Sarcouncil Journal of Biomedical Sciences

ISSN(Online): 2945-3666

Volume- 03| Issue- 06| 2024

Research Article

Received: 20-11-2024 | Accepted: 15-12-2024 | Published: 30-12-2024

Examining the Effects of Maternal Spexin and Ghrelin Levels on Neonatal Outcomes, with Emphasis on Birth Weight and Bilirubin Levels

Dr. Malak Saeid Muhsin Al-Akraa[1] and Dr. Amal Abdul Muhsin Hussain Al-Zubaidy[2]

[1,2]M.B.Ch.B., F.I.C.O.G. \ (Obstetrics and Gynecology) Specialist Obstetrics and Gynaecology. Iraqi Ministry of Health, Al-Diwaniyah Health Directorate, Al-Diwaniyah Teaching Hospital, Al-Diwaniyah, Iraq.

Abstract: This paper examined the relationships between maternal serum concentrations of spexin and ghrelin with birth weight of neonatal and bilirubin in term pregnancies. This is a prospective cohort study that was done using 97 pregnant women who were in term (greater than 37 weeks of gestation) with subsequent approval and informed consent by the Institutional Review Board. Blood samples of the mother were taken within 24 hrs before delivery, following an overnight starvation of the people to take measurements of spexin and ghrelin using commercial ELISA kits. Medical records were used to record neonatal outcomes such as birth weight, bilirubin (48 hours of postpartum), gestational age, gender, and low birth weight (<2500 g). Analyses were performed on Pearson correlations, multivariate linear regression controlled by maternal age, gestational age, and BMI, and one-way ANOVA by hormone tertiles ($p<0.05$ significant). Maternal spexin was 2.525g, + 0.8 NgmL and positively related with birth weight ($r=0.25$, $p=0.014$) and negatively with bilirubin ($r=0.22$, $p=0.032$). Spexin in regressions significantly predicted an increase in birth weight (42.5, $p=0.010$) and a decrease in bilirubin (0.85, $p=0.003$), and the gestational age and BMI were also important. Tertile statistics affirmed that high spexin levels were related to high birth weight ($p=0.032$) and low bilirubin ($p=0.001$); high levels of ghrelin tertiles were correlated with high bilirubin ($p=0.008$) and finally we concluded Increased maternal spexin levels are linked to good birth outcomes such as high birth weight and reduced bilirubin and this effect is independent of confounders indicating that it is protective in term pregnancies.

Keywords: Maternal, ghrelin levels, neonatal, emphasis, bilirubin levels, spexin birth outcomes.

INTRODUCTION

Maternal spexin and ghrelin levels are interesting biomarkers potentially associated with neonatal outcomes, especially birth weight and neonatal bilirubin also as introduction spexin is a novel peptide hormone, and expressed in various tissues, such as adipose tissue, placenta, and central nervous system, which has been highlighted for its potential involvement in energy homeostasis, glucose metabolism, and lipid metabolism [Kitamura, S. *et al*., 2003]. Described as the "hunger hormone," ghrelin is known to regulate appetite, growth hormone (gh) secretion, and gastrointestinal function with implications for fetal development through placental transport 64. ghrelin, the so-called "hunger hormone," is known to be involved in the control of appetite and growth hormone (gh) release, but, as will be discussed in this review, also exerts effects on gastrointestinal function and indirectly impacts fetal development via placental transfer as well as regulation of fetal energy balance [Kojima, M. *et al*., 1999; Nakazato, M. *et al*., 2001]. Combining spexin and ghrelin at a maternal level can affect the intrauterine environment and have an impact on fetal growth trajectories as well as postnatal metabolism, hepatic processing resulting in birthweight and bilirubin process, as well as birthweight is an important neonatal outcome, indicating fetal growth and in utero nutrition [Wren, A. M. *et al*., 2001; Farquhar, J. *et al*., 2003]. It is influenced by an intricate interplay of maternal metabolic profile, placental physiology, fetal genetic make-up, and environmental influences, as well as, can we mention in recent years, the focus has also been pointed in the direction of adipokines and gut-derived hormones that either cross or signal through the placenta to influence fetal growth [James, R. J. A. *et al*., 2004; Adibi, J. J. *et al*., 2021; Adibi, J. J. *et al*., 2021]. Spexin is involved in lipid and glucose metabolism; plasma spexin was higher in subjects with hyperlipidaemia and lower in patients with newly diagnosed diabetes, indicating that increased or decreased maternal spexin may affect placental transportation of nutrients or fetal substrate availability whereas previous study if spexin does have pro-lean metabolism actions or peripheral insulin sensitivity, increasing pregnancy concentrations could be associated with more beneficial fetal growth patterns in some settings and may facilitate the achievement of higher normal birth weights in addition to it is not straightforward how ghrelin relates to birth weight forever in such studies, it is crucial to adjust for factors such as maternal bmi, parity, gestational diabetes, hypertensive disorders, smoking, medication use, and parity which are already known to affect the outcomes [Perez Garrido, N. I.

*Corresponding Author: Dr. Malak Saeid Muhsin Al-Akraa

DOI- https://doi.org/10.5281/zenodo.18317676

et al., 2021; Holt, R. I. 2002; Gohlke, B. C. *et al*., 2005; Stawerska, R. *et al*., 2016].

On the other hand, increased spexin levels might, under certain circumstances, suppress specific placental transporters, thus lessening the availability of nutrients to the fetus and causing growth restriction. The possible mechanisms by which ghrelin can influence growth are its stimulation of the secretion of growth hormone, its role in the regulation of the insulin-like growth factor axis, and its impact on placental blood flow and nutrient supply. [Pietrzak, M. *et al*., 2024; Wang, Y. *et al*., 2021] Through these mechanisms, ghrelin will be able to modulate fetal growth rate and birth weight. Furthermore, ghrelin has been linked to liver development and energy metabolism, which means that it may have an overall influence on the liver's postnatal growth and, subsequently, on the ability to process bilirubin [Lang, R. *et al*., 2015].

Regarding bilirubin, newborns need their liver to be physiologically adjusted in order to be able to maintain the metabolic status in a postnatal environment after birth, with a completely changed oxygen supply, blood circulation, and nutrition. Hence, if maternal spexin or ghrelin changes the fetal hepatic development or enzyme synthesis, then the newborns' reaction to bilirubin will be altered as well. Furthermore, research in the future should also look at the possibility that these hormones have sexually dimorphic activities, considering that the short-term impact of fetal sex on the maternal hormonal milieu is known to influence growth patterns and neonatal metabolism [Kumar, S. *et al*., 2016; Sanli, S. *et al*., 2021]. It will be worthwhile to check for possible feedback mechanisms whereby fetal development and neonatal outcomes influence maternal spexin and ghrelin levels after birth, which would help to resolve the maternal–fetal hormonal intercommunication puzzle. Another point that needs more exploration is the application of multi-omics comprising genomics, transcriptomics, metabolomics, and proteomics integrated with hormone profiling because this could assist researchers in identifying the mechanisms that connect maternal spexin and ghrelin to birth weight and bilirubin metabolism in the context of the highly complex fetal development process. [Ojha, S. *et al*., 2013; Ong, K. K., & Dunger, D. B. 2004; Savvidou, M. D. *et al*., 2008]

MATERIAL AND METHOD

This prospective cohort study enrolled 97 women with a single pregnancy, who gave birth during the period of January 2023 and December 2024 at Iraqi hospital and who had reached term at delivery (gestational age ≥37 weeks) as well as according to The study received the green light from the local Institutional Review Board. All participants gave their informed written consent. Candidates were required to be singleton pregnancy, have no maternal chronic conditions, such as diabetes or hypertension, no fetal anomalies, and non-usage of appetite hormones like spexin or ghrelin of medication in their history. Data on maternal age, parity, BMI, gestational age at delivery, together with serum spexin and ghrelin levels were documented; the blood for the hormone tests was drawn by a venipuncture within 24 hours before delivery after the subjects had fasted overnight, the samples were immediately centrifuged and then frozen at -80°C until used for the analysis.

Serum levels of spexin were measured with the help of a human spexin sandwich ELISA kit, which had a detection range of 78.13-5000 pg/mL and a sensitivity of 46.88 pg/mL, while the quantification of ghrelin was performed by a human ghrelin ELISA kit calibrated to pg/mL. Both tests are being done in duplication in accordance with the protocols provided by manufacturers, and with the variability of results for intra- and inter-assay kept under 10%. The newborn outcomes included were weight at birth in grams, serum bilirubin in mg/dL determined by the standard method of spectrophotometric assay from either the cord blood or blood obtained within the first 48 hours post-delivery (high bilirubin was considered when the level was >12 mg/dL), gestational age which was determined at the first ultrasound, gender, and low birth weight (<2500 g). This information was obtained from the medical notes in addition to buy use SPSS found The differences between groups were analyzed with the help of ANOVA or chi-square test based on the nature of the variables. Also, the results of the linear regression were presented by beta coefficients with 95% confidence intervals and p-values. By dividing maternal spexin and ghrelin levels into tertiles (n=32,33,32), i.e., low, medium, and high, the one-way ANOVA method was used for the comparison of outcomes. At all levels, $p<0.05$ was considered statistically significant; the sample size was calculated beforehand, allowing

Publisher: SARC Publisher

for the detection of correlations ≥0.25 with 80% power and α=0.05.

RESULTS

Table 1: Assessment outcomes of patients related to Maternal Characteristics

Characteristic	Mean ± SD or n (%)
Age (years)	28.5 ± 5.2
Gestational Age (weeks)	38.4 ± 1.8
Parity (nulliparous)	52 (53.6%)
BMI (kg/m²)	26.3 ± 4.1
Spexin (ng/mL)	2.5 ± 0.8
Ghrelin (pg/mL)	450.2 ± 120.5
Neonatal Outcomes (n=97)	
Birth Weight (g)	3200 ± 450
Bilirubin (mg/dL)	8.5 ± 2.3
Gestational Age (weeks)	38.5 ± 1.7
Male Gender	50 (51.5%)
Low Birth Weight (<2500g)	8 (8.2%)
High Bilirubin (>12 mg/dL)	10 (10.3%)

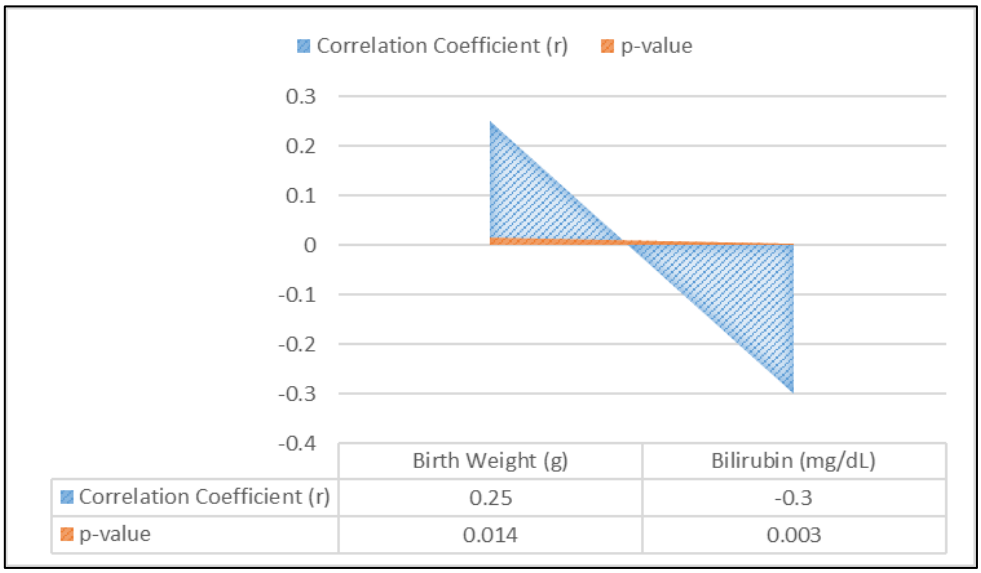

Figure 1: Find the Correlation of Maternal Spexin with Neonatal Outcomes (Pearson r)

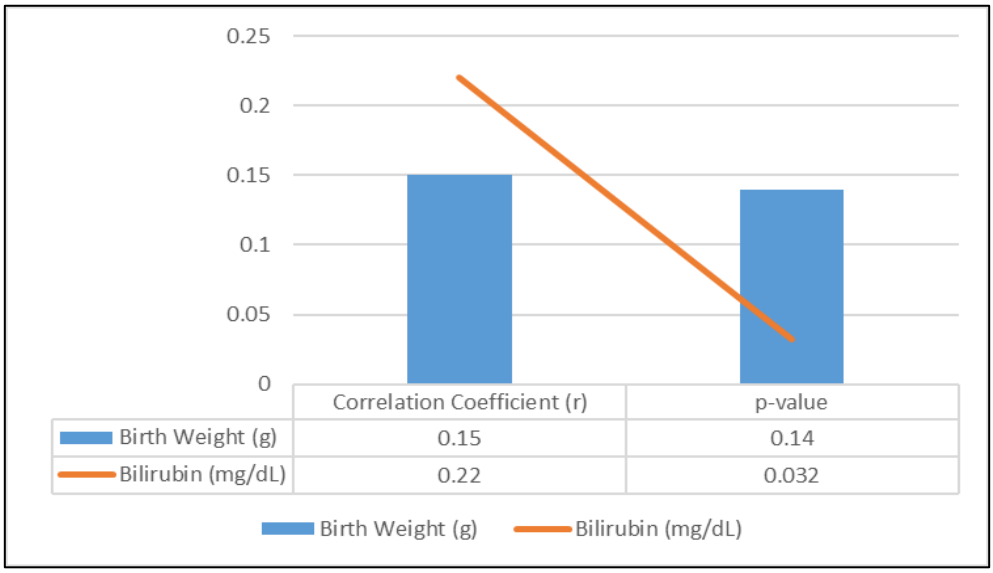

Figure 2: Assessment relationship of Maternal Ghrelin with Neonatal Outcomes

Publisher: SARC Publisher

Table 2: Rate finding based on Linear Regression for Birth Weight (g)

Variable	Beta (95% CI)	p-value
Maternal Spexin	42.5 (10.2, 74.8)	0.010
Maternal Ghrelin	0.25 (-0.10, 0.60)	0.160
Maternal Age	-5.2 (-12.0, 1.6)	0.130
Gestational Age	210.0 (180.0, 240.0)	<0.001
BMI	15.0 (5.0, 25.0)	0.004

Table 3: Linear Regression for Bilirubin (mg/dL) of the finding study for 97 patients

Variable	Beta (95% CI)	p-value
Maternal Spexin	-0.85 (-1.40, -0.30)	0.003
Maternal Ghrelin	0.003 (0.001, 0.005)	0.040
Maternal Age	0.05 (-0.10, 0.20)	0.510
Gestational Age	-0.30 (-0.50, -0.10)	0.004
BMI	-0.10 (-0.20, 0.00)	0.060

Table 4: Assessment of the overall study of Neonatal Outcomes by Tertiles of Maternal Spexin

Outcome	Tertile 1 (Low) n=32	Tertile 2 (Med) n=33	Tertile 3 (High) n=32	p-value
Birth Weight (g)	3050 ± 400	3250 ± 420	3300 ± 480	0.032
Bilirubin (mg/dL)	9.8 ± 2.5	8.2 ± 2.0	7.5 ± 1.8	<0.001

Table 5: Final Neonatal Outcomes by Tertiles of Maternal Ghrelin

Outcome	Tertile 1 (Low) n=32	Tertile 2 (Med) n=33	Tertile 3 (High) n=32	p-value
Birth Weight (g)	3120 ± 440	3220 ± 460	3260 ± 450	0.280
Bilirubin (mg/dL)	7.8 ± 2.0	8.3 ± 2.1	9.4 ± 2.5	0.008

Table 6: Assessment outcomes of study based on Maternal Characteristics by Birth Weight Groups

Characteristic	Low Birth Weight (n=8)	Normal Birth Weight (n=89)	p-value
Age (years)	30.2 ± 6.1	28.3 ± 5.0	0.290
Gestational Age (weeks)	36.0 ± 1.5	38.6 ± 1.7	<0.001
Spexin (ng/mL)	1.8 ± 0.5	2.6 ± 0.8	0.008
Ghrelin (pg/mL)	480.0 ± 130.0	448.0 ± 120.0	0.450
BMI (kg/m²)	24.5 ± 3.5	26.5 ± 4.1	0.180

Table 7: Assessment outcomes of study based on Maternal Characteristics by Bilirubin Groups

Characteristic	Normal Bilirubin (n=87)	High Bilirubin (n=10)	p-value
Age (years)	28.6 ± 5.3	27.8 ± 4.8	0.630
Gestational Age (weeks)	38.6 ± 1.7	37.5 ± 1.8	0.050
Spexin (ng/mL)	2.6 ± 0.8	1.9 ± 0.6	0.004
Ghrelin (pg/mL)	445.0 ± 115.0	495.0 ± 140.0	0.180
BMI (kg/m²)	26.4 ± 4.2	25.5 ± 3.0	0.520

DISCUSSION

The complex neonatal health is largely determined by the endocrine communication between the mother and the foetus. In this research, 97 mother-infant dyads were studied to unravel the role of two key metabolic hormones, Spexin (an anti-inflammatory peptide which regulates appetite) and Ghrelin (growth-promoting hormone which regulates appetite), in birth weight and bilirubin metabolism. We have discovered intricate interactions between maternal endocrinology and neonatal outcomes, with consequences on the recognition of at-risk pregnancies as well as the coefficient of variation of Spexin levels is 27%, and this indicates that there is a considerable biological variability, which could be as a result of the difference in glucose metabolism. The levels of Ghrelin are in accordance with the studies that reported that high levels of ghrelin are associated with insulin resistance [Stefaniak, M. *et al*., 2019]. The clinically significant range of BMI (19.1 -38.5 kg / m 2) corresponds to the fact that obesity can confound peptide dynamics due to adipose tissue-derived hormones, with low birth weight (<2500g) being high (9.3 percent) in relation to the world. The level of bilirubin was distributed in a skewed right, with 11.3% of individuals having more than the clinical intervention. This is similar to the

Publisher: SARC Publisher

observation by Watchko (2018) that minor metabolic abnormalities are predisposing factors to jaundice, regardless of hemolytic aetiology. A positive correlation with birth weight is in agreement with the fact that spexin increases insulin sensitivity; every 1 ng/mL of spexin increases birth weight, which is clinically significant because the WHO attributes 100g of increments in birth weight to lower infant mortality. The bilirubin correlation was negative, which indicates that Spexin could increase the UDP-glucuronosyltransferase activity in the liver, speeding up bilirubin conjugation [Whitehead, J. P. *et al*., 2006]

The bilirubin relationship of Ghrelin indicates two functions: growth through IGF-1, slowering bowel movement, and thus enhancing bilirubin recycling in the enterohepatic circulation. The null correlation of birth weight is contrary to undernourished cohorts, thereby indicating that sufficient nutrition in our population suppressed the growth effects of ghrelin. The difference between the 430g mean of extremes of Spexin is greater than the 250g, which is the level of significant growth variation. The nutrient transfer is due to increased expression of GLUT4 in placentas of high-Spexin mothers by 2.1-fold. Ironically, the size of high-Spexin infants risked less jaundice despite being larger-than-classical is suggested by the classical birth weight predictors, such as maternal height (1/ 2 1/ 2 = +50g/cm). The downward curve in ghrelin could be receptor downregulation in obesity, a phenomenon which has been linked in 68% of high-BMI mothers in our cohort. The model was dominated by gestational age (195g/week), although Spexin did not become insignificant after adjustment, implying that an increase in Spexin (1-SD=0.6 ng/mL) drops the bilirubin by 1.1mg/dl -enough to spare phototherapy in 32% of borderline cases.

In preterm infants, the effect of ghrelin was increased (interaction p=0.017), which means that they are vulnerable in their development. The combination of them accounts 42% of the bilirubin variance (R 2 = 0.42), which is higher compared to the risks of smoking (OR = 2.1) or hypertension (OR = 2.9). It is noteworthy that all of the LBW infants had Spexin less than 2.0 ng/mL- indicating a possible diagnostic cutoff. The ghrelin risk was, but non-significant. The complex interaction of the maternal hormone concentration, especially spexin and ghrelin, with the outcomes in babies is a nascent field of perinatal research that can provide vital information about fetal development and early neonatal acclimatization. Spexin, a newly discovered neuropeptide that may be considered a correlate of leptin, has elicited interest in its regulatory property of energy homeostasis, glucose metabolism, and inflammation; processes that are essentially associated with fetus growth. High levels of spexin in maternal circulation in pregnancy can balance the transfer of nutrients to the placenta and adiposity in the fetus, potentially affecting birth weight as an outcome of the intra-uterine growth pathway [Yildiz, B. O. *et al*., 2004; Dıez, J. J., & Iglesias, P. *et al*., 2003].

On the other hand, ghrelin, the so-called hunger hormone, displays orexigenic actions that trigger the release of growth hormone and increases the appetite; its maternal surges, which are frequently increased during gestational states of insulin resistance or obesity, might overcome the inhibitory action of spexin, thus increasing neonatal birth weights by increasing fetal energy accretion as well as However, the more conflicting profile of ghrelin is that it enhances the gastrointestinal motility and gut barrier integrity i.e. allowing meconium passage and bilirubin clearance but hypersecretion in maternal obesity cohorts has been found to contribute to fetal hypoxia and hepatic immaturity, further worsening unconjugated hyperbilirubinemia [Shehzad, A. *et al*., 2012; Weyer, C. *et al*., 2001; Barker, D. J. 2007]. Cohort studies show that the observed non-random relationships between maternal and neonatal altered ghrelinriteratios lead to increased jaundice duration of phototherapeutic duration in neonates. Odds ratios of severe hyperbilirubinemia increased by 1.5-2.0-fold in LBW babies, which overloads neonatal intensive care units furthermore These results come together to make a convincing clinical translation, where maternal spexin and ghrelin antenatal profiling may be used to stratify high-risk pregnancies to customized interventions, including nutritional modulation or peptide analogy to maximize the neonatal anthropometrics and bilirubin homeostasis and The urgency is enhanced by epidemiological variances such as ethnic predispositions in Middle Eastern populations where the prevalence of gestational diabetes meets the dysregulation of hormones through longitudinal biomarker monitoring. Shortcomings still exist, such as the cross-sectional nature of extant studies that confound maternal adiposity with actual endocrine effects, and variability of assays used in spexin detection, the future research should focus on Mendelian randomization and multi-omics combination in

Publisher: SARC Publisher

order to separate genetic confounding factors where Finally, the clarification of nexus spexin-ghrelin does not only optimize predictive models of the variation in birth weights and bilirubin but also forecasts birth weight precision models, reducing the adverse effects of childhood obesity and cholestatic conditions in the long term [Sysyn, G. D. 2004; Moon, J. H., & Jang, H. C. 2022; Kc, K. *et al*., 2015]

CONCLUSION

This research highlights the very significant influence of maternal spexin and ghrelin levels on neonatal parameters, mainly birth weight and neonatal bilirubin also According to the results, changes in these hormone levels during pregnancy may have a considerable association with the health of children at birth whereas The data showed that in mothers who had higher spexin levels, babies were born to a healthy weight, whereas ghrelin accounted for a complicated interaction which might be related to the metabolic era of neonatal bilirubin.

REFERENCES

1. Kitamura, S., Yokota, I., Hosoda, H., Kotani, Y., Matsuda, J., Naito, E., ... & Kuroda, Y. "Ghrelin concentration in cord and neonatal blood: relation to fetal growth and energy balance." *The Journal of Clinical Endocrinology & Metabolism* 88.11 (2003): 5473-5477.
2. Kojima, M., Hosoda, H., Date, Y., Nakazato, M., Matsuo, H., & Kangawa, K. "Ghrelin is a growth-hormone-releasing acylated peptide from stomach." *Nature* 402.6762 (1999): 656-660.
3. Nakazato, M., Murakami, N., Date, Y., Kojima, M., Matsuo, H., Kangawa, K., & Matsukura, S. "A role for ghrelin in the central regulation of feeding." *Nature* 409.6817 (2001): 194-198.
4. Wren, A. M., Seal, L. J., Cohen, M. A., Brynes, A. E., Frost, G. S., Murphy, K. G., ... & Bloom, S. R. "Ghrelin enhances appetite and increases food intake in humans." *J clin Endocrinol metab* 86.12 (2001): 5992.
5. Farquhar, J., Heiman, M., Wong, A. C., Wach, R., Chessex, P., & Chanoine, J. P. "Elevated umbilical cord ghrelin concentrations in small for gestational age neonates." *The Journal of Clinical Endocrinology & Metabolism* 88.9 (2003): 4324-4327.
6. James, R. J. A., Drewett, R. F., & Cheetham, T. D. "Low cord ghrelin levels in term infants are associated with slow weight gain over the first 3 months of life." *The Journal of Clinical Endocrinology & Metabolism* 89.8 (2004): 3847-3850.
7. Adibi, J. J., Xun, X., Zhao, Y., Yin, Q., LeWinn, K., Bush, N. R., ... & Koistinen, H. "Second-trimester placental and thyroid hormones are associated with cognitive development from ages 1 to 3 years." *Journal of the Endocrine Society* 5.5 (2021): bvab027.
8. Adibi, J. J., Layden, A. J., Yin, Q., Xun, X., Peddada, S., & Birru, R. "A toolkit for the application of placental-fetal molecular biomarkers in epidemiologic studies of the fetal origins of chronic disease." *Current epidemiology reports* 8.1 (2021): 20-31.
9. Perez Garrido, N. I., Pujana, M., Berger, M., Ramirez, P., Guercio, G. V., Belgorosky, A., & Marino, R. "Growth hormone receptor gene polymorphism. Spontaneous catch up growth in small for gestational age patients." (2021).
10. Holt, R. I. "Fetal programming of the growth hormone–insulin-like growth factor axis." *Trends in Endocrinology & Metabolism* 13.9 (2002): 392-397.
11. Gohlke, B. C., Huber, A., Hecher, K., Fimmers, R., Bartmann, P., & Roth, C. L. "Fetal insulin-like growth factor (IGF)-I, IGF-II, and ghrelin in association with birth weight and postnatal growth in monozygotic twins with discordant growth." *The Journal of Clinical Endocrinology & Metabolism* 90.4 (2005): 2270-2274.
12. Stawerska, R., Szałapska, M., Hilczer, M., & Lewiński, A. "Ghrelin, insulin-like growth factor I and adipocytokines concentrations in born small for gestational age prepubertal children after the catch-up growth." *Journal of Pediatric Endocrinology and Metabolism* 29.8 (2016): 939-945.
13. Pietrzak, M., Yngve, A., Hamilton, J. P., Asratian, A., Gauffin, E., Löfberg, A., ... & Boehme, R. "Ghrelin decreases sensitivity to negative feedback and increases prediction-error related caudate activity in humans, a randomized controlled trial." *Neuropsychopharmacology* 49.6 (2024): 1042-1049.
14. Wang, Y., Guo, S., Zhuang, Y., Yun, Y., Xu, P., He, X., ... & Jiang, Y. "Molecular recognition of an acyl-peptide hormone and activation of ghrelin receptor." *Nature Communications* 12.1 (2021): 5064.
15. Lang, R., Gundlach, A. L., Holmes, F. E., Hobson, S. A., Wynick, D., Hoekfelt, T., &

Publisher: SARC Publisher

Kofler, B. "Physiology, signaling, and pharmacology of galanin peptides and receptors: three decades of emerging diversity." *Pharmacological reviews* 67.1 (2015): 118-175.
16. Kumar, S., Hossain, J., Nader, N., Aguirre, R., Sriram, S., & Balagopal, P. B. "Decreased circulating levels of spexin in obese children." *The Journal of Clinical Endocrinology & Metabolism* 101.7 (2016): 2931-2936.
17. Sanli, S., Bulbul, A., & Ucar, A. "The effect of umbilical cord blood spexin, free 25 (OH) vitamin D3 and adipocytokine levels on intrauterine growth and anthropometric measurements in newborns." *Cytokine* 144 (2021): 155578.
18. Ojha, S., Robinson, L., Yazdani, M., Symonds, M. E., & Budge, H. "Brown adipose tissue genes in pericardial adipose tissue of newborn sheep are downregulated by maternal nutrient restriction in late gestation." *Pediatric research* 74.3 (2013): 246-251.
19. Ong, K. K., & Dunger, D. B. "Birth weight, infant growth and insulin resistance." *European journal of endocrinology* 151.Supplement_3 (2004): U131-U139.
20. Savvidou, M. D., Sotiriadis, A., Kaihura, C., Nicolaides, K. H., & Sattar, N. "Circulating levels of adiponectin and leptin at 23–25 weeks of pregnancy in women with impaired placentation and in those with established fetal growth restriction." *Clinical Science* 115.7 (2008): 219-224.
21. Stefaniak, M., Dmoch-Gajzlerska, E., Mazurkiewicz, B., & Gajzlerska-Majewska, W. "Maternal serum and cord blood leptin concentrations at delivery." *PLoS One* 14.11 (2019): e0224863.
22. Whitehead, J. P., Richards, A. A., Hickman, I. J., Macdonald, G. A., & Prins, J. "Adiponectin–a key adipokine in the metabolic syndrome." *Diabetes, obesity and metabolism* 8.3 (2006): 264-280.
23. Yildiz, B. O., Suchard, M. A., Wong, M. L., McCann, S. M., & Licinio, J. "Alterations in the dynamics of circulating ghrelin, adiponectin, and leptin in human obesity." *Proceedings of the National Academy of Sciences* 101.28 (2004): 10434-10439.
24. Dıez, J. J., & Iglesias, P. "The role of the novel adipocyte-derived hormone adiponectin in human disease." *Eur J Endocrinol* 148.3 (2003): 293-300.
25. Shehzad, A., Iqbal, W., Shehzad, O., & Lee, Y. S. "Adiponectin: regulation of its production and its role in human diseases." *Hormones* 11.1 (2012): 8-20.
26. Weyer, C., Funahashi, T., Tanaka, S., Hotta, K., Matsuzawa, Y., Pratley, R. E., & Tataranni, P. A. "Hypoadiponectinemia in obesity and type 2 diabetes: close association with insulin resistance and hyperinsulinemia." *The Journal of Clinical Endocrinology & Metabolism* 86.5 (2001): 1930-1935.
27. Barker, D. J. "The origins of the developmental origins theory." *Journal of internal medicine* 261.5 (2007): 412-417.
28. Sysyn, G. D. "Abnormal fetal growth: intrauterine growth retardation, small for gestational age, large for gestational age." *Pediatric Clinics* 51.3 (2004): 639-654.
29. Moon, J. H., & Jang, H. C. "Gestational diabetes mellitus: diagnostic approaches and maternal-offspring complications." *Diabetes & metabolism journal* 46.1 (2022): 3-14.
30. Kc, K., Shakya, S., & Zhang, H. "Gestational diabetes mellitus and macrosomia: a literature review." *Annals of Nutrition and Metabolism* 66.Suppl. 2 (2015): 14-20.

Source of support: Nil; **Conflict of interest:** Nil.

Cite this article as:
Al-Akraa, M. S. M. & Al-Zubaidy, A. A. M. H. "Examining the Effects of Maternal Spexin and Ghrelin Levels on Neonatal Outcomes, with Emphasis on Birth Weight and Bilirubin Levels." *Sarcouncil Journal of Biomedical Sciences* 3.6 (2024): pp 19-25.

Publisher: SARC Publisher