying according to the high fat diet of this study. The composition of the experimental diets shown in **Table1**.

Table 1: composition of the experimental diets in the study.

Ingredients	Control (10% fat)	HF (45% fat)	M.d (22.5%) tallow or olive oil
Casein	200	200	200
L- cystine	3	3	3
Cornstarch	315	72.8	286.55
Sucrose	385	272.8	286.55
Cellulose powder	50	50	50
Vegetable oil	25	25	25
Beef tallow	20	177.5	76.4*
Mineral mix	10	10	10
Dicalcium phosphate	13	13	13
Calcium carbonate	5.5	5.5	5.5
Potassium citrate	16.5	16.5	16.5
Vitamin mix	10	10	10
Choline bitartrate	2	2	2
Total weight gm	1055	858.1	984.5
Total Kcal	4057	4057	4057.3
Total Kcal/ gm	3.85	4.73	4.078

* use beef tallow or olive oil in the diet.

Animals: Female Wister albino rats (216±3 gm weight) were acclimatizing on low fat diet for one week. The virgin female rats were time mated by monitored oysters cycle in vaginal smear before introducing to the male (one male for each female, aged 17 -18 week). Day one of pregnancy was determined by the present of spermatozoa. The females feeding during pregnancy either on low fat diet (control diet LF: 10 % energy from tallow) n=12 or on a high fat diet (HF 45% energy from tallow) n=12, or received moderate fat diet (22.5 % fat) either tallow (M.d-SFA) n=12 or olive oil (M.d-UFA) n=12 through gestation period (3 weeks).

Pregnant rats were housed in group (n=3 each cage) in standard cage, containing sawdust and maintained on their assigned diet with free access to water. All animals were kept in constant room temperature (25-30C°) and 12:12h light: dark cycle with free access to food and water. During the gestation period, daily food intake and body weight were recorded for each group. The energy intake was calculated¹⁶: Energy intake = food consumed (gm) × total kcal/gm diet. Pregnant rats from each group either allow to get birth or sacrificed (n=3) in the day 18 of gestation after anesthetizing with pentobarhital sodium 60 mg/kg body weight, blood samples were collected and plasma stored at -78C° in deep freezing for biochemical and hormonal measurements. On the days 21 and 22 of pregnancy, animals were monitored and observed during delivery. After delivery dams' body weight were recorded. The dams from each dietary group were postpartum feeding with control diet for 4 weeks, at the end of the experimental period, dams body weight were recorded and sacrificed (n=3), blood samples were collected and stored at -78C° for plasma analysis.

Plasma analysis: Plasma glucose, total cholesterol (T-ch), triglycerides (TG), High density lipoprotein (HDL) concentrations were measured by enzymatic method using diagnostic Kit from Randox (UK) and Biolabo companies (France). Plasma rat Leptin and Insulin concentrations (ng/ml) were measured using Rat Elisa kit from CRYSTAL CHEM INC. The homeostatic index of Insulin resistance (HOMA-IR) was calculated according to the following equation¹⁷: $HOMA-IR = [Glucose (mmol/L) \times Insulin (pmol/L)] \div 155$. Insulin converting to pmol/L: multiplying by 150¹⁸.

Statistical analysis: Data were analyzed by one-way ANOVA or general liner model procedure using SPSS version 15 statistic program. Comparisons between means were made using least significant differences (LSD). Differences were considered to be significant at $p < 0.05$. Data are presented as means \pm standard deviation.

RESULTS

Maternal food consumed and energy intake: The female rats consumed high fat diet during pregnancy period showed significantly ($p < 0.05$) low rate of food consumption (125.663 ± 5.275 gm) compared to all groups but higher energy intake (595.0 ± 7.241 kcal/3 weeks) as high fat percentage in their diet, following by dams in M.d-SFA diet (133.331 ± 5.470 gm, 538.0 ± 8.100 kcal/3 weeks) with significant between the two groups. No significant differences in maternal consumption between control and M.d-UFA diets (129.59 ± 5.773 gm, 135.091 ± 7.100 gm) while the energy intake increased significantly ($p < 0.05$) in the last group 529.67 ± 7.560 kcal/3 weeks (**figure 1 and 2**).

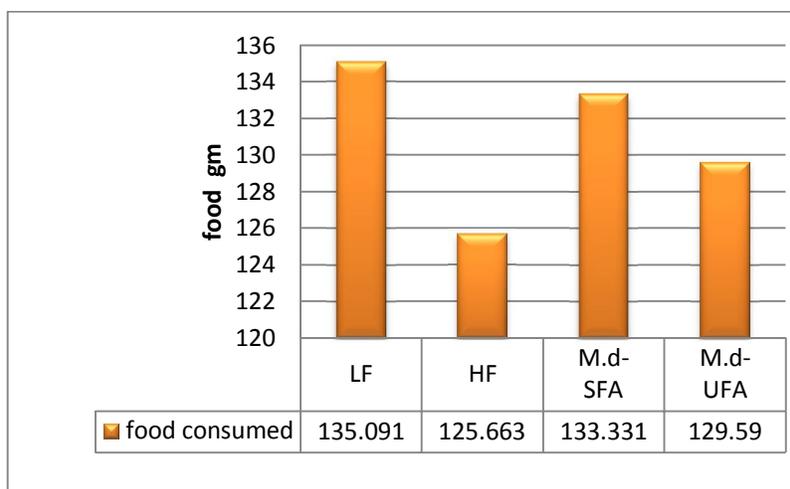


Figure 1: Food consumed during pregnancy period in dams of the tested groups, LF(low fat 10%), HF(high fat 45%), M.d-SFA(22.5% saturated fat), M.d-UFA(22.5% unsaturated fat). Mean ($p < 0.05$).

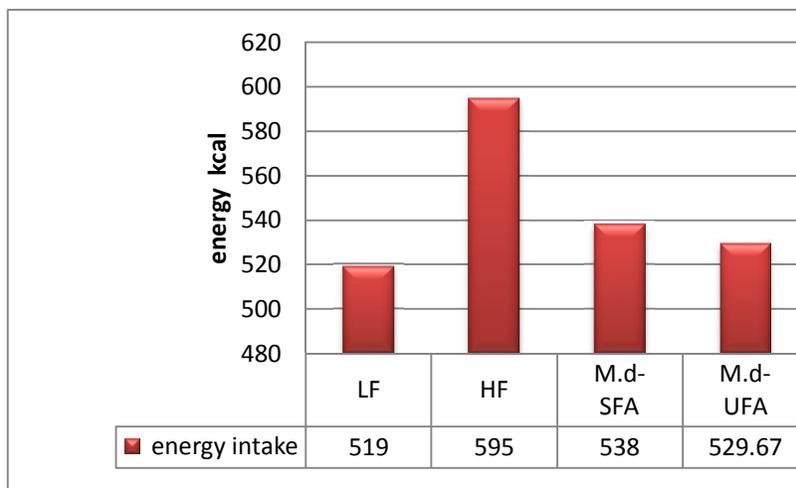


Figure 2: Energy intake during pregnancy period in dams of the tested groups, LF(low fat 10%), HF(high fat 45%), M.d-SFA(22.5% saturated fat), M.d-UFA(22.5% unsaturated fat). Mean (p<0.05).

Gestational body weight: HF dams during gestation period had significantly (p<0.05) higher body weight compared to all groups (348.33±9.652 gm) following with significantly by M.d-SFA dams (330.33±7.166 gm). The consumption of control or M.d-UFA diets resulted in low rate of body weight with significantly between the two groups (318.67±8.900 gm, 315.66±9.127gm) (**Figure 3**).

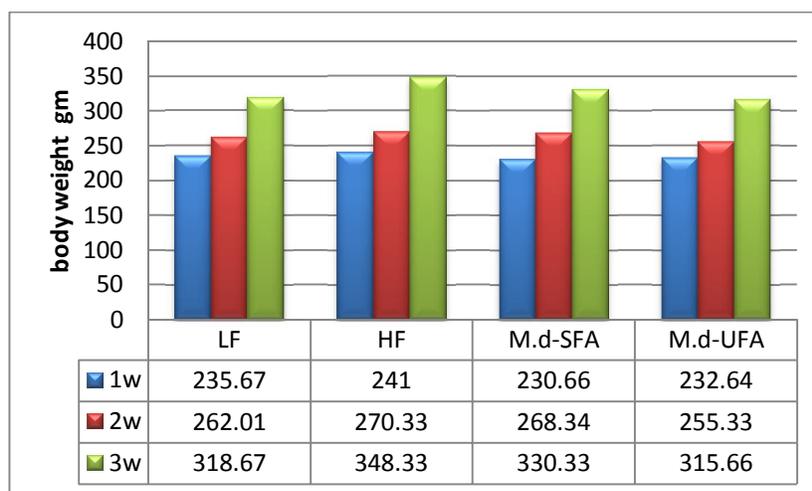


Figure 3: Body weight during pregnancy period in dams of the tested groups, LF(low fat 10%), HF(high fat 45%), M.d-SFA(22.5% saturated fat), M.d-UFA(22.5% unsaturated fat). Mean (p<0.05).

Gestational biochemical parameters: High fat diet consumption during pregnancy elevated levels of blood biochemical concentrations included glucose, T.ch and TG with significantly ($p<0.05$) from all groups, this effect also observed in dams on M.d-SFA diet. Fasting plasma glucose and lipoprotein concentrations were low in dams consumed unsaturated fat diet (M.d-UFA) with significant ($p<0.05$) than the control group except in TG level, also the HDL levels were significant ($p<0.05$) enhanced in this group while decreased in HF and M.d-SFA groups compared with control group (**table 2**).

Table 2: Biochemical parameters in day 18 of pregnancy in dams of the tested groups, LF(low fat 10%), HF(high fat 45%), M.d-SFA(22.5% saturated fat), M.d-UFA(22.5% unsaturated fat). Mean \pm S.D. ($p<0.05$).

Treatments	Gluco mmol/L	T.ch mmol/L	TG mmol/L	HDL mmol/L
LF	12.233 \pm 0.179d	1.129 \pm 0.015d	0.750 \pm 0.020c	1.134 \pm 0.019b
HF	16.196 \pm 0.153a	1.900 \pm 0.050a	1.520 \pm 0.024a	1.011 \pm 0.058d
M.d-SFA	14.482 \pm 0.425b	1.462 \pm 0.017b	1.203 \pm 0.063b	1.090 \pm 0.011c
M.d-UFA	13.233 \pm 0.100c	1.141 \pm 0.011c	0.745 \pm 0.017c	1.207 \pm 0.049a

Gestational hormonal parameters: Consumption of saturated fats in high (HF) or moderate percentage (M.d-SFA) during the gestation period increased significant ($p<0.05$) fasting plasma Leptin and Insulin concentrations this reflected to impair Insulin sensitivity by elevated HOMI concentrations. In moderate unsaturated fat diet (M.d-UFA), the dams had normal levels of hormones and HOMI with slightly differences compared control group (**table 3**).

Table 3: Hormonal parameters in day 18 of pregnancy in dams of the tested groups, LF(low fat 10%), HF(high fat 45%), M.d-SFA(22.5% saturated fat), M.d-UFA(22.5% unsaturated fat). Mean \pm S.D. ($p<0.05$).

Treatments	Leptin ng/ml	Insulin ng/ml	HOMI
LF	2.701 \pm 0.001d	1.391 \pm 0.006c	16.467 \pm 0.110d
HF	4.630 \pm 0.005a	3.439 \pm 0.005a	53.901 \pm 0.106a
M.d-SFA	4.151 \pm 0.008b	3.126 \pm 0.005b	43.810 \pm 0.116b
M.d-UFA	2.759 \pm 0.009c	1.379 \pm 0.004d	17.659 \pm 0.122c

Maternal weight gain: The maternal consumption of dietary fats types during pregnancy showed differences in weight gain after birth, it increased significant ($p<0.05$) in HF and M.d-SFA dams (57.871 \pm 5.422 gm, 47.923 \pm 6.110 gm) while the M.d-UFA dams had the less weight gain (36.855 \pm 5.299 gm) compared to all groups with significantly ($p<0.05$) (**Figure 4**).

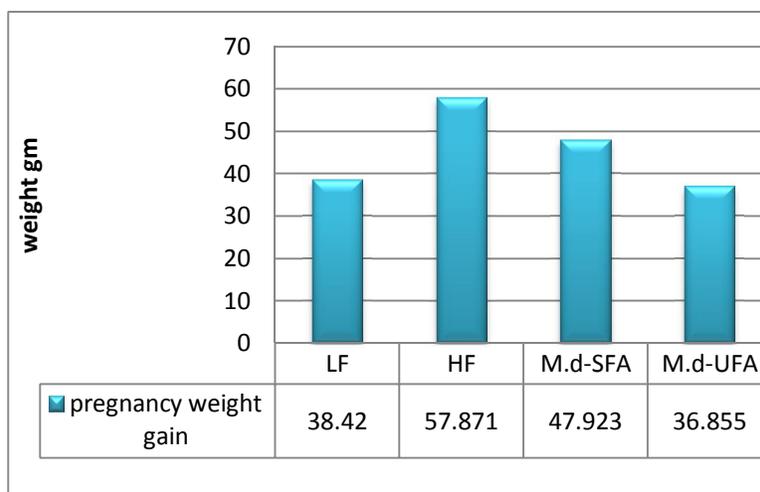


Figure 4: Pregnancy weight gain in dams of the tested groups, LF(low fat 10%), HF(high fat 45%), M.d-SFA(22.5% saturated fat), M.d-UFA(22.5% unsaturated fat). Mean ($p < 0.05$).

Postpartum body weight: Feeding dams postpartum on control diet (low fat diet) for 4 weeks showed differences in body weight in the tested groups. HF females had significant ($p < 0.05$) higher body weight (370.549 ± 6.330 gm) following by M.d-SFA females (348.861 ± 8.211 gm) with differences than the control group (328.781 ± 7.729 gm). The last body weight was recorded in M.d-UFA females comparing with all groups (319.397 ± 6.835 gm) (**Figure 5**).

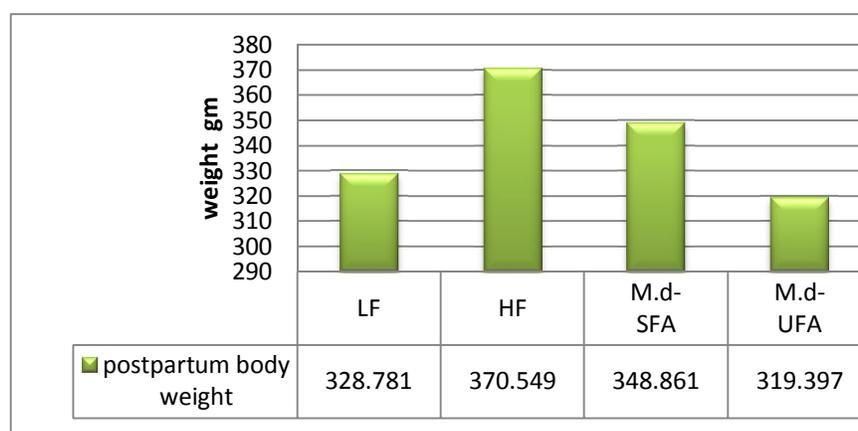


Figure 5: Postpartum body weight in dams of the tested groups after 4 weeks feeding on control diet, LF(low fat 10%), HF(high fat 45%), M.d-SFA(22.5% saturated fat), M.d-UFA(22.5% unsaturated fat). Mean ($p < 0.05$).

Postpartum biochemical parameters: Fasting plasma glucose, T.ch and TG were significantly ($p < 0.05$) still higher in HF female group after 4 weeks postpartum consuming control diet. Also this elevation was presented in M.d-SFA group with significance between the two groups while the HDL level was low in the two groups. The females of M.d-UFA group showed enhancing in postpartum biochemical concentration even when fed on low fat diet without significant than the control group except in T.ch and HDL levels with low concentrations (**table 4**).

Table 4: Postpartum biochemical parameters in dams of the tested groups after 4 weeks feeding on control diet, LF(low fat 10%), HF(high fat 45%), M.d-SFA(22.5% saturated fat), M.d-UFA(22.5% unsaturated fat). Means \pm S.D. ($p < 0.05$).

Treatments	Gluco mmol/L	T.ch mmol/L	TG mmol/L	HDL mmol/L
LF	13.046 \pm 0.541c	1.278 \pm 0.012c	0.911 \pm 0.014c	1.547 \pm 0.010a
HF	16.033 \pm 0.472a	2.276 \pm 0.022a	1.263 \pm 0.020a	1.269 \pm 0.029d
M.d-SFA	14.433 \pm 0.399b	1.790 \pm 0.024b	1.135 \pm 0.010b	1.364 \pm 0.015c
M.d-UFA	13.033 \pm 0.457c	1.270 \pm 0.031d	0.889 \pm 0.011c	1.528 \pm 0.019b

Postpartum hormonal parameters: The females of HF and M.d-SFA groups had significantly ($p < 0.05$) elevated levels of fasting plasma Leptin and Insulin after 4 weeks postpartum feeding on control diet 6.876 ng/ml, 4.832 ng/ml, 1.721 ng/ml, 1.689 ng/ml respectively, while the females of M.d-UFA group continue to show preferring Leptin and Insulin concentrations 3.184 ng/ml and 0.803 ng/ml with slight differences than the control group (**table 5**).

Table 5: Postpartum hormonal parameters in dams of the tested groups after 4 weeks feeding on control diet, LF(low fat 10%), HF(high fat 45%), M.d-SFA(22.5% saturated fat), M.d-UFA(22.5% unsaturated fat). Means \pm S.D. ($P < 0.05$).

Treatments	Leptin ng/ml	Insulin ng/ml	HOMI
LF	2.785 \pm 0.007d	0.719 \pm 0.007b	9.077 \pm 0.120 b
HF	6.876 \pm 0.020a	1.721 \pm 0.005a	26.702 \pm 0.200a
M.d-SFA	4.832 \pm 0.010b	1.689 \pm 0.009a	23.590 \pm 0.219a
M.d-UFA	3.184 \pm 0.009c	0.803 \pm 0.011b	10.127 \pm 0.311b

DISCUSSION

The obesity during pregnancy associated with change in metabolic response resulted in many health problems. The maternal physiology and metabolism were changed according to the nutritional needs for

mother and fetus, this metabolic system differ in obese/overweight mother from their normal counterpart during pregnancy^{19,20}.

Our results showed that during the pregnancy period, the calorie intake by HF dams was more than the control group this related that HF diet more calorie dense compared to LF diet (4.73 kcal/gm, 3.85 kcal/gm) this explanation was agree with other²¹, also this effect was shown in rats consumed moderate saturated fat (M.d-SFA) with highly calorie consumption during pregnancy. The moderate unsaturated fat diet (M.d-UFA) reduced the maternal energy intake, this result was agreed with other²² that lowering in food intake throughout pregnancy in UFA diet (18%) compared to control group.

The increasing in maternal weight through gestation in HF and M.d-SFA dams resulted from higher body fat deposition as the saturated fat less oxidize tendency¹³, this indicated from increasing in body weight gain after pregnancy, our study result accordance with others^{23,24}. The unsaturated fat in M.d-UFA diet change the pattern of gestational body weight, that clearly from the reduction in pregnancy weight and weight gain after birth, the reason may be related to unsaturated fatty acids oxidation viability^{25,26} that manage weight²⁷ and increased energy expenditure²⁶.

The maternal hyperlipidemia and lipoprotein concentrations in HF and M.d-SFA dams as resulted from high level of liver triglycerides synthesis²⁸ that have low oxidative rate²⁹ which released VLDL³⁰ suppressed LDL receptors in hepatocyte and increased plasma LDL³¹.

The unsaturated fat diet enhance plasma lipoprotein concentrations in M.d-UFA dams as resulted from antioxidant activity³² and diminish oxidative stress³³.

The hyperLeptinemia and hyperinsulinemia in HF and M.d-SFA dams in late pregnancy period may related to resistance to their action in hypothalamus²¹ and abnormal transport and signal transduction³⁴ and referred to hyperglycemia and reduced insulin resistance related to impair glucose transport (GLUT1, 4) in the cell³⁵.

The unsaturated fat diet was effective to improve Leptin sensitivity as resulted to decreased triglycerides level which enhance Leptin transport that in turn increased Leptin action³⁶ also enhance insulin level by modulating or changing glucose receptors or insulin binding proteins³⁷.

The results of this study demonstrated that differences in postpartum body weight reflected the metabolic changes in dams during gestation period in spite of postpartum exposure to control diet, this effect specially present in HF and M.d-SFA dams while the M.d-UFA dams showed favorable body weight and continuous on their weight management, this mean the type of dietary fatty acid is important factor.

The maternal obesity resulted from genetic and environmental factors interaction, the diet belong to the later that led to physiological change in adipose tissue³⁸. The normal maternal metabolic pregnancy process included fat deposition in visceral compartment¹⁴ but the dietary fat type and composition showed metabolic effectiveness in this study by biochemical and hormonal variations among the dietary groups. The postpartum hyper-lipidemia, Leptinemia and Insulinemia in HF and M.d-SFA dams may be related to differences in maternal metabolic adaptation resulted from feeding saturated fat diet during pregnancy while the UFA diet enhance postpartum plasma lipid and hormonal levels that alter or activate metabolic process of adipose tissue. The concentration of Leptin is correlated to body fatness before, during and after pregnancy^{39, 40, 41} which influence gestational weight gain and postpartum body weight by altering sensitivity to Leptin action⁴². The maternal feeding with dietary fat diet may affect the adipose distribution

in the body actively in visceral compartment in addition to fat deposited which in turn influence metabolic process, this effect was observed by other¹⁴.

CONCLUSION

This study elevated the effectiveness of nutrition fat type throughout pregnancy on development of maternal obesity, the results demonstrated that the dietary fat type and proportion is important factor by causing metabolic alternation reflected in body weight, Leptin level and Insulin sensitivity. Prepartum feeding with unsaturated fat diet (22.5% olive oil) showed enhancing effect by preventing maternal obesity, improve Leptin and Insulin action with favorable concentrations for glucose and plasma lipoproteins in spite of postpartum feeding on different diet.

ACKNOWLEDGEMENT

The author thank Biology Department- College of Science- University of Basrah to carry out the research work.

REFERENCES

1. S. Thangaratinam, K. Jolly, *BJOG*; 2010,117,1309.
2. F. Galtier-Dereure, C. Boegner and J. Bringer, *Am. J. Clin. Nutr.*; 2000, 71, 1242.
3. P.M. Catalano and H.M. Ehrenberg, *J. Bjog*; 2006, 113, 1126 .
4. Y. Yogev, P.M. Catalano, *Obstet Gynecol Clin N Am*; 2009, 36, 285.
5. M. Abdollahi, M. Cushman, F.R. Rosendaal, *Thromb Haemost*; 2003, 89, 493.
6. N. Sattar and I.A. Greer, *Br Med J*; 2002, 325, 15.
7. E. Leikin, J.H. Jenkins and W.L. Graves, *Obstet Gynecol*; 1987, 70, 587.
8. Y. Linne, B. Barkeling and S. Rossner, *BJOG*; 2002, 1127.
9. E. Oken, *Obstet Gynecol Clin N Am*; 2009, 377.
10. M. Diamant, H.J. Lamb, M.A. van de Ree, E.L. Endert, Y. Groeneveld, M.L. Bots, P.J. Kostense, J.K. Radder. *J Clin Endocrinol Metab*; 2005, 90, 1495.
11. R.V. Considine, M.K. Sinha, M.L. Heiman, A. Kriauciunas, T.W. Stephens, M.R. Nyce, J.P. Oshannesian, C.C. Marco, L.J. Mc-Kee, T.L. Bauer, and J.F. Caro, *N. Engl. J. Med.*; 1996, 334, 292.
12. L. H. Storlien, J.A. Higgins, T.C. Thomas, A.M. Brown, H.Q. Wang, X.F. Huang and P.L. Else, *Br. J. Nutr.*; 2000, 83, 85.
13. J.P. DeLany, M.M. Windhauser, C.M. Champagne, and G.A. Bray, *Am. J. Clin. Nutr.*; 2000, 72, 905.
14. J.K. Straughen, S. Trudeau and V.K. Misra, *Nutrition & Diabetes*; 2013, 1.
15. Research diet INC (2004). USA. WWW. Researchdiet.com.
16. M.L. Scott, M.S. Mischeil, and R.J. Young, (1982). *Nutrition of the chicken*, 3rd Ed. ITC. USA, New York .
17. D.R. Matthews, J.P. Hosker, A.S. Rudenski, B.A. Naylor, D.F. Treacher, and R.C. Turner, *Diabetologia*, 1985, 28, 412.
18. J.H. Lee, J.W. Bullen, V.L. Stoyneva, and C.S. Mantzoros, *Am. J. Physiol. Endocrinol. Metab.*, 2005, 288, 625.
19. A. Vahratian, V.K. Misra, S. Trudeau, D.P. Misra, *Obstet Gynecol.*, 2010, 116: 107–113.
20. A.M. Stuebe, T.F. McElrath, R. Thadhani, J.L. Ecker, S, *Matern Child Health J*, 2010, 14: 254–260.

21. A. Gupta, M.Srinivasan, S. Thamadilok and M.S. Pate, *Endocrinology*, 2009, 200: 293–300 .
22. M. Siemelink, A. Verhoef, J.A.M.A Dormans, P. N. Span and A. H. Piersma, *Diabetologia*, 2002, 45:1397–1403.
23. M.A. Shaw, M. Rasmussen and T.R. Myers, *Nutrition*, 1997,127: 64-69.
24. G.J. Howie, D.M. Sloboda, T. Kamal and M.H. Vickers, *J. Physiol.*, 2009, 587(4): 905–915.
25. Y. Shimomura, T. Tamura and M. Suzuki, *Nutrition*, 1990, 120: 1291-1296.
26. T. Matsuo, H. Takeuchi, H. Suzuki and M. Suzuki, *J. Clin. Nutr.*, 2002, 11(4): 302–308.
27. P. Flachs, O. Horakova, P. Brauner, M. Rossmeisl, P. Pecina, H.N. Franssen-Van, J. Ruzickova, J. Sponarova, Z. Drahota, C. Vlcek, J. Keijer, J. Houstek and J. Kopecky, *Iabetologia.*, 2005, 48: 2365-2375.
28. M.T. Chen, L.N. Kaufman, T. Spennetta and E. Shrago, *Metabolism*, 1992, 41: 564–569.
29. B. Mittendorfer and L.S. Sidossis, *Am. J. Clin. Nutr.*, 2001, 1(73):892–899.
30. A.C. Guyton and J.E. Hall, (1996). *Treaty of Medical Physiology (Tratado de fisiologia médica)*. Rio de Janeiro: Guanabara Koogan.
31. S.M. Grundy, *Am. J. Clin. Nutr.*, 1987, 45:1168-1175.
32. R. Krzeminski, S. Gorinstein, H. Leontowicz, M. Leontowicz, M. Gralak, J. Czerwinski, A. Lojek, M. Cyiazy, O. Martin-Belloso, N. Gligelmo-Miguel and S. Trakhtenberg, *J. Agric. Food Chem.*, 2003, 51:5774-5779.
33. T. Psaltopoulou, A. Naska, P. Orfanos, D. Trichopoulos, T. Mountokalakis and A. Trichopoulou, *Am. J. Clin. Nutr.*, 2004, 80: 1012-1018.
34. K. El-Haschimi, D.D. Pierroz, S.M. Hileman, C. Bjorbaek and J.S. Flier, *J. Clin. Inves.*, 2000, 105: 1827–1832.
35. Y. Takahashi and T. Ide, *Br. J. Nutr.*, 2000, 84: 175-184.
36. W.A. Banks, A.B. Coon, S.M. Robinson, A. Moinuddin, J.M. Shultz, R. Nakaoko and J.E. Morley, *Diabetes*, 2004, 53:1253–1260.
37. A.H. Lichtenstein and U.S. Schwab, *Atherosclerosis*, 2000, 150, 227–243.
38. M. Bluher, *Exp Clin Endocrinol Diabetes*, 2009, 117: 241–250.
39. N.F. Butte, J.M. Hopkinson and M.A. Nicolson, *J Clin Endocrinol Metab*, 1997, 82, 585–589.
40. T.J. Highman, J.E Friedman, L.P. Huston, W.W. Wong, P.M. Catalano. *Am J Obstet Gynecol* , 1998, 178, 1010–1015.
41. B. Eriksson, M. Lofl, H. Olausson and E. Forsum, *British Journal of Nutrition*, 2010, 103, 50–57.
42. T. P. Stein, T. O. Scholl, M. D. Schluter and C. M. Schroeder, *Am J Clin Nutr.*, 1998, 68:1236–40.

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