

Correlation of Maternal and Cord Blood Leptin Concentrations with Neonatal Birth Weight in Term, Preterm and Growth-Restricted Fetuses

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Background: The study aimed to determine the association of cord blood and maternal leptin levels with neonatal birth weight in preterm deliveries, intrauterine growth retarded neonates, and term neonates.

Materials and Methods: Sixty parturient were enrolled in this prospective observational study, and data of fifty-one parturient were analyzed. There were nine dropouts. Of these patients, 20 had intrauterine growth restriction (Group I), 15 had moderate preterm labor (Group II), and 16 had labor with regular term pregnancies (Group III). Obstetric and demographic data were recorded for the mothers and neonates. Cord blood sampling was done just before clamping of the cord, and maternal venous blood was sampled within five minutes after parturition.

Results: Mean cord blood leptin levels in Groups I, Group II, and Group III were 2.29 ± 2.92 , 0.93 ± 0.64 , and 2.26 ± 1.78 , respectively. Maternal leptin levels in Group I, Group II, and Group III were 8.79 ± 5.92 , 4.91 ± 3.76 , and 5.83 ± 4.36 . Mean birth weights were 2395 ± 461 g in Group I, 2142 ± 464 g in Group II, and 3472 ± 419 g in Group III. There was no statistically significant difference in neonatal birth weight between Group I and Group II ($p=0.487$), whereas the difference was significant for both Group I and Group II compared to Group III ($p<0.001$).

Conclusion: Leptin has an essential role in intrauterine fetal growth. Cord blood leptin concentrations have a positive correlation with neonatal birth weight in preterm fetuses.

Keywords: Leptin, intrauterine growth restriction, preterm labor, pregnancy

Introduction

Intrauterine growth restriction is a severe health problem related to neonatal morbidity and mortality (1). The mortality rates in intrauterine growth restricted newborns are higher than term newborns with healthy intrauterine growth (2,3). Congenital anomalies, intra uterine infections, perinatal asphyxia, hypothermia, hypoglycemia, meconium aspiration

pulmonary hemorrhage, and are leading causes of morbidity and mortality in the growth-restricted newborns.

Leptin, a polypeptide hormone with 16kda molecular weight and 167 amino acids, is involved in many metabolic regulatory steps in humans (4-6). Under normal physiological conditions, leptin is produced by adipose tissue (6). In pregnancy, placental secretion of leptin

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dominates, and 95% is secreted to the maternal circulation (7, 8). Maternal leptin increase with increasing adiposity (9, 10). During pregnancy, maternal leptin levels increase gradually at the first trimester, reaches a peak at the second trimester and gains a plateau at the third trimester (11).

Although fetal cord blood leptin levels are lower than maternal leptin levels, it can be determined at birth. The source of fetal cord blood leptin is fetal adipose tissue (12). Cord blood leptin levels increase gradually until the 34th gestational week in association with the increasing neonatal adipose tissue, and term neonates have a six-fold higher leptin level than preterm neonates (13).

In the present study, we assessed the relationship of maternal and cord blood leptin levels with neonatal birth weight in fetuses with intrauterine growth restriction, preterm-appropriate for gestational age (AGA) fetuses, and term-AGA fetuses.

Materials and Methods

Subjects

After approval from the institutional review board and ethics committee, the observational study was prospectively designed. We enrolled sixty patients (20 patients in each group). There were nine dropouts (six patients did not meet inclusion criteria, and three patients declined to participate). Data obtained from 51 pregnant patients were considered for statistical analysis. All patients gave written informed consent. The study was conducted following the declaration of Helsinki. All patients had at least three antenatal visits. Gestational week of the patients was determined according to their last menstrual cycle and confirmed by obstetric ultrasound examination at their first antenatal

visit. Patients with a body mass index (BMI) greater than 26 kg/m² on the first antenatal visit, gestational diabetes, polycystic ovary syndrome, multiple pregnancies, hypertension, were excluded from the study.

Patients were divided into three subgroups. Patients with intrauterine growth-restricted fetuses were included in Group I (n=20), patients with preterm labor-appropriate for gestational age fetuses (AGA) were included in Group II (n=15), and term pregnancy-AGA fetuses were included in Group III (n=16). Gestational week of the patients was assessed with an ultrasound just before parturition. Patients with at least two weeks of delay in fetal growth according to the predetermined gestational week at their first antenatal visit and fetuses under ten percentile according to Brenner scale (14) were included in Group I. Patients that delivered before 37th gestational week with AGA fetuses were included in Group II. Patients delivered after 37th gestational week with AGA fetuses were included in Group III.

Previous obstetric history (gravid, parity, abortus, dilatation, and curettage) were recorded. Age, weight, height, BMI at first antenatal visit, and delivery were recorded. Amniotic index, route of parturition, indication for cesarean section, neonatal birth weight, Apgar score, and gender of the newborn were recorded.

Patients Blood Sampling

Maternal blood samples were obtained within five minutes following delivery. Cord blood was sampled after birth just before cord clamping. Blood samples were centrifuged at 2000 rpm for ten minutes and stored at -20 °C. Analysis of blood samples was carried out with BioSource Leptin Easia Kit (Biosource Europe S.A., I'Industrie 8B-1400, Nivelles, Belgium).

Values between 3.6–9.6 ng/ml were accepted in the normal range for leptin.

Statistical Analysis

Statistical package for the social sciences program (IBM, SPSS, Chicago, IL, USA) was used for the analysis of data. Qualitative data were analyzed with the chi-square test. Normal distribution of data was analyzed with the Shapiro–Wilk test. The comparison of data between groups for the normally distributed data was analyzed with a one-way analysis of variance. If the difference was statistically significant, Tukey test was used for multiple comparisons. Data without normal distribution were analyzed with the Kruskal-Wallis test. A significant difference was analyzed with the Mann-Whitney-U after Bonferroni correction. Correlation of leptin levels with other study variables was analyzed with Pearson and Spearman correlation tests as appropriate. P value <0.05 was accepted as a statistically significant difference.

Results

In the present study, we analyzed the data collected from 51 pregnant women. The age of the patients included in the study ranged from 18 to 33 years. The distribution of age in groups was comparable. ($p=0.596$) Pregravid BMI of the patients was comparable between groups and ranged from 16.61–24.98 kg/m² ($p=0.707$). BMI at delivery ranged from 20.76 to 31.64, and there was no statistically significant difference between groups ($p=0.258$). Gestational week ranged from 33 weeks to 41 weeks-three days in Group I. Gestational week in Group II ranged from 27 weeks-five days to 36 weeks-three days. Gestational week in Group III ranged from 37 weeks-four days to 41 weeks-three days. The difference in the gestational week at

parturition was statistically significant ($p<0.001$). Of 51 pregnant patients, 23 (45.1%) of them were primiparous, and 28 (54.9%) were multi parous. The distribution of patients was comparable between groups according to parity (0.667) (Table-1).

The amniotic index was evaluated with ultrasound scanning (Logic 200 Pro, GE Company, Malaysia) just before labor and patients that had amniotic index < 50 mm was accepted as oligohydramnios. Fifteen patients (75%) in Group I, four patients (26.7%) in Group II, and one patient (6.3%) in Group III had oligohydramnios. Oligohydramnios was more frequent in Group I. ($p<0.001$) Cesarean section rates were 65%, 20%, and 37.5% in Group I, II, and III respectively and the higher rate in Group I was statistically significant ($p<0.001$) The indications of cesarean section are presented in Table 2. Cesarean section due to fetal distress was higher in Group I ($p<0.001$)

Fetal birth weight in Group I was 2395±461 g, in Group II 2142±464g, and 3472±419 g in Group III. Fetal birth weight was comparable between Group I and Group II ($p=0.487$), but both groups had a lower fetal birth weight than Group III ($p<0.001$).

Maternal leptin levels in Group I was significantly higher than Group II and Group III ($p=0.023$). The maternal leptin levels were comparable between Group II and Group III ($p=0.762$). The change in maternal leptin levels, according to gestational week was comparable between groups ($p=0.421$). There was a positive correlation between maternal leptin concentrations and BMI at delivery in Group I ($p=0.027$) and Group III ($p=0.046$), but there was not a significant correlation in Group II ($p=0.073$) (Figure-1). Maternal leptin levels did not have a significant correlation with neonatal

birth weight in Group I ($p=0.066$, $r= -0.419$), Group II ($p=0.525$, $r=0.178$) and Group III ($p=0.433$, $r= -0,211$) (Figure-2).

Cord blood leptin concentration was lower in Group II than Group I and Group III ($p<0.001$). Cord blood leptin concentrations were comparable between Group I and Group III ($p=0.256$). There was a positive correlation between cord blood leptin concentration and fetal birth weight in Group II ($p=0.016$) but Group I and Group III did not have a correlation ($p=0.766$ and 0.863 respectively, Figure-3). Besides, there was not any correlation between

the fetal gender and cord blood leptin concentrations in all groups ($r=0.257$, 0.254 , and 0.132 respectively). There was not any significant correlation between maternal leptin concentrations and cord blood leptin concentrations in all groups ($p=0.720$, 0.970 , 0.289 , respectively, Figure-4). Neonates born with cesarean section in Group I had a higher cord leptin concentration than newborns delivered vaginally ($p=0.017$). Cord leptin concentrations in Group II and Group III were comparable between vaginally delivered newborns and those delivered with cesarean section ($p=0.347$, 0.642 respectively).

Table-1. Maternal and neonatal characteristics

Patient Characteristics	Group I (n:20)	Group II (n:15)	Group III (n:16)	P Value
Age (years)	24.35±3.1	24.73±3.43	25.63±4.33	0.596
Pregravid BMI (kg/m ²)	21.25±2.58	21.91±2.24	21.63±2.14	0.707
BMI at parturition (kg/m ²)	26.03±3.13	26.03±2.49	27.38±2.21	0.258
Gestational week (week)	38.3±2.2	33.2±2.4	39.1±1.2	<0.001
Primiparous (n)	10	7	6	0.667
Multiparous (n)	10	8	10	0.667
Oligohydramnios (n)	15	4	1	<0.001
Cesarean delivery (n)	13	3	6	<0.001
Vaginal delivery (n)	7	12	10	<0.001
Neonatal birth weight (g)	2895±461	2142±464	3472±419	<0.001
Maternal Leptin (ng/ml)	8.79±5.92	4.91±3.76	5.83±4.36	0.023
Cord Leptin (ng/ml)	2.29±2.92	0.93±0.64	2.26±1.78	<0.001
Neonatal Gender (F/M)	13 / 7	6 / 9	6 / 10	0.257

Table-2. Indications for Cesarean Section

Groups	Fetal distress (n, %)	Previous cesarean (n, %)	Other reasons (n, %)	Total (n, %)
Group - I	7, 53%	2, 15%	4, 32%	13, 100%
Group - II	1, 33.3%	0, 0%	2, 66.7%	3, 100%
Group - III	1, 16.7%	4, 66.6%	1, 16.7%	6, 100%
Total	9, 40.9%	6, 27.3%	7, 31.8%	22, 100%

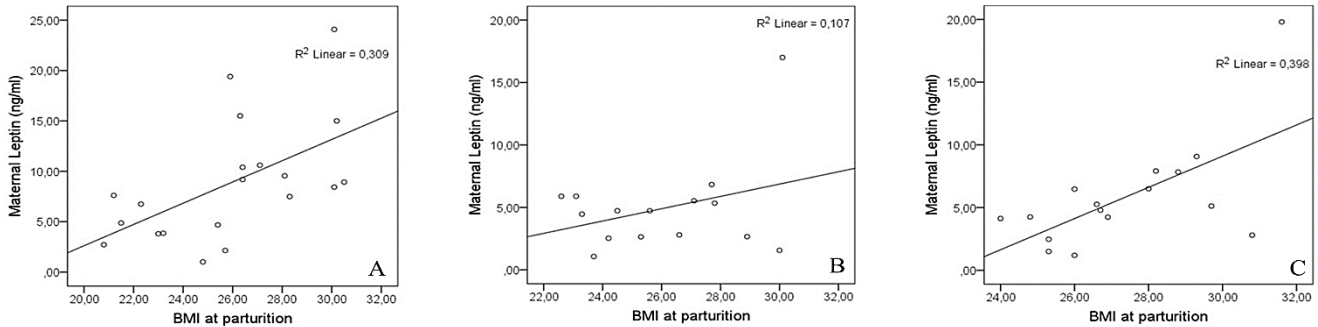


Figure-1. Correlation of maternal leptin concentrations with maternal BMI at parturition. A: Group-I (intrauterine growth restricted fetuses group), B: Group-II (preterm fetuses with appropriate for gestational age group), C: Group-III (term fetuses with appropriate for gestational age fetuses), BMI: body mass index (kg/m²), R²: Coefficient of determination.

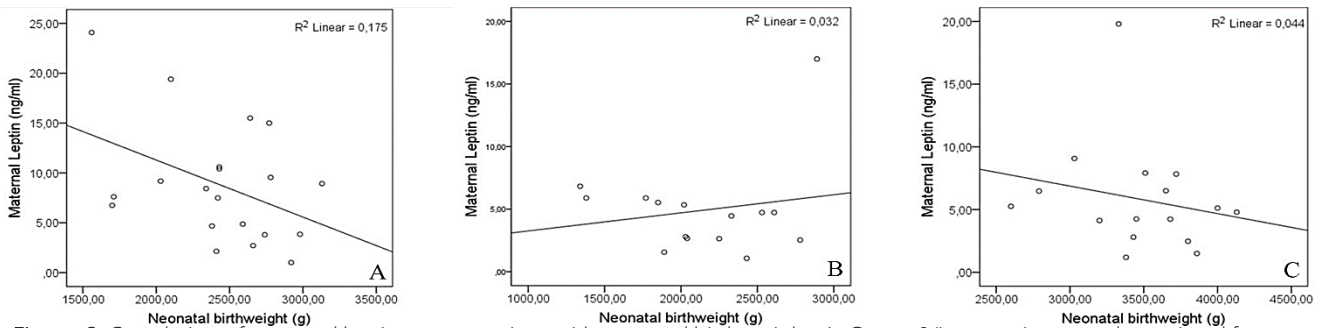


Figure-2. Correlation of maternal leptin concentrations with neonatal birth weight. A: Group-I (intrauterine growth restricted fetuses group), B: Group-II (preterm fetuses with appropriate for gestational age group), C: Group-III (term fetuses with appropriate for gestational age fetuses), BMI: body mass index (kg/m²), R²: Coefficient of determination.

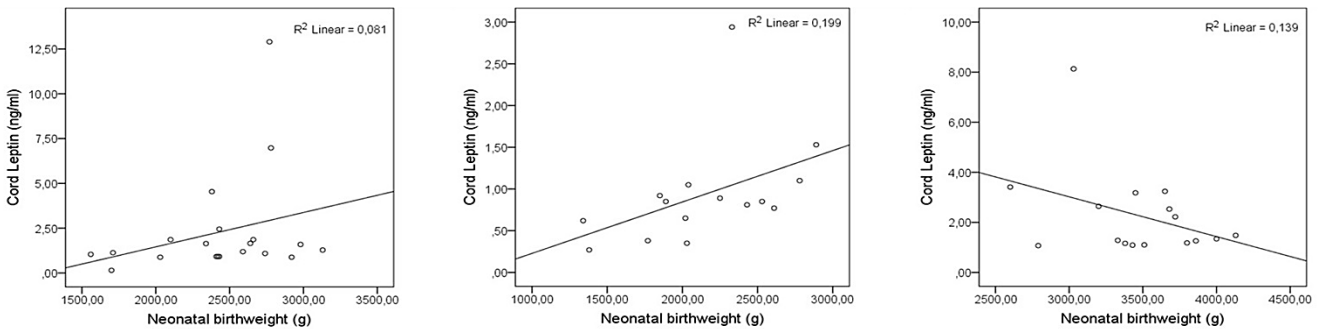


Figure-3. Correlation of cord leptin concentrations with neonatal birth weight. A: Group-I (intrauterine growth restricted fetuses group), B: Group-II (preterm fetuses with appropriate for gestational age group), C: Group-III (term fetuses with appropriate for gestational age fetuses), BMI: body mass index (kg/m²), R²: Coefficient of determination.

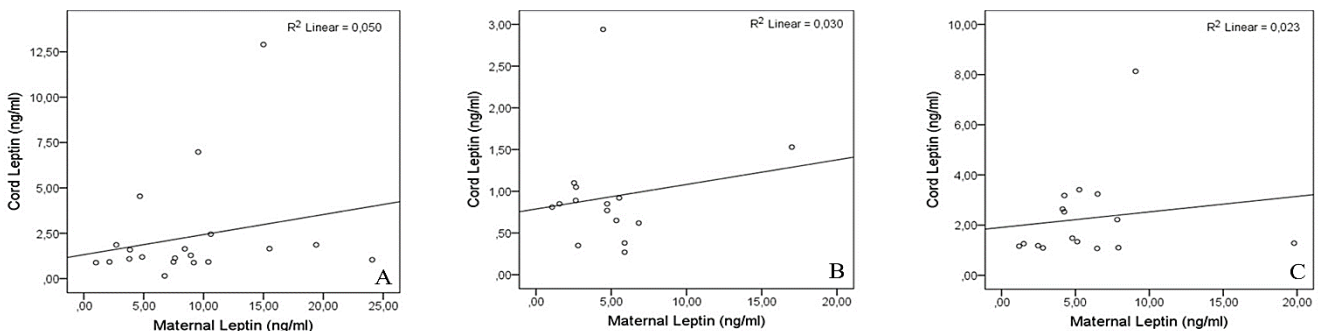


Figure-4. Correlation of cord leptin concentrations with maternal leptin concentrations. A: Group-I (intrauterine growth restricted fetuses group), B: Group-II (preterm fetuses with appropriate for gestational age group), C: Group-III (term fetuses with appropriate for gestational age fetuses), BMI: body mass index (kg/m²), R²: Coefficient of determination.

Discussion

Results of the present study demonstrated that maternal leptin concentrations have a positive correlation with maternal BMI but not with cord blood leptin concentrations and fetal birth weight. There was a positive correlation between cord blood leptin concentrations and fetal birth weight in preterm-AGA newborns and term-AGA newborns. Although neonates with intrauterine growth restriction have a higher cord blood leptin levels, it does not have a significant correlation with fetal birth weight.

Tamura et al. (15) reported that there was a positive correlation between pregravid BMI and maternal leptin concentrations. Therefore, in the present study, we excluded patients with BMI > 26 kg/m² to prevent bias on the results of maternal leptin concentrations. In our study, we found a positive correlation between maternal leptin concentrations and BMI at parturition in Group I and Group III, but there was not a significant correlation in Group II, consistent with the results of Tamura et al (15).

The effect of maternal leptin concentrations on neonatal outcome was assessed in a recent study (16). The authors analyzed the effect of maternal leptin on neonatal birth weight, length, and the sum of skinfold with pregravid obese and non-obese mothers. They concluded that leptin levels were not associated with birth weight when stratified by obesity status. They also concluded that maternal leptin levels are associated with multiple pathways that influence fetal growth differentially, depending on the timing in pregnancy and maternal pregnancy obesity status. In the present study, we enrolled pregravid non-obese mothers and following the study by Hinkle et al. we could not find a significant

correlation between the maternal leptin levels and neonatal birth weight.

Tamura et al.(15) reported that maternal leptin concentrations were comparable between the intrauterine growth restriction group and the control group. Pighetti et al. (17) reported that a maternal serum leptin concentration in the growth-restricted group was higher than patients with term pregnancy-AGA fetuses. Our results were in accordance with the results of Pighetti et al. (17) but in contrast with the results of Tamura et al. (15) which can be explained by the patients included in the study groups. In the study by Pighetti et al. and the present study, patients with pregravid BMI >26 kg/m² were excluded to prevent bias on the maternal leptin concentrations.

Previous studies reported positive correlation between maternal blood leptin concentrations and preterm labor (18, 19). They suggested that maternal leptin levels in term-AGA infants were higher than preterm-AGA infants. Our results were in accordance with the results of these previous studies.

Conflicting results have been reported on the relationship between cord blood leptin concentrations and intrauterine fetal growth. While some of them reported that cord leptin concentrations in IUGR fetuses were lower than term-AGA fetuses (12,20), others have reported that IUGR have higher leptin concentrations (21, 22). Takahashi et al. suggested that the chronic hypoxic conditions and the acute hypoxic periods during parturition with uterine contractions are responsible for the elevated cord blood leptin concentrations in IUGR fetuses. In the present study, fetal cord blood leptin levels in Group-I was comparable to Group III, but both were higher than the cord blood leptin levels in Group II. However, serum

leptin levels adjusted for the gestational week were comparable in all groups in this study.

A positive correlation was reported between fetal cord blood leptin concentrations and neonatal birth weight (23-25). On the other hand, Clapp et al. (26) have assessed the relationship of cord blood leptin levels with maternal weight, weight gain during pregnancy, maternal fat ratio, placenta weight, neonatal birth weight, and neonatal fat ratio. They reported that there was a positive correlation, only, between cord blood leptin concentration and fetal fat ratio. In both Cetin et al.'s (12) and Jaquet et al.'s (20) studies, cord blood leptin levels were lower in growth-restricted fetuses than AGA fetuses after 34th gestational week. However, it was also reported in the latter 2 studies that leptin concentrations were comparable between IUGR and AGA fetuses when adjusted for fetal birth weight (12, 20). In the present study, we could not find any significant correlation between cord blood leptin levels and neonatal birth weight in Group I and Group III; however, there was a positive correlation in Group II. In the present study, fetal distress rate during parturition was higher in Group I when compared to Group II and Group III. The stress condition may have increased the production and secretion of fetal leptin. This may explain the insignificant difference in cord blood leptin levels between Group I and Group III.

Yoshimitsu et al. (27) have assessed the effect of vaginal delivery and an elective cesarean section on the level of cord blood leptin concentrations. They concluded that cord blood leptin concentration in the vaginal delivery group was higher than the elective cesarean section group. They accused the stress condition during vaginal delivery were

more than the elective cesarean section, which results in increased cord blood leptin levels. In our study, we found higher cord blood leptin levels in patients undergoing cesarean section than those delivering vaginally in Group I. There was not any significant relationship between the route of parturition and cord blood leptin concentrations in Group II and Group III. In Group I, 65% of patients delivered with cesarean section. The indication of cesarean section was fetal distress in 53% of patients. The cesarean sections in the study by Yoshimitsu et al. (27) were elective cases, unlike our study, whereas more than half of cesarean sections were carried out in an emergency setting in our study. This may explain why the cord blood leptin concentrations were comparable between Group I and Group III in the present study.

Several previous studies reported that female gender is associated with higher cord blood leptin levels (24, 28). However, others have reported that cord blood leptin concentration is not affected by gender (12, 29). In the present study, there was not any significant correlation between the cord blood leptin concentrations and neonatal gender.

One of the limitations of the present study was patients undergoing cesarean section in Group I were mostly emergent cesarean sections with fetal distress. This may have influenced the leptin concentration levels in maternal serum and cord blood. Another limitation was the relatively small sample size of the study groups, although fifteen patients per group were found to be appropriate to conclude for a statistically significant difference.

Leptin is an crucial polypeptide hormone that has an essential role in fetal growth. Maternal obesity, maternal leptin concentrations, the

timing of pregnancy, and cord blood leptin concentrations may affect neonatal growth. Further studies are needed to investigate the effect of these parameters in relation to leptin on fetal growth. Moreover, we suggest that future studies with higher sample sizes are warranted to validate our results.

In conclusion, leptin has an essential role in intrauterine fetal growth. Unlike maternal leptin concentrations, cord blood leptin levels have a positive correlation with neonatal birth weight in preterm newborns. Further studies with larger sample sizes are warranted to validate our results.

Conflict of Interests

The author declares that there is no conflict interests in the present study.

Reference

1. Beck S, Wojdyla D, Say L, Betran AP, Merialdi M, Requejo JH, et al. The worldwide incidence of preterm birth: a systematic review of maternal mortality and morbidity. *Bull World Health Organ* 2010;88:31-8
2. Black RE, Cousens S, Johnson HL, Lawn JE, Rudan I, Bassani DG, et al. Global, regional, and national causes of child mortality in 2008: a systematic analysis. *Lancet* 2010;375:1969-87
3. Liu L, Johnson HL, Cousens S, Perin J, Scott S, Lawn JE, et al. Global, regional, and national causes of child mortality: an updated systematic analysis for 2010 with time trends since 2000. *Lancet* 2012;379:2151-61
4. Henson MC, Castracane VD. Leptin in pregnancy: an update. *Biol Reprod* 2006;74:218-29
5. Mantzoros CS, Moschos SJ. Leptin: in search of role(s) in human physiology and pathophysiology. *Clin Endocrinol* 1998;49:551-67
6. Zhang Y, Proenca R, Maffei M, Barone M, Leopold L, Friedman JM. Positional cloning of the mouse obese gene and its human homologue. *Nature* 1994;372:425-32
7. Lepercq J, Guerre-Millo M, Andre J, Cauzac M, Hauguel-de Mouzon S. Leptin: a potential marker of placental insufficiency. *Gynecol Obstet Invest* 2003;55:151-5
8. Linnemann K, Malek A, Sager R, Blum WF, Schneider H, Fusch C. Leptin production and release in the dually in vitro perfused human placenta. *J Clin Endocrinol Metab* 2000;85:4298-301
9. Bouloumie A, Drexler HC, Lafontan M, Busse R. Leptin, the product of Ob gene, promotes angiogenesis. *Circ Res* 1998;83:1059-66
10. Mise H, Yura S, Itoh H, Nuamah MA, Takemura M, Sagawa N, et al. The relationship between maternal plasma leptin levels and fetal growth restriction. *Endocr J* 2007;54:945-51
11. Hauguel-de Mouzon S, Lepercq J, Catalano P. The known and unknown of leptin in pregnancy. *Am J Obstet Gynecol* 2006;194:1537-45
12. Cetin I, Morpurgo PS, Radaelli T, Taricco E, Cortelazzi D, Bellotti M, et al. Fetal plasma leptin concentrations: relationship with different intrauterine growth patterns from 19 weeks to term. *Pediatr Res* 2000;48:646-51
13. Dotsch J, Nusken KD, Knerr I, Kirschbaum M, Repp R, Rascher W. Leptin and neuropeptide Y gene expression in human placenta: ontogeny and evidence for similarities to hypothalamic regulation. *J Clin Endocrinol Metab* 1999;84:2755-8
14. Brenner WE, Edelman DA, Hendricks CH. A standard of fetal growth for the United States of America. *Am J Obstet Gynecol* 1976;126:555-64
15. Tamura T, Goldenberg RL, Johnston KE, Cliver SP. Serum leptin concentrations during pregnancy and their relationship to fetal growth. *Obstet Gynecol* 1998;91:389-95
16. Hinkle SN, Rawal S, Liu D, Chen J, Tsai MY, Zhang C. Maternal adipokines longitudinally measured across pregnancy and their associations with neonatal size, length, and adiposity. *Int J Obes* 2018 Nov 21
17. Pighetti M, Tommaselli GA, D'Elia A, Di Carlo C, Mariano A, Di Carlo A, et al. Maternal serum and umbilical cord blood leptin concentrations with fetal growth restriction. *Obstet Gynecol* 2003;102:535-43
18. Fakor F, Sharami SH, Milani F, Mirblouk F, Kazemi S, Pourmarzi D, et al. The association between level of maternal serum leptin in the third trimester and the occurrence of moderate preterm labor. *J Turk Ger Gynecol Assoc* 2016;17:182-5
19. Shroff MR, Holzman C, Tian Y, Evans RW, Sikorskii A. Mid-pregnancy maternal leptin levels, birthweight for gestational age and preterm delivery. *Clin Endocrinol* 2013;78:607-13
20. Jaquet D, Leger J, Levy-Marchal C, Oury JF, Czernichow P. Ontogeny of leptin in human fetuses and newborns: effect of intrauterine growth retardation on serum leptin concentrations. *J Clin Endocrinol Metab* 1998;83:1243-6
21. Shekhawat PS, Garland JS, Shivpuri C, Mick GJ, Sasidharan P, Pelz CJ, et al. Neonatal cord blood leptin: its relationship to birth weight, body mass index, maternal diabetes, and steroids. *Pediatr Res* 1998;43:338-43
22. Takahashi Y, Yokoyama Y, Kawabata I, Iwasa S, Tamaya T. Leptin as an acute stress-related hormone in the fetoplacental circulation. *Obstet Gynecol* 2002;100:655-8

23. Harigaya A, Nagashima K, Nako Y, Morikawa A. Relationship between concentration of serum leptin and fetal growth. *J Clin Endocrinol Metab* 1997;82:3281-4
24. Matsuda J, Yokota I, Iida M, Murakami T, Naito E, Ito M, et al. Serum leptin concentration in cord blood: relationship to birth weight and gender. *J Clin Endocrinol Metab* 1997;82:1642-4
25. Chen H, Xu L, Zhu W, Wu Y, Xu M, Wang Z. Impact of Cord Blood Adiponectin and Leptin Levels and Maternal Obesity on Birth Weight of Infants Born to Women with Gestational Diabetes Mellitus. *J Reprod Med.* 2017;62(3-4):179-83
26. Clapp JF, 3rd, Kiess W. Cord blood leptin reflects fetal fat mass. *J Soc Gynecol Investig* 1998;5:300-3
27. Yoshimitsu N, Douchi T, Kamio M, Nagata Y. Differences in umbilical venous and arterial leptin levels by mode of delivery. *Obstet Gynecol* 2000;96:342-5
28. Kayemba-Kay's S, Geary MP, Pringle J, Rodeck CH, Kingdom JC, Hindmarsh PC. Gender, smoking during pregnancy and gestational age influence cord leptin concentrations in newborn infants. *Eur J Endocrinol* 2008;159:217-24
29. Pardo IM, Geloneze B, Tambascia MA, Barros-Filho AA. Does maternal smoking influence leptin levels in term, appropriate-for-gestational-age newborns? *J Matern Fetal Neonatal Med* 2004;15:408-10

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