Arab. J. Lab. Med. (1996), Vol. 22 No. 2: 453 - 465

THE FAST OF RAMADAN, ITS EFFECTS ON ENDOCRINE CONTROL OF GLUCOSE HEOMEOSTASIS

SHAARAWY M., SHOBOKSHI A., and EL-NOURI A.
Department of Obstetrics & Gynecology, Faculty of Medicine, Cairo University,
Department of Obstetrics and Gynecology, Faculty of Medicine and Allied Sciences,
King Abdul-Aziz University, Jeddah, Saudi Arabia

Although pregnant women are excused fasting during Ramadan, many pregnant women choose to fast with their families during Ramadan rather than make up the time later. The fast of Ramadan during pregnancy was reported to induce impaired glucose heomeostasis. Accordingly this study was initiated to evaluate the effects of fast of Ramadan on the endocrine control of glucose heomeostatis in pregnant women.

Blood samples were obtained from 25 normal pregnant women after 12 hr overnight fast, in addition to postprandial blood samples taken 2 hr after breakfast (controls). Meanwhile blood samples were obtained from 30 normal pregnant women who were fasting Ramadan at the end of the fasting day immediately before the intake of food at sunset (Iftar) and 2 hrs after Iftar. Both groups comprised women at their first, second and third trimesters of pregnancy. Blood samples were assayed for glucose by glucose oxidase method and insulin, glucagon, cortisol, growth hormone (HGH), somatostatin and human placental lactogen (hPL) by the corresponding radioimmunoassay.

Endocrine consequence of fasting Ramadan during pregnancy revealed significant fall in the concentration of both blood glucose and insulin with a concomitant rise of hPL throughout the first trimester, without any significant changes of other hormones and all parameters in the second and third trimesters. Moreover there were no significant changes of postprandial levels of glucose, insulin, glucagon, cortisol, HGH, hPL, somatostatin in Ramadan fasting women throughout pregnancy.

Hormonal changes associated with the decline of maternal blood glucose engendered by fasting Ramadan in the first trimester may preserve fetal metabolic heomeostasis. However, the possible effect of maternal fasting on fetal carbohydrate requirements needed for normal fetal growth could not be overlooked. Postabsorptive measures proved similar glucose output in both Ramadan fasting women and fasting pregnant women (controls) indicating that substrate and hormonal changes in the fasting state were transient and were reestablished 2 hr after Iftar meal.

Health adult Muslims are required to abstain from food and drink from dawn to sunset daily during the month of Ramadan. Dispensation from fasting is allowed during sickness, menstruation, pregnancy, breast feeding and travel. In people who are well normal heomeostatic mechanisms seem to cope: urinary volume, electrolytes, pH and nitrogen excretion remain within physiological limits (Cheath et al., 1990). Some studies have reported substantial weight loss and increase uric acid consistent with catabolism of body mass (El Hazmi et al., 1987), but these findings have not been confirmed (Sliman and Katib, 1988). Some of the variations may be attributable to local traditions and food quality. A trial of high carbohydrate intake (consumed after sunset) during the first fortnight of Ramadan was associated with a fall in blood urea concentration, a change to a high fat diet over the next fortnight was associated with a fall in glucose concentration, which the authors believed was due to impaired glucose heomeostasis (Mohammad et al., 1989). Those who consume high energy foods after sunset, unsurprisingly, gain weight (Begma and Khan, 1990).

The metabolic consequences of fasting during pregnancy revealed significant fall in concentrations of glucose, insulin, lactate and creatinine and a rise in concentrations of triglycerides and hydroxybutyrate at the end of the fasting day (Malhotra et al., 1989). This pattern of accelerated starvation was also reported by Prentice et al., (1983) only among women who fasted in late pregnancy. A study of birth weights of more than 1300 babies showed no effect of maternal fasting at any stage of pregnancy (Cross et al., 1990). In fact little is known with any certainty about the clinical problems during the fast of Ramadan during pregnancy. Although pregnant women are excused fasting during Ramadan, many pregnant women chose to fast with their families during Ramadan rather than make up the time later. There was no significant difference in either the patient's age or the duration of pregnancy between those who were and

were not fasting (Reeves 1992). The issue of whether a women is eating enough may arise only if she fails to gain weight (Reeves, 1992). As many pregnant women choose fasting during Ramadan, the topic deserves more thorough scientific attention. This study was undertaken to investigate the effects of the fast of Ramadan on the circulatory levels of insulin, glucagon, cortisol, human growth hormone (HGH), somatostatin and human placental lactogen (hPL) which represent the main endocrine control of glucose heomeostasis, at the end of the fasting day in pregnant women in their first, second and third trimester of pregnancy.

Patients and methods:

Fifty five pregnant women participated in this study. They comprised 25 normal pregnant women, of them 4,9 and 12 were in their first, second and third trimester of pregnancy respectively (controls). In addition, another group of 30 women of matched age and parity and of the same socioeconomic class represent those who were fasting Ramadan, being 4, 10 and 16 in their first, second and third trimester of pregnancy respectively. Both groups were healthy, free from any systemic disease and not receiving any drugs which may invalidate hormonal assay. Clinical obstetric examination, history taking and ultrasonographic findings, proved normal gestation. Blood samples were obtained from control pregnant women after 12 hours overnight fast in addition to postprandial blood samples taken 2 hours after breakfast. Blood samples from pregnant women who were fasting Ramadan were taken at the end of the fasting day immediately before the intake of food at sunset (Iftar meal). Postprandial samples were also taken 2 hours after Iftar meal. Blood samples were assayed for glucose, insulin, glucagon, HGH, cortisol, hPL and somatostatin. Blood glucose was determined by glucose oxidase method (Boehringer, Mannheim, Germany). Serum insulin was measured by second antibody radioimmunoassay using I^{125} as a tracer. Reagent kit was purchased from Sorin Biomedica, Italy. The intra and interassay coefficient of variation (CV) were 5.5 and 6.2% respectively). Blood for glucagon assay was collected by venipuncture into iced EDTA tubes, where 5000 kallikrein inactivating units per 10 ml of whole blood was added. Plasma was separated in a refrigerated centrifuge. Plasma glucagon was assayed by double antibody I^{125} radioimmunoassay. The intra and interassay CV were 4.4% and 5.7% respectively. Reagent was parachased from DPC, CA, USA. Serum cortisol was determined by a solid phase 1^{125} radioimmunoassay using reagent kit which was purchased from Sorin Biomedica, Italy. The intra and interassay CV were 2.9% and 5.5% respectively. Serum HGH was determined by a solid phase I^{125} radioimmunoassay using reagent kit of Sorin, Biomedica, Italy. The in-

Table (1): Fasting maternal blood glucose, insulin, glucagon, cortisol, growth hormone, somatostatin and hPL in Ramadan fasting pregnant women (RFW) and controls.

	First trimester		Second trimester		Third trimester	
	Controls	RFW	Controls	RFW	Controls	RFW
No of cases	4	4	9	10	12	16
Blood glucose (mg/dl)						
mean ± SE	75 ± 3.55	64± 3.25	65±2.03	72.5±5.56	67±4	70± 2.575
t/df, P value	2.2855/6, <0.025		1.1826/17,>0.05		0.9201/26, > 0.05	
Serum Insulin (uU/ml)						
mean ± SE	6.5± 0.07	3.55±0.4	5.1 ± 0.33	6±0.89	7.2± 0.46	7.1±0.665
t/df, P value	7.3891/6, < 0.0005		0.9482/17, >0.05		0.1237/26, > 0.05	
Plasma Glucagon (pg/ml)						
mean ± SE	113.5±17.15	73.5±17	124±5.23	111.4±6.21	125.4±4.24	117.5±3.475
t/df, P value	1.6564/6, >0.05		1.5519/17, >0.05		1.4410/26, > 0.05	
Serum cortisol (ng/ml)						
mean ± SE	118±12	109±18	148±1457	115±14.4	151±7.97	162±7.17
t/df, P value	0.4160/6, > 0.05		1.6109/17, >0.05		1.0261/26, > 0.05	
Serum HGH(ng/ml)						
mean ± SE	3.94 ± 1.46	1.78 ± 0.43	1.64±0.22	1.4±0.186	1.67±0.13	2.10±0.24
t/df, P value	1.4192/6, >0.05		0.833/17, >0.05		1.5756/26, >0.05	
Plasma Somatostatin (pg/ml)						
mean ± SE	67.5±6.5	52±9	63±3.53	60.9±4.17	64±2.54	57.8±6.7
t/df, P value	1.3961, >0.05		0.3843/17, >0.05		0.8653/26, > 0.05	
Serum hPL(ug/L)						
mean ± SE	1.2 ± 0.18	1.6± 0.12	3.2±0.42	3.3±0.25	8.42±0.85	7.36±0.76
t/df, P value	0.8493/6, >0.05		0.2046/17, > 0.05		0.9297/26, >0.05	

tra and interassay CV were 1.9% and 2.3% respectively. Plasma for somatostatin assay was collected as for glucagon assay and somatostatin concentration was determined by double antibody radioimmunoassay. Reagent kit was purchased from Incstar corporation, Minnesota, USA. The intra and interassay CV were 4% and 6.5% respectively. Serum hPL was determined by radioimmunoassay using reagent kits purchased from DPC, CA, USA and the intra and interassay CV were 3.5% and 4.2% respectively.

Results obtained were statistically analyzed using student t test.

Results:

During the first trimester of pregnancy fasting blood glucose level was significantly lower in Ramadan fasting women than that of control pregnant women. The mean percent decrease amounted to 15% of the mean control value. There were no significant changes of circulating levels of glucose throughout the second and third trimester of pregnancy between both groups. Throughout the first trimester of pregnancy of Ramadan fasting women, there was a significant decrease of blood insulin concentration, when compared to the corresponding concentration of controls. The mean percent decrease of serum insulin was 45%. this was associated with a concomitant increase in the serum human placental lactogen (Table 1) with a mean percent rise of 33%. Throughout the second and third trimesters of pregnancy, there was no significant changes of blood levels of insulin, glucagon, cortisol, HGH, somatostatin and hPL in Ramadan fasing women and controls (Table 1). All postprandial blood levels of glucose, insulin, glucagon, cortisol, HGH, hPL and somatostatin in Ramadan fasting women did not differ significantly from the corresponding levels of controls (Table 2).

Discussion:

The normal progress of pregnancy is directed by the endocrine milieu, which has been well described. Very large increments in placenta-derived steroid and protein hormones occur throughout pregnancy (Speroff et al., 1984). Alternations in these hormonal patterns would presumably be undesirable. The metabolic demands of pregnancy are also considerable because the energy for fetal development is derived primarily from glucose, and the increasing insulin resistance during pregnancy (Leturque et al., 1984 and Leturque et al., 1986), increases maternal fat utilization and impedes maternal glucose utilization (Fioretti et al., 1970) to provide glucose for the fetus. In the present study blood glucose level in Ramadan fasting women was significantly decreased during the first trimester of pregnancy when compared to the corresponding level of controls. The average percent decrease was 15%. The

Table (2): Postprandial blood glucose, insulin, glucagon, cortisol, growth hormone, somatostatin and hPL in Ramadan fasting pregnant women (RFW) and controls.

	First trimester		Second trimester		Third trimester	
	Controls	RFW	Controls	RFW	Controls	RFW
No of cases	4	4	9	10	12	16
Blood glucose (mg/dl)						
mean ± SE	97±3.55	87±5.35	91.5±2.87	103±6.32	92.5±5.9	94±4.5
t/df, P value	1.5575/6, > 0.05		1.6568/17, >0.05		0.2021/26,>0.05	
Serum Insulin (uU/ml)						
mean ± SE	84±2.67	79±1.55	37.5±5.9	48.8±5.57	58.4±5.7	65.2±9.75
t/df, P value	1.6195/6, >0.05		1.4173/17, > 0.05		0.6021/26, >0.05	
Plasma Glucagon (pg/ml)						
mean ± SE	113.5±13.4	87±8.5	99.5±3	93±6.51	123±7.97	115.5 ± 6.375
t/df, P value	1.6699/6, >0.05		0.9068/17, >0.05		0.7445/26, >0.05	
Serum cortisol (ng/ml)						
mean ± SE	112±17.25	105±12.5	127±14.27	100±9.04	162.5±9.12	144±7.05
t/df, P value	0.3286/6, > 0.05		1.5983/17, > 0.05		1.6049/26, >0.05	
Serum HGH(ng/ml)						
mean ± SE	1.175±0.02	1.05±0.22	1.134±0.107	0.99±0.085	1.5±0.12	1.8±0.17
t/df, P value	0.5658/6, >0.05		1.0175/17, >0.05		1.4416/26, >0.05	
Plasma Somatostatin (pg/ml)						
mean ± SE	81.0±6	65±8.9	65±4.15	56.1±3.7	50±2.89	62±3.8
t/df, P value	1.4906/6, >0.05		1.6007/17, >0.05		1.6757/26, >0.05	
Serum hPL(ug/L)						
mean ± SE	1.35±0.32	1.1±0.18	3.8±0.5	3.6±0.62	8.83±0.78	7.64±0.52
t/df, P value	0.6810/6, >0.05		0.2511/17, >0.05		1.2844/26, >0.05	

decrease of blood glucose in Ramadan fasting pregnant women was associated with a significant decrease of serum insulin and a significant increase of human placental lactogen. Schreiner et al., (1981), reported that significant maternal and fetal hypoglycemia and hypoinsulinemia developed with 24 hr of fasting of the pregnant ewe and these changes were associated with a 50% decrease of fetal glucose supply via the umbilical circulation. Since glucose is considered the major metabolic fuel utilized by the developing fetus (Battaglia and Mechia, 1978), the decrease of insulin and the increase of hPL secretion seem to be a regulatory mechanism for metabolic adjustments that aim at increasing the supply of glucose to the fetus and to maintain maternal blood glucose levels far from a sharp decline during the fasting condition. Koski and Fergusson (1991) reported that all amniotic fluid constituents were significant affected by the level of carbohydrate in the maternal diet. As the amount of either glucose or fructose increased in the maternal diet, there was a significant rise in amniotic fluid glucose concentration. Lowered amniotic fluid glucose has been associated with intrauterine growth retardation and has been produced during prolonged fasting and starvation (Drazancic and Kuvacic, 1974 and Kim and Felig, 1972). Johnson and Greenberg (1992), reported that placental lactogen regulates the uptake and utilization of carbohydrates, lipids and proteins in maternal and fetal tissues, suggesting that the hormone plays a role in the control of nutrient metabolism during pregnancy (Fremark and Handwerger, 1989). Nutritional factors, in turn appear to regulate the production and secretion of hPL and to modulate the cellular response to hPL in the mother and fetus. Fasting of pregnant ewes for 72 hr in late gestation stimulate a 50% increase in the concentration of ovine placental lactogen in fetal plasma (Brinsmead et al., 1981, and Fremark et al., 1989) and causes a 50 - 70% reduction in the specific binding of ovine placental lactogen to maternal and fetal liver membranes (Fremark et al., 1989 and Fremark et al., 1990). These effects of fasting are reversed entirely by refeeding the ewes a standard diet of oats and hay. During fasting, maternal plasma insulin and IGF-1 concentrations fall consequent to maternal hypoglycemia, protein restriction (Maes et al., 1991). The reduction in maternal insulin and IGF-1 concentrations limits maternal uptake and utilization of glucose and amino acids, facilitating their transport to the fetus. Fetal plasma glucose concentrations decline in proportion to maternal glucose levels (Hay, 1979 and Leturque et al., 1987), producing fetal hypoinsulinemia, hyposomatomedinemia (Fremark et al., 1989 and Davenport et al., 1990). The reduction in fetal insulin and IGF-1 levels and the decline in fetal placental lactogen binding likely reduce the uptake of glucose and amino acids in fetal

lowering blood glucose in the first trimester of pregnancy. The present investigation showed that there were no significant changes of circulatory levels of glucagon, cortisol, HGH and somatostatin in Ramadan fasting women, when compared the corresponding levels of controls. These results indicate that hormonal changes controlling glucose homeostasis were confined to maternal serum insulin and human placental lactogen.

In the present investigation the postprandial levels of blood glucose, insulin, glucagon, cortisol, HGH, hPL and somatostatin in Ramadan fasting women throughout the first, second or third trimester of pregnancy were not significantly different from the corresponding postprandial levels of controls. These results indicate that the substrate and hormonal changes which occurred in the fasting state were, however, quite transient and in most instances fasting concentrations of blood glucose, insulin and hPL were reestablished after 2 hrs from Iftar meal. Thus, in the postabsorptive state there was a similar glucose output in Ramadan fasting pregnant women and overnight fasting pregnant women (controls).

References:

Battaglia F.C., Mechia G. (1978): Principal substrates of fetal metabolism. Physiol Rev. 58: 499-527.

Begma I. A., Khan I. H. (1990): Variations in ionic concentration of blood serum during Ramadan fasting. Pakistan Journal of Science 33: 546-53.

Bonen A., Campagna P., Gilchrist L., Young D.C., Beresford P. (1992): Substrate and endocrine responses during exercise at selected stages of pregnancy. J. Appl. Physiol. 73 (1): 134-42.

Brinsmead M.W., Bancroft B.J., Thorburn G.D., Waters M.J. (1981): Fetal and maternal ovine placental lactogen during hyperglycemia, hypoglycemia and fasting. J. Endocrinol 90: 337-43.

Cheath S.H., Ch'ng S.L., Hussein R., Ducan N.T. (1990): Effects of fasting during Ramadan on urinary excretion in Malaysian Muslims. Br. J. Nutr 63: 329-37.

Cross J.H., Eminson J., Wharton B.A. (1990): Ramadan and birth weight at full term in Asian Moslem pregnant women in Birmingham. Arch. Dis. Child. 65: 1053-6.

Davenport M.L., D' Ercole A.J., Underwood L.E. (1990): Effect of maternal fasting on fetal growth, serum insulin-like growth factors (IGFs) and tissue IGFmRNAs. Endocrinology. 126: 2062-7.

Drazancic A., Kuvacic I. (1974): Amniotic fluid glucose concentrations. Am. J. Obstet Gyencol 120: 40-8.

El-Hazmi M.A.F., Alfaleh F.Z., Almofleh I.A. (1987): Effect of Ramadan fasting on the values of hematological and biochemical parameters. Saudi Medical Journal 8 (2): 171-6.

Fioretti P., Gonazzani A., Aubert M.M., Grangoli G., Pupillo A. (1970); Correlations between human chorionic somatomanamotrophin (lactogen), immunoreactive insulin, glucose and lipid fractions in plasma of pregnant women. Obstet Gynecol 77: 745-51.

Fremark M., Comer M., Mularoni T., D' Ercole A.J., Grandis A., Kokack L. (1989): Nutritional regulation of the placental lactogen receptor in fetal liver; implications for fetal metabolism and growth. Endocrinology 125: 1504-12.

Fremark M., Comer M., Mularoni T., D' Ercole A.J., Grandis A., Kokack L. (1990): Placental lactogen receptors in maternal sheep liver: Effect of fasting and refeeding. Am. J. Physiol 258: 338-46.

Fremark M., Handwerger S. (1985): Ovine placental lactogen inhibits glucagon-induced glycogenolysis in fetal rat hepatocytes. Endocrinology 116: 1275-80.

Fremark M., Handwerger S. (1989): The role of placental lactogen in the regular of fetal metabolism and growth. J. Pediatr Gastroenterol Nutr 8: 281-7.

Fremark M., Keen A., Fowlkes J., Mularoni T., Comer M., Grandis A., Kodack L. (1992): The placental lactogen receptor in maternal and fetal sheep liver: regulation by glucose and role in the pathogenesis of fasting during pregnancy. Endocrinology 130 (2): 1063-70.

Haugel S., Leturque A., Gilbert M., Kande J., Girard J. (1988): Glucose utilization by the placenta and fetal tissues in fed and fasted pregnant rabbits. Pediatr Res. 23: 480-3.

Hay Tr W.W. (1979): Fetal glucose metabolism. Semin Perinatol 3: 157-76.

Johnson J.D., Greenberg R.E. (1992): Protein turnover in rat placenta: effects of maternal fasting and maternal protein restriction. Placenta 13: 141-50.

Kim Y.J., Felig P. (1972): Maternal amniotic fluid substrate levels during caloric deprivation in human pregnancy. Metabolism 21: 507-12.

Koski K.G., Fergusson M.A. (1991); Amniotic fluid composition responds to changes in maternal dietary carbohydrate and is related to metabolic status in term fetal rats. Am. Institute of Nutrition 385-92.

Lemons J.A., Morrehead H.C., Hag G.P. (1986): Effects of fasting on gluconeogenic enzymes in the ovine fetus. Pediatr Res. 20: 676-9.

Leturque A., Burnol A., Ferre P., Girard J. (1984): Pregnancy-induced insulin resistance in the rat: assessment by glucose clamp technique. Am. J. Physiol 246: 25-31.

Leturque A., Ferre P., Burnol A., Kande J., Millard P., Girard J. (1986): Glucose utilization rates and insulin sensitivity in vivo in tissues of virgin and pregnant rats. Diabetes 35: 172-7.

Leturque A., Hauguel S., Ferre P., Girard J. (1987): Glucose metabolism in pregnancy. Biol Neonate 51: 64-9.

Leturque A., Hauguel S., Revelli J.P., Burnol A.F., Kande J., Girard J. (1989): Fetal glucose utilization in response to maternal starvation and acute hyperketonemia. Am. J. Physiol 256: 699-703.

Liechy E.A., Lemons J.A. (1984): Changes in ovine fetal hindlimb amino acid metabolism during maternal fasting. Am. J. Physiol 246: 430-5.

Maes M., Maiter D' Thissen J.P., Underwood L.E., Ketelstagers J.M. (1991): Contributions of growth hormone receptor and post receptor defects to growth hormone resistance in malnutrition. Trends Endocrinol 2: 92-7.

Malhotra A., Scott P.H., Scott J., Gee H., Wharton B.A. (1989): Metabolic changes in Asian Muslim pregnant mothers observing the Ramadan fast in Britain. Br. J. Nutr 61: 663-712.

Mohammad Z.A.N., Mohamad H.H., Sayida N., Ishrat P.S. (1989): Changes in blood urea and glucose and their association with energy-containing nutrients in men on hypocaloric diet during Ramadan. Am. J. Clin. Nutr 49: 1141-5.

Prentice A.M., Prentice A., Lamb W.H., Lunn P.G., Austin S. (1983): Metabolic consequences of fasting during Ramadan in pregnant and lactating women. Hum Nutr Clin. Nutr. 37 (4): 283-94.

Reeves J. (1992): Pregnancy and fasting during Ramadan. BMJ 304: 843-4.

Speroff L., Glass R.H., Kase N.G. (1984): Clinical Gynecologic Endocrinology and Infertility. Baltimore M.D: Williams & Wilkins.

Schreiner R.L., Lemons J.A., Gresham E.L. (1981): Metabolic and hormonal response to chronic maternal fasting in the ewe. Ann. Nutr. Metab 25: 38-47.

Sliman N.A., Katib F.A. (1988): Effect of fasting Ramadan on body weight and some blood constituents of healthy Muslims. Nutrition Report International 38 (6): 1299-306.

صيام رمضان وتأثيراته على الهرمونات التى تتحكم فى استقرار جلوكوز الدم

محمد شعراوى – أمال شبكشى – عفاف النورى

أقسام النساء والتوليد بكليتى الطب جامعتى القاهرة والملك عبد العزيز بالمملكة العربية السعودية

بالرغم من أن الشريعة الإسلامية تبيح للسيدات الحوامل الأفطار فى شهر رمضان ، إلا أن البعض تفضل الصيام مع عائلاتهن دون قضاء ذلك فى وقت آخر ، وقد أثبتت أحد الدراسات من قبل أن صوم رمضان أثناء الحمل قد يؤدى إلى هبوط توازن جلوكوز الدم ، لذا اجرينا هذه الدراسة لتقييم تأثير صوم رمضان على إفراز الهرمونات المتحكمة فى إستقرار جلوكوز الدم ، وقد تم أخذ عينات من الدم من ٢٥ سيدة حامل ، فى الصباح بعد صيام ١٢ ساعة ليلا ، وبعد الإفطار بساعتين (مجموعة ضابطة) بالإضافة إلى عينات أخرى من ثلاثين سيدة حامل حملاً طبيعياً من اللاتى صمن رمضان حيث أخذت العينة الأولى قبل إفطار رمضان مباشرة عن المغرب والعينة الأخرى بعد ساعتين من تناول طعام الإفطار ، وقد شملت المجموعتان سيدات حوامل فى الجزء الأول والثانى والثالث من الحمل ، هذا وقد تم قياس معدلات الجلوكوز وهرمونات الأنسولين والجلوكاجون والكورتيزول وهرمون النمو والسوماتوستاتين والهرمون اللبنى المشيمى الآدمى وذلك بطرق المناعة الإشعاعية .

وقد ثبتت أن صوم رمضان أثناء الجزء الأول من الحمل يؤدى إلى نقص ذى مغزى فى معدلات الجلوكوز والأنسولين مصحوبا بزيادة ذى مغزى فى معدل الهرمون اللبنى المشيمى الآدمى بينما لم يكن هناك أى تغيرات ذات مغزى فى باقى الهرمونات .

أما فى السيدات الصائمات فى الجزء الثانى أو الثالث من الحمل فلم يكن هناك أى تغيرات ذى مغزى فى جميع المقاييس المشار اليها سلفاً . هذا بالإضافة إلى أن جميع معدلات هذه المقاييس بعد الإفطار لم تتغير فى السيدات الصائمات بالمقارنة بالمجموعة الضابطة .

وخلاصة هذا البحث توضح أن التغيرات الهرمونية التى صحبت نقص جلوكوز دم الأم التى صامت فى الجزء الأول من الحمل ماهى إلاوسيلة للحفاظ على توازن التمثيل الغذائى للجنين ، علماً بأن إحتمال تأثير صيام الأم على احتياجات الجنين من الجلوكوز اللازم لنموه لا يمكن تجاهله ، هذا وقد أثبتت المقاييس بعد تناول الأفطار عدم إختلاف مستوى الجلوكوز والهرمونات المتحكمة فيه فى كل السيدات الصائمات وغير الصائمات ، مما يدل على أن التغيرات الهرمونية التى صاحبت الصيام فى الجزء الأول من الحمل كانت وقتية وتم عودتها إلى المعدلات الطبيعية بعد ساعتين من الأفطار .