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Research Article

**ANALYSIS OF METABOLIC ADAPTATIONS DURING
PREGNANCY AMONG FEMALES OF PAKISTAN**¹Dr. Abdul Wahid Ayub, ²Dr. Mahrukh Tabassam, ³Dr. Afaf Arshad¹Medical Officer at DHQ Hospital, Bhakkar²Women Medical Officer at DHQ Hospital, Bhakkar³Women Medical Officer at RHC Bagga Sheikhan, Rawalpindi**Abstract:**

Introduction: Human pregnancy is characterized by alterations in maternal lipid metabolism, which could be divided into 2 phases: an anabolic phase and a catabolic phase. The anabolic phase occurs in the first 2 trimesters of human gestation and is attributed to several factors that cooperatively increase the deposition of lipids in maternal tissues. **Aims and objectives:** The basic aim of the study is to assess the metabolic changes during pregnancy among local female population of Pakistan. **Methodology of the study:** This study was conducted at DHQ Hospital, Bhakkar during January 2018 to May 2018. We collected the data from 100 pregnant females who visited the hospital regularly. In this cross sectional study we design a questionnaire regarding metabolic changes during pregnancy. We also conduct some interviews to find the daily intake of nutrients and daily changes of metabolic level among pregnant women. **Results:** The level of glycated hemoglobin was significantly higher in the overweight and obese groups than in normal weight and underweight groups ($P < 0.05$). In addition, birth weight was significantly higher in overweight or obese women than in underweight women ($P < 0.05$). Human placental lactogen increases progressively throughout pregnancy. **Conclusion:** It is concluded that the rapid rate of fetal growth during the last half of gestation dictates changes in basal metabolism, protein, and mineral accretions. About 60% of the increase in basal metabolic rate (BMR) occurs during the last half of gestation, when the metabolic cost of fetal tissue synthesis is the greatest.

Corresponding author:

Dr. Abdul Wahid Ayub,
Medical Officer at DHQ Hospital,
Bhakkar

QR code



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INTRODUCTION:

Human pregnancy is characterized by alterations in maternal lipid metabolism, which could be divided into 2 phases: an anabolic phase and a catabolic phase. The anabolic phase occurs in the first 2 trimesters of human gestation and is attributed to several factors that cooperatively increase the deposition of lipids in maternal tissues. The first factor is maternal hyperphagia, which progressively increases throughout gestation, thereby boosting the availability of exogenous metabolic substrates [1]. Enhanced de novo lipogenesis is another factor contributing to early pregnancy anabolism.

Pregnancy is a dynamic, anabolic state. Within several weeks of conception, a new endocrine organ, the placenta, is already formed and is secreting hormones that affect the metabolism of all nutrients. These adjustments in nutrient metabolism, in addition to changes in the anatomy and physiology of the mother, support fetal growth and development while maintaining maternal homeostasis and preparing for lactation [2]. The first half of pregnancy is primarily a time of preparation for the demands of rapid fetal growth that occur later in pregnancy [3]. The corpus luteum and the placenta secrete hormones that maintain pregnancy and influence metabolism. Human chorionic gonadotropin is detected in the serum and urine within a few days of implantation. Consequently, the decreased adipose tissue lipolytic activity together with the augmented capacity of maternal tissues to employ both glucose and intracellular glycerol for the production of glycerol-3-phosphate result in net triglyceride accumulation [4].

Aims and objectives

The basic aim of the study is to assess the metabolic changes during pregnancy among local female population of Pakistan.

METHODOLOGY OF THE STUDY:

This study was conducted at DHQ Hospital, Bhakkar during January 2018 to May 2018. We collected the data from 100 pregnant females who visited the hospital regularly. In this cross sectional study we design a questionnaire regarding metabolic changes during pregnancy. We also conduct some interviews to find the daily intake of nutrients and daily changes of metabolic level among pregnant women. We also collected the blood samples for finding serum lipid profile and antioxidants ratio among pregnant females.

Statistical analysis

The data of respiratory function were compared between the smoker and non-smoker groups using the independent t-test for normally distributed data or the Mann-Whitney U test for other distributions. Differences were considered statistically significant at $p < 0.05$.

RESULTS:

The level of glycated hemoglobin was significantly higher in the overweight and obese groups than in normal weight and underweight groups ($P < 0.05$). In addition, birth weight was significantly higher in overweight or obese women than in underweight women ($P < 0.05$). Human placental lactogen increases progressively throughout pregnancy. The precise function of human placental lactogen is not clear; however, because it is biologically similar to growth hormone, it may represent some type of growth factor for the fetus and the placenta. There were no significant differences between the four pre-pregnancy BMI categories in maternal age, parity, height and gestational weeks (Table 1).

Table 01: Effects of pre-pregnancy body mass index on pregnancy outcomes

	Time of Gestation			
	10 wk	20 wk	30 wk	40 wk
Serum placental hormones				
Human chorionic gonadotropin ($\times 10^4$ U/L)	1.3	4.0	3.0	2.5
Human placental lactogen (nmol/L)	23.148	138.888	254.628	393.516
Estradiol (pmol/L)	5.507	22.026	55.065	66.078
Progesterone (nmol/L)	79.5	159.0	318.0	413.4
Nutrient metabolism				
Basal metabolism, net change (MJ/d)	0.19	0.41	0.62	0.95
Serum albumin (g/L)	32	29	28	28
Serum triacylglycerol (%) ²	120	150	210	280
Serum alpha tocopherol (%) ²	110	120	135	150
Serum vitamin A (%) ²	75	75	75	75
Serum vitamin C (%) ²	85	75	68	62

Serum folic acid (%) ²	78	68	60	58
Products of conception				
Fetus (g)	5	300	1500	3400
Placenta (g)	20	170	430	650
Amniotic fluid (g)	30	250	750	800
Maternal tissue gain				
Uterus (g)	140	320	600	970
Mammary gland (g)	45	180	360	405
Plasma Volume (mL) ³	50	800	1200	1500
Total nutrient accretion in mother and fetus				
Protein (g)	36	165	498	925
Fat (g)	328	2064	3594	3825
Calcium (g)	—	—	—	30
Iron (mg)	—	—	—	565
Zinc (mg)	—	—	—	100

DISCUSSION:

Biosynthesis of the estrogens (ie, estrone, estradiol, and estriol) is a complicated process involving the mother, fetus, and placenta. In addition to influencing the uterus and other reproductive organs, estrogens cause a rise in certain binding hormones, which result in the elevation of total hormone concentrations, whereas the amounts of unbound and biologically active hormones remain unchanged [5]. Estrogens also influence carbohydrate, lipid, and bone metabolism. Progesterone concentrations rise progressively throughout pregnancy, of which the initial source is the corpus luteum, but placental sources of progesterone predominate later during pregnancy. Progesterone relaxes smooth muscle, which causes atony of the gastrointestinal and urinary tracts [6].

Although fetal demand for nutrients occurs primarily during the last half of gestation when >90% of the fetal growth occurs, adjustments in nutrient metabolism are apparent within the first weeks of pregnancy (1). After the first 10 wk of pregnancy, serum triacylglycerol concentrations in pregnant women are 20% higher than those of nonpregnant women; they reach a value ≈ 3 times that of nonpregnant women by term. Other serum lipids (ie, phospholipids, cholesterol, glycerol, and fatty acids) also increase during pregnancy, but the net change is less than that of triacylglycerol [7]. Circulating concentrations of most nutrients decrease by the end of the first 10 wk of gestation and remain lower than nonpregnant values until term. This decrease in circulating nutrients occurs before there is an increase in plasma volume. Serum albumin decreases ≈ 8 –10% in the first 10 wk of pregnancy. Because albumin is a carrier protein for many nutrients, the marked decline in albumin early in pregnancy may explain the sudden decrease in circulating nutrient concentrations

[8]. The net increase in plasma volume rises from 50 mL at 10 wk gestation to 800 mL at 20 wk gestation [9]. Although the concentration of nutrients in the circulation declines during the same period of time, the reduction is less than the 40-fold change in plasma volume. Thus, the total amount of vitamins and minerals in circulation increases during pregnancy [10].

CONCLUSION:

It is concluded that the rapid rate of fetal growth during the last half of gestation dictates changes in basal metabolism, protein, and mineral accretions. About 60% of the increase in basal metabolic rate (BMR) occurs during the last half of gestation, when the metabolic cost of fetal tissue synthesis is the greatest. The metabolic physiology during pregnancy is the normal adaptations in which maternal body changes its physiological and homeostatic mechanisms to provide and ensure the basic foetal physiological needs for its survival and adequate growth.

REFERENCES:

1. McCoy MG, Sun GS, Marchadier D, Maugeais C, Glick JM, Rader DJ: Characterization of the lipolytic activity of endothelial lipase. *J Lipid Res* 2002;43:921-929.
2. Stanley K, Fraser R, Bruce C. Physiological changes in insulin resistance in human pregnancy: longitudinal study with the hyperinsulinaemic euglycaemic clamp technique. *BJOG*. 1998;105:756–9.
3. Inouye M, Kettunen J, Soininen P, Silander K, Ripatti S, Kumpula LS, et al. Metabonomic, transcriptomic, and genomic variation of a population cohort. *Mol Syst Biol*. 2010;6:441.
4. Gauster M, Hiden U, Blaschitz A, Frank S, Lang

- U, Alvino G, Cetin I, Desoye G, Wadsack C: Dysregulation of placental endothelial lipase and lipoprotein lipase in intrauterine growth-restricted pregnancies. *J Clin Endocrinol Metab* 2007;92:2256-2263.
5. Kazantzis M, Stahl A: Fatty acid transport proteins, implications in physiology and disease. *Biochim Biophys Acta* 2012;1821:852-857.
 6. Weaver DD, Solomon BD, Akin-Samson K, Kelley RI, Muenke M: Cyclopia (synophthalmia) in Smith-Lemli-Opitz syndrome: first reported case and consideration of mechanism. *Am J Med Genet C Semin Med Genet* 2010;154C:142-145.
 7. Kc K, Shakya S, Zhang H. Gestational diabetes mellitus and macrosomia: a literature review. *Ann Nutr Metab* 2015; 66:14-20.
 8. Abell SK, De Courten B, Boyle JA, Teede HJ. Inflammatory and other biomarkers: role in pathophysiology and prediction of gestational diabetes mellitus. *Int J Mol Sci* 2015; 16:13442-73
 9. Harreiter J, Dovyjak G, Kautzky-Willer A. Gestational diabetes mellitus and cardiovascular risk after pregnancy. *Womens Health (Lond Engl)* 2014; 10:91-108.
 10. Newbern D, Freemark M: Placental hormones and the control of maternal metabolism and fetal growth. *Curr Opin Endocrinol Diabetes Obes* 2011;18:409-416.