Salud, Ciencia y Tecnología - Serie de Conferencias. 2025; 4:1785
doi: 10.56294/sctconf20251785

ORIGINAL

Association of birth weight with blood pressure and renal function variables in children aged 3 to 6 years

Asociación del peso al nacer con la presión arterial y variables funcionales renales en niños de 3 a 6 años

Carlos Rolando Maldonado Bernardo[1], Adina Pérez Mejías[2], Miguel Alfonso Álvarez Fornaris[1]

[1]Facultad de Ciencias Médicas Salvador Allende. La Habana, Cuba.
[2]Instituto de Ciencias Básicas y Preclínicas Victoria de Girón. La Habana, Cuba.

Cite as: Maldonado Bernardo CR, Pérez Mejías A, Álvarez Fornaris MA. Association of birth weight with blood pressure and renal function variables in children aged 3 to 6 years. Salud, Ciencia y Tecnología - Serie de Conferencias. 2025; 4:1785. https://doi.org/10.56294/sctconf20251785

Submitted: 27-10-2024 **Revised:** 12-01-2025 **Accepted:** 10-04-2025 **Published:** 11-04-2025

Editor: Prof. Dr. William Castillo-González

Corresponding author: Carlos Rolando Maldonado Bernardo

ABSTRACT

Introduction: intrauterine growth retardation (IUGR) has recently been related to an increase in blood pressure figures in different countries.
Objective: the objective of this research was to evaluate the relationship between blood pressure and kidney function with birth weight in children aged 3 to 6 years.
Method: thirty-two healthy, normotensive children with a history of low birth weight due to IUGR or normal birth weight, aged between 3 and 6 years, were studied in the Marianao municipality of Havana. Their nutritional status was determined based on their body surface area, using the Haycock formula expressed in square meters. Arterial, systolic and diastolic pressures were measured using the Riva Rocci-Korotkoff method, calculating the average. Glomerular filtration rate (GFR) was determined using the Schwartz 2 formula (GFR WS) and Pottel for serum creatinine and for serum Cystatin C using Pottel. The renal and blood pressure variables were adjusted to body surface area and analyzed using Pearson's correlation. Ethical standards for research on humans were respected.
Results: systolic, diastolic and mean arterial pressures, as well as GFR, were inversely correlated with birth weight. Children with a history of low birth weight due to IUGR presented higher blood pressure values, although not pathological with respect to their peers; the IFG values in this group of children were higher with respect to their peers, calculated both by creatinine through the Pottel method and serum Cystatin C.
Conclusion: there is a tendency for higher blood pressure values in children with low birth weight due to IUGR. The correlation between IFG and birth weight supports theories about the influence of hyperfiltration on high blood pressure, so we suggest more extensive studies of the variables studied, as well as the use of the Pottel formula for its study.

Keywords: Delayed Intrauterine Growth; Glomerular Filtration Intensity.

RESUMEN

Introducción: el crecimiento intrauterino retardado (CIUR) se ha relacionado recientemente con un incremento de las cifras de presión arterial en diferentes países.
Objetivo: el objetivo de esta investigación fue evaluar la relación existente entre presión arterial y función renal con el peso al nacer en niños de 3 a 6 años de edad.
Método: fueron estudiados 32 niños sanos y normotensos; con antecedentes de bajo peso al nacer por CIUR o peso normal al nacer, entre 3 y 6 años de edad del municipio Marianao en La Habana. Se determinó su

evaluación nutricional, a partir del área de superficie corporal, a través de la fórmula de Haycock expresado en metros cuadrados. Las presiones arteriales, sistólicas y diastólicas se midieron por el método de Riva Rocci-Korotkoff, calculándose la media. Se determinó la intensidad de filtración glomerular (IFG) mediante las fórmulas de Schwartz 2 (IFG WS) y Pottel para la creatinina sérica y para la Cistatina C sérica mediante Pottel. Las variables renales y de presión arterial fueron ajustadas al área de superficie corporal y analizadas mediante la correlación de Pearson. Se respetaron las normas éticas para la investigación en humanos.
Resultados: las presiones arteriales sistólicas, diastólicas y medias; así como la IFG se correlacionaron inversamente con el peso al nacer. Los niños con antecedentes de bajo peso al nacer por CIUR presentaron valores de presión arterial superiores, aunque no patológicos con respecto a sus semejantes; los valores de IFG en este grupo de niños fueron superiores con respecto a sus pares, calculados tanto por la creatinina a través del método de Pottel como la Cistatina C sérica.
Conclusión: existe una tendencia a mayores valores de presión arterial en los niños con bajo peso al nacer por CIUR. La correlación entre la IFG y el peso al nacer, apoya las teorías sobre la influencia de la hiperfiltración en la hipertensión arterial por lo que sugerimos estudios más amplios de las variables estudiadas, así como el uso de la fórmula de Pottel para su estudio.

Palabras clave: Crecimiento Intrauterino Retardado; Intensidad de Filtración Glomerular.

INTRODUCTION

High blood pressure (HBP) is an elevation of systemic blood pressure above 140-90 mmHg in adults.[1] In children, it is diagnosed when systolic and/or diastolic blood pressure is equal to or greater than the 90th percentile for age, gender, and height.[2]

A personal history of low birth weight due to intrauterine growth restriction (IUGR), also known as "fetal or intrauterine growth restriction, IUGR," is one of the new predictive factors for developing HTN.[3,4,5,6] Brenner and other researchers have shown that subjects with low birth weight due to IUGR and congenital nephron deficiency (decreased functional glomeruli) have a smaller glomerular filtration area. This triggers compensatory hyperfiltration mechanisms in the remaining nephrons, leading to glomerular hypofunction, alterations in the tubular-glomerular balance, decreased sodium regulation capacity, and, ultimately, the onset of HTN.[7,8,9,10,11]

In recent years, the association between low birth weight and blood pressure levels has been confirmed in different countries. However, it has been suggested that even if there is a congenital nephron deficit, the development of hypertension will depend on the individual's relationship with the environment, living conditions, and other familial pathological factors.[12,13,14,15,16,17,18,19]

This study aimed to evaluate the relationship between blood pressure and renal function with birth weight in children aged 3 to 6 years.

METHOD

A longitudinal, cross-sectional, correlational, non-experimental cohort study was conducted over a period of one year in the municipality of Marianao, in Havana, among 32 healthy, normotensive children between the ages of 3 and 6 with a personal history of low birth weight by IUGR or normal birth weight. All children with a history of preterm birth, twin birth, or any chronic disease were excluded.

The nutritional status of all subjects was assessed using body surface area calculated using the Haycock formula expressed in m2, given by:

x=0,024265 x weight (kg)0,5378 x height (cm)0,3964 and was used as a measure to homogenize variables such as blood pressure and glomerular filtration rate (GFR).[20]

Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were determined using the Riva-Rocci-Korotkoff method, expressed in mmHg, and mean arterial pressure (MAP) was calculated as MAP: DBP+(SBP-DBP)/3. Systolic, diastolic, and/or mean blood pressure values below the 90th percentile for age, sex, and height were considered normal.[21]

Glomerular filtration rate was determined by serum creatinine using two formulas: Schwartz 2 (GFR WS) GFR = K.L / Crp, where K is a constant dependent on muscle mass, with an average value of 0,55, L is height in centimeters, and Crp is serum creatinine. GFR was considered normal above 90 ml/min/1,73 m2.[22]

The Pottel formula (Pottel GFR) was also used: GFR = 107. 3 (ScratC /QScreatC) where ScratC is the serum creatinine concentration and QScreatC is a serum creatinine constant according to age: 3 years (27 mmol/L), 4 years (30 mmol/L), 5 years (34 mmol/L), and 6 years (36 mmol/L). GFR was considered normal above 90 ml/min/1,73 m2.[23]

The glomerular filtration rate was also determined by serum cystatin C (Pottel GFR) using the Pottel formula GFR = 107,3 (ScysC/QScysC), where ScysC is the serum concentration of cystatin C, QScysC is a serum cystatin

https://doi.org/10.56294/sctconf20251785

C constant with a value of 0,82 mg/L. GFR was considered normal above 90 ml/min/1,73 m².[23]

Means were used as summary measures and adjusted for body surface area in the SBP, DBP, and MAP variables. They were analyzed using Pearson's correlation for statistical significance equal to or less than 0,05.[20]

All data were stored in an individual file, including the informed consent of the minors' parents, by the Helsinki Protocol and ethical standards for research involving human subjects.[24]

RESULTS

This study included a total of 32 children, 71 % of whom were female; the most common age was 4 years, and 75 % of the sample reported a family history of high blood pressure.

Table 1. Demographic description of the sample, according to sex, age, and family history

General information		Number	%
Sex	F	23	71,875
	M	9	28,125
	Total	32	100
Age	3 years	7	21,875
	4 years	12	37,5
	5 years	6	18,75
	6 years	7	21,875
	Total	32	100
Family History of Hypertension		24	75

Table 1 shows that the percentage of females was higher than that of males. The largest number of individuals were 4 years old, and a family history of hypertension was present in 75 % of the sample.

Table 2. Relationship between birth weight and body surface area

Correlations		Birth weight	Surface
Birth weight	Pearson correlation	1	,544**
	Sig. (bilateral)		,001
	N	32	32

**. The correlation is significant at the 0,01 level (two-tailed).

Table 2 shows that there is a direct and significant correlation between birth weight and body surface area. The body surface area of children was greater for the same age as birth weight increased, a statistically significant result.

Table 3. Relationship between birth weight and systolic (SBP), diastolic (DBP), and mean (MABP) blood pressure, adjusted for body surface area

Correlations		Birth weight	PAMC	PADC	PASC
Birth weight	Pearson correlation	1	-,315	-,277	-,238
	Sig. (bilateral)		,079	,125	,190
	N	32	32	32	32

Table 3 shows that there is an inverse correlation between birth weight and blood pressure-related variables, without reaching pathological levels.

Table 4. Relationship between birth weight and serum creatinine and glomerular filtration rate based on serum creatinine, according to the Schwartz II (GFR.WS) and Pottel (GFR Pottel) formulas

Correlations		Birth weight	Creatinine	IFG.WS	IFG. POTTEL
Birth weight	Pearson correlation	1	,054	,325	-,228
	Sig. (bilateral)		,768	,070	,209
	N	32	32	32	32

The glomerular filtration rate based on serum creatinine was calculated using two methods. Using the Schwartz II method[22], we obtained a direct correlation between birth weight and this variable, such that children with higher birth weights had higher GFR; however, using the Pottel method[23], we found an inverse correlation between these variables, which contradicts the previous result.

https://doi.org/10.56294/sctconf20251785

Table 5. Relationship between birth weight and serum cystatin C and glomerular filtration rate by serum cystatin C, according to the Pottel formula

Correlations		Birth weight	Cystatin C	IFG. Pottel
Birth weight	Pearson correlation	1	,116	-,106
	Sig. (bilateral)		,527	,564
	N	32	32	32

This table shows that there is a direct correlation between birth weight and serum cystatin C values. It was observed that IFG by this method had an inverse correlation with birth weight.

DISCUSSION

In research conducted with children aged 3 to 6 years with low birth weight according to CIUR or normal birth weight, we found the same association as Hoy[25] and Gamboa et al.[26] in studies conducted in Australian and Colombian populations, respectively, showing similar behavior in such different and distant environments.

The trend in adjusted PAD, PAS, and PAM values is entirely consistent with Pérez[27], Barker[28], Cosmi[29], Law[30], Lenfant[31], and Keijzer[32], who show in their studies that there is a direct relationship between low birth weight and increased blood pressure values regardless of body surface area and age at the time of blood pressure measurement, without reaching pathological levels in our case.

Serum creatinine is a substance whose concentration depends on the balance between its majority production by skeletal muscle and its renal excretion;[33] as the body surface area is smaller in these children, their muscle mass is lower, and therefore, their creatinine production is lower, which is consistent with the findings of other authors[3] and confirmed in our study.

Regarding the contradictory results for IFG values, the discrepancy found between the Schwartz II and Pottel methods can be explained by the fact that the former uses a constant dependent on the muscle mass and height of the infant. Therefore, children with a larger body surface area had higher IFG values. Secondly, the Pottel equation adjusts for serum creatinine concentrations according to age and sex and not according to body surface area. The bias is. Therefore, lower and higher IFG values are found as birth weight decreases, making it a more suitable equation for calculating IFG in children, adolescents, and adults.[23]

Serum cystatin C is a compound constantly formed by all nucleated cells, freely filtered by the glomerulus, and almost entirely reabsorbed in the proximal convoluted tubule, where it is almost wholly catabolized without being secreted. Its concentration does not vary with age, sex, or muscle mass, making it a better marker of IFG than serum creatinine.[34] Cystatin C values in the study sample correlated directly with birth weight; however, IFG in these children showed an inverse correlation between birth weight and glomerular filtration, meaning that these children had higher IFG values than their peers who were born with higher birth weight. This result is consistent with the measurement made using the Pottel method with serum creatinine.[35] Although our results are not statistically significant, they are consistent with those of several authors, including Vehaskari[36], Brenner[37], Bagby[38], and Bakker[39], who found that in children with low birth weight due to IUGR, compensatory glomerular hyperfiltration of isolated nephrons is observed due to their lower number at birth compared to healthy children, regardless of age.

Based on the results obtained, in children with low birth weight due to IUGR, there is an early state of compensatory glomerular hyperfiltration in response to the congenital deficit in the number of nephrons present at the early age of 3 years. This state of glomerular hyperfiltration should lead to activation of the glomerular-tubular balance mechanism and, therefore, to an increase in reabsorption at the proximal tubule level, which undergoes some degree of compensatory hypertrophy over time, considering that more than two-thirds of the filtered sodium load and volume are reabsorbed in this segment.[40]

A chronic increase in reabsorptive activity should increase plasma sodium concentrations and blood volume; this effect, if maintained over time, could produce a glomerular-tubular balance at a higher filtration-reabsorption level, as reported by other researchers,[3] which affects the regulatory systems that control the pressure-dependent diuresis-natriuresis mechanism at the tubular level and, therefore, impairs the renal ability to control blood pressure and causes increases in these values, such as those found in this study.

CONCLUSIONS

In children aged 3 to 6 years with a history of low birth weight due to IUGR, it has been confirmed that the lower the birth weight, the higher the blood pressure values for the same age, without reaching pathological levels.

The discrepancies between GFR values based on serum creatinine and birth weight were calculated using two different formulas (Schwartz II and Pottel); the glomerular filtration rate based on cystatin C was calculated using the Pottel formula, which obtained a result similar to its equivalent for creatinine, are explainable and recommend the latter formula for further studies. The GFR results confirm the hyperfiltration theory as the

https://doi.org/10.56294/sctconf20251785

cause of high blood pressure in the sample studied.

REFERENCES

1. McEvoy JW, McCarthy CP, Bruno RM, et.al 2024 ESC Guideline for the management of elevated blood pressure and hypertension of the European Society of Cardiology and endorsed by the European Society of Endocrinology and the European Stroke Organization. European Heart Journal. October 2024;45(38):3912-4018. Available from: https://www.academic.oup.com/eurheartj/article/45/38/3912/7741010.

2. Rosas-Peralta. M, Medina-Concebida. LE, Borrayo-Sánchez. G, et.al. Hipertensión arterial sistémica en el niño y adolescente. Rev Med Inst Mex Seguro Soc. 2016;54(1):52-66. Available from: https://www.redalyc.org/articulo.oa?id=457746536003.

3. Santiesteban IE, Mesa. LB, Ramos. AP, et. al. Desbalance glomérulo-tubular en niños y adolescentes con antecedentes de bajo peso al nacer. Revista Habanera de Ciencias Médicas. 2016;15(3):484-93. Available from: http://www.revhabanera.sld.cu/index.php/rhab/article/view/1067/1043.

4. Schreuder MF, Nauta J. Prenatal programming of nephron number and blood pressure. Kidney Int. 2007;72(3):265-8. Available from: http://www.ncbi.nlm.nih.gov/pubmed/17495859.

5. Ritz E, Amann K, Koleganova N, Benz K. Prenatal programming-effects on blood pressure and renal function. Nature reviews Nephrology. 2011;7(3):137-44. Available from: http://www.ncbi.nlm.nih.gov/pubmed/21283139.

6. Yiu V, Buka S, Zurakowski D, McCormick M, Brenner B, Jabs K. Relationship between birthweight and blood pressure in childhood. American journal of kidney diseases : the official journal of the National Kidney Foundation. 1999;33(2):253-60. Available from: http://www.ncbi.nlm.nih.gov/pubmed/10023635.

7. Chong. E, Yosypiv. I. Development programming of hypertension and kidney disease. International Journal of Nephrology. 2012;2012:15. Available from: https://www.hindawi.com/journals/ijn/2012/760580/.

8. Goldblatt H, Lynch J. Studies on expermiental hypertension: the production of persistent elevation of systolic blood pressure by means of renal ischemia. J Exp Med. 1934;59(3):347-79. Available from: https://www.pubmed.ncbi.nlm.nih.gov/19870251/.

9. Hemachandra AH, Klebanoff MA, Furth SL. Racial disparities in the association between birth weight in the term infant and blood pressure at age 7 years: results from the collaborative perinatal project. Journal of the American Society of Nephrology : JASN. 2006;17(9):2576-81. Available from: http://www.ncbi.nlm.nih.gov/pubmed/16870709.

10. Mejías. AP, Fox. MOB, Ramos. AP, et. al. Desbalance glomérulo-tubular en la fisiopatología de la hipertensión arterial asociada al bajo peso al nacer. Revista Habanera de Ciencias Médicas. 2011;10(2):224-32. Available from: http://www.revhabanera.sld.cu/index.php/rhab/article/view/1830.

11. Zanardo V, Bertin M, Luca Fd, Zaninotto M. Albuminuria and sodiuria in IUGR children. The Journal of Maternal-Fetal & Neonatal Medicine. 2015;28(3):362-5. Available from: https://www.pubmed.ncbi.nlm.nih.gov/24853042/.

12. Martinez-Aguayo. A, Aglony. M, Bancalari. R, Avalos. C, et.al. Birth weight is inversely associated with blood pressure and serum aldosterone and cortisol levels in children. . Clin Endocrinol (Oxf) 2012;76(5):713-8. Available from: https://www.pubmed.ncbi.nlm.nih.gov/22145676/.

13. Strufaldi. M, Silva. E, Franco. M, Puccini. R. Blood pressure levels in childhood: probing the relative importance of birth weight and current size. . Eur J Pediatr. 2009;168(5):619-24. Available from: https://www.pubmed.ncbi.nlm.nih.gov/18830709/.

14. Pereira. J, Rondó. P, Lemos. J, Souza. JPd, Dias. R. The influence of birthweight on arterial blood pressure of children. . Clin Nutr 2010;29(3):337-40. Available from: https://www.sciencedirect.com/science/article/abs/pii/S0261561410000105/.

15. Chen. X, Zhang. Z, George. L, et.al. Birth measurements, family history, and environmental factors associated with later-life hypertensive status. Am J Hypertens 2012;25(4):464–71. Available from: https://www.pubmed.ncbi.nlm.nih.gov/pmc/articles/PMC3309157/.

16. Eriksson. J, Forsén. T, Tuomilehto. J, Osmond. C, Barker. D. Early growth and coronary heart disease in later life: longitudinal study. . BMJ Case Rep. 2001;322(7292):949–53. . Available from: https://www.pubmed.ncbi.nlm.nih.gov/11312225/.

17. Falkner. B, Hulman. S, Kushner. H. Effect of birth weight on blood pressure and body size in early adolescence. Hypertension. 2004;43(2):203-7. Available from: https://www.pubmed.ncbi.nlm.nih.gov/14676220/.

18. Eriksson. J. Early growth and coronary heart disease and type 2 diabetes: findings from the Helsinki Birth Cohort Study (HBCS). Am J Clin Nutr 2011;94(6):1799S–802S. Available from: https://www.pubmed.ncbi.nlm.nih.gov/21613556/.

19. Paixão. AD, Alexander. BT. How the Kidney Is Impacted by the Perinatal Maternal Environment to Develop Hypertension. Biol Reprod. 2013;89(6):144. Available from: https://www.pubmed.ncbi.nlm.nih.gov/24227755/.

20. Moreno VM, Cabrerizo MM, Gandoy JBG, et.al. Evalúan parámetros antropométricos como indicadores de la distribución de la grasa corporal. Sociedad Iberoamericana de Información Científica. 2007. Available from: https://www.siicsalud.com/des/expertoimpreso.php/85727./.

21. Hipertensión arterial. Guía para la prevención, diagnóstico y tratamiento/ Comisión Nacional Técnica Asesora del Programa de Hipertensión arterial. La Habana: Editorial Ciencias Médicas; 2008; 36-38.

22. Schwartz. G, Haycock. G, Edelman. C, Spitzer. A. A simple estimate of glomerular filtration rate in children derived form body lenght and plasma creatinine. Pediatric Nephrology. 1976;58:259-63. Available from: https://www.pediatrics.aappublications,org/content/58/2/259.long.

23. Pottel H, Pierre D, et.al. Estimated glomerular filtration rate for the full age spectrum from serum creatinine and cystatin C. Nephrol Dial Transplantation. 2017;32(3):405-7. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC58337496/.

24. Carrera. ALM. Importancia ético-científica de los protocolos sobre las investigaciones en humanos. Medleg Costa Rica. 2003;20(1). Available from: https://www.scielo.sa.cr/scielo.php?script=sci_arttext&pid=S1409-00152003000100005

25. Hoy W, Rees M, Kile E, Mathews JD, Wang Z. A new dimension to the Barker hypothesis: Low birthweight and susceptibility to renal disease. Kidney International. septiembre 1999;56(3):1072-7. Available from: https://www.pubmed.ncbi.nlm.nih.gov/10469376/.

26. Delgado. EG, Díaz. YR, Gómez. YG. Asociación entre el peso al nacer y factores de riesgo cardiometabólicos en niños de Bucaramanga, Colombia. Nutrición Hospitalaria. 2017;34(5). Available from: https://www.scielo.isciiii.es/scielo.php?script=sci_arttext&pid=S0212-16112017000500013/.

27. Pérez. M, Valdés. R, Tasis. M. Bajo peso al nacer y su relación con la hipertensión arterial en adolescentes y jóvenes. Revista Cubana de Medicina. 2004;43(5-6). Available from: https://www.scielo.sld.cu/scielo.php?script=sci_arttext&pid=S0034-75232004000500002.

28. Barker DJP, Hales CN, Fall CHD, Osmond C, Phipps C, Clark PMS. Type 2 diabetes mellitus, hypertension and hyperlipidemia (syndrome X): Relation to reduced fetal growth. Diabetologia. 1993;36:62-7. Available from: https://www.link.springer.com/article/10.1007/BF00399095.

29. Cosmi E, Fanelli T, Visentin S, Trevisanuto D, Zanardo V. Consequences in infants that were intrauterine growth restricted. Journal of pregnancy. 2011;2011:364381. Available from: http://www.ncbi.nlm.nih.gov/pubmed/21547088.

30. Law. C, Shiell. A. Is blood pressure inversely related to birth weight? The strength of evidence from a systematic review of the literature. Journal of Hypertension. 1996;14(8):935-41. Available from: https://www.

journals.lww.com/jhypertension/Abstract/1996/08000/Is_blood_pressure_inversely_related_to_birth.2.aspx.

31. Lenfant C. Low birth weight and blood pressure. Metabolism: Clinical and Experimental. 2008;57(2):S32-S5. Available from: https://www.metabolismjournal.com/article/S0026-0495(08)00253-9/pdf.

32. Keijzer-Venn. M, Finken. M. Is blood pressure increased 19 years after intrauterine growth restriction and preterm birth? A prospective follow-up study in the Netherlands. Pediatrics. 2005;116(3):725-31. Available from: https://www.pubmed.ncbi.nlm.nih.gov/16140714/.

33. Ku. E, Xie. D, Shlipak. M, et.al. Change in Measured GFR Versus eGFR and CKD Outcomes. Journal of the American Society of Nephrology. 2016;27(7):2196-204. Available from: https://www.pubmed.ncbi.nlm.nih.gov/26604213/.

34. Barbati A, Cappuccini B, Aisa MC, Grasselli C, Zamarra M, Bini V, et al. Increased Urinary Cystatin-C Levels Correlate with Reduced Renal Volumes in Neonates with Intrauterine Growth Restriction. Neonatology. 2016;109(2):154-60. Available from: http://www.ncbi.nlm.nih.gov/pubmed/26756983.

35. Bukabau J, Yayo E, et. al. Perfomance of creatinine or cystacin C based equations to estimate glomerular filtration rate in sub-Saharan African populations. . Kidney Int. 2019;95:1181-9. Available from: https://www.kidney-international.org/article/S0085-2538(19)30042-0/fulltext.

36. Vehaskari V, Woods. L. Prenatal programming of hypertension: lessons from experimental models. . J Am Soc Nephrol. 2005;16:254-6. Available from: https://www.pubmed.ncbi.nlm.nih.gov/16049066/.

37. Brenner B, Chertow. G. Congenital oligonephropathy and the etiology of adult hypertension and progressive renal injury. American journal of kidney diseases : the official journal of the National Kidney Foundation. 1994;3:171-5. Available from: https://www.pubmed.ncbi.nlm.nih.gov/8311070/.

38. Bagby. S. Maternal nutrition, low nephron number, and hypertension in later life: pathways of nutritional programming. J Nutr. 2007;137:1066-72. Available from: https://www.pubmed.ncbi.nlm.nih.gov/17374679/.

39. Bakker H, Gaillard R, Hofman A, Reiss IK, Steegers EA, Jaddoe VW. Fetal first trimester growth is not associated with kidney outcomes in childhood. Pediatric nephrology. 2017;32(4):651-8. Available from: http://www.ncbi.nlm.nih.gov/pubmed/27796619.

40. Skorecki K, Chertow GM, Marsden PA, Taal MW, Yu ASL, Wasser WG. BRENNER & RECTOR'S. THE KIDNEY. Karl Skorecki GMC, Philip A. Marsden, Maarten W. Taal ASLY, editors: Elsevier; 2016.

FUNDING

The authors did not receive funding for the development of this research.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

AUTHOR CONTRIBUTION

Conceptualization: Carlos Rolando Maldonado Bernardo, Miguel Alfonso Álvarez Fornaris, Adina Pérez Mejías.

Research: Carlos Rolando Maldonado Bernardo, Miguel Alfonso Álvarez Fornaris, Adina Pérez Mejías.

Drafting of the original manuscript: Carlos Rolando Maldonado Bernardo, Miguel Alfonso Álvarez Fornaris, Adina Pérez Mejías.

Revision and editing: Carlos Rolando Maldonado Bernardo, Miguel Alfonso Álvarez Fornaris, Adina Pérez Mejías.