Impact of pre-term labor on infant kindney

Srujitha Kandregula1*, Akhila2 and Satya Rao3

¹Department of Pharmaceutics, Vignan Institute of Pharmaceutical Technology, Vishakapatnam, Andhrapradesh ²Department of Biotechnology, Gonna Institute of Information Technology, Vishakapatnam, Andhrapradesh ³Department of Molecula Biology, Gonna Institute of Information Technology, Vishakapatnam,

Andhrapradesh

Research Article

Received: 09/08/2016 Revised: 29/09/2016 Published: 08/10/2016

*For Correspondence

Department of Pharmaceutics, Vignan Institute of Pharmaceutical Technology, Vishakapatnam, Andhrapradesh.

Keywords: Nephrogenesis, Neonatal care, Kidney

E-mail: Srujikandregula@gmail.com Preterm birth (characterized as birth before 37 finished weeks of incubation), happens in roughly 10% of all births and is one of the main sources of neonatal dreariness and mortality around the world. Preterm newborn children are conceived when kidney improvement is as yet continuous, and thus can prompt renal weakness (in both the transient and long haul), and in addition serious glomerular anomalies in some preterm babies. Since the glomerular anomalies are not present in all preterm kidneys, this proposes it is not preterm birth as such that prompts the glomerular variations from the norm however may identify with variables connected with the etiology of the unexpected labor, or considers neonatal consideration. In this survey, we give a review of what is as of now known of how pre-birth and postnatal elements can possibly affect on the youthful kidneys of newborn children conceived preterm.

ABSTRACT

INTRODUCTION

Preterm birth happens in around 10% of all births and is one of the main sources of neonatal horribleness and mortality around the world. Preterm babies are conceived when their organ frameworks are youthful and henceforth, being conceived early can prompt antagonistic consequences for organ structure and capacity both in the transient and in the long haul. Preterm birth can prompt renal weakness in the neonatal period and can prompt glomerular variations from the norm in some preterm newborn children. Since the glomerular irregularities are not present in all preterm kidneys, this recommends it is not preterm birth in essence that prompts the glomerular variations from the norm however may identify with components connected with the etiology of the unexpected labor or figures neonatal consideration. To be sure, the etiology of preterm birth is multifactorial and the neonatal consideration of preterm newborn children is distinctive for all people, contingent upon their postnatal sequelae. In this audit, we give a diagram of what is at present known of how pre-birth and postnatal components can conceivably affect on the youthful kidneys of babies conceived preterm [1-10].

PRETERM LABOR

Sub side heading

Preterm birth happens in around 10% of all births and is one of the main sources of neonatal dismalness and mortality overall. Preterm birth is characterized as birth before 37 finished weeks of growth, with birth between 38-42 weeks of development considered as full term. Preterm birth can be further sub-characterized into reasonably preterm, exceptionally preterm and to a great degree preterm. Respectably preterm babies are named those conceived between 32 to 36 weeks of growth, exceptionally preterm births are those conceived somewhere around 28 and 31 weeks development, to a great degree preterm births are those conceived before28 weeks incubation. Babies conceived preceding 23 weeks more often than not don't survive. The dominant part (60-70%) of preterm babies are conceived somewhere around 34 and 36 weeks of incubation. The frequency of preterm babies conceived at 32-33 weeks growth is ~20% and ~15% are conceived at 28-31 weeks, preterm birth preceding 28 weeks is the minimum basic ^[11-20].

The worldwide number of preterm conveyances every year has been gradually expanding and right now it is around 10% of births overall. In the USA the frequency of preterm birth is 12.3%, in Europe it is 5-7%, and in Australia it is 8.2%. Be that as it may, inside these populaces some ethnic gatherings have a higher rate of preterm birth. For instance in African Americans the frequency of preterm birth is high at 17.5% and in Indigenous Australians 13.3% of all births are preterm ^[6]. Of concern, the pervasiveness of preterm birth in creating nations is high; for instance, up to 17.5% of the reported birth sin South Africa are preterm and this is prone to be significantly higher the same number of births are not recorded ^[21-25].

Survival taking after preterm birth (particularly in those conceived, to a great degree preterm) has enhanced significantly since the main presentation of neonatal concentrated consideration units (in the 1960s). With ensuing refinements in pre-birth and neonatal consideration, infants conceived as ahead of schedule as 25 weeks incubation now have a 80% shot of survival ^[8,9]. Specifically, the utilization of antenatal/neonatal corticosteroids (which quicken lung development in the infant) and surfactant treatment (which lessens alveolar surface pressure within the sight of respiratory pain disorder) have encouraged the late change in survival ^[26-30].

Preterm Birth and its Effects on Renal Function and Nephrogenesis

Renal capacity

On account of preterm babies, they are conveyed when nephrogenesis is regularly progressing. In preterm neonates glomerular filtration rate (GFR) is low during childbirth, and does not ascend as quickly as full term newborn children amid the neonatal period ^[20,21]. Not surprisingly, glomerular filtration rate has appeared to build all the more quickly following 34 weeks growth [22,23] which agrees with the planning of the fruition of nephrogenesis. Various studies have demonstrated that preterm birth can prompt a high rate of renal brokenness in the neonate and under extreme circumstances this can prompt renal disappointment ^[24,25]. The rate of renal debilitation in preterm babies is hard to unmistakably characterize given that the kidneys are extremely youthful at the season of birth. Subsequently, renal capacity is very diverse in the preterm newborn child when contrasted with the term baby and a considerable lot of these distinctions are because of adolescence as opposed to a fundamental impedance. Absolutely, both glomerular and tubular capacity are impacted by gestational age during childbirth and henceforth, it is hard to set up whether the distinctions in renal capacity in preterm newborn children contrasted with term babies are exclusively because of underdevelopment of the nephrons or the aftereffect of harm in a youthful kidney. Amid the principal week after birth, glomerular filtration rate (GFR) is altogether lower in preterm newborn children contrasted with term babies [26-28] and it is emphatically associated with gestational age during childbirth and postnatal age [31-40]. Similarly, creatinine freedom, a standout amongst the most regularly utilized markers of renal capacity, is emphatically associated with both gestational age and postnatal age [41]. What's more, preterm neonates discharge high measures of sodium in the early neonatal period contrasted with term neonates, with the fragmentary discharge of sodium contrarily corresponded with gestational age and postnatal age [42-47].

The nearness of elevated amounts of protein in the pee is characteristic of obsessive proteinuria (pee complete protein \geq 500 mg/l) and can be glomerular and/or tubular in starting point. In particular, the nearness of proteins with a high sub-atomic weight (egg whites) in the pee, is characteristic of a disturbance in the respectability of the glomerular filtration obstruction ^[44]. Then again, elevated amounts of low atomic weight proteins, (for example, β 2-microglobulin) are characteristic of decreased reuptake by the proximal tubule cells ^[45,46]. The event of proteinuria in neonates is firmly connected to gestational age during childbirth with studies in preterm newborn children reporting fundamentally more noteworthy egg whites and β 2-microglobulin fixations over the principal month of life in babies conceived <32 weeks incubation, contrasted with neonates conceived >32 weeks development ^[39,47]. To date, it stays vague whether the watched proteinuria in preterm newborn children is a consequence of their renal youthfulness or because of postnatal renal harm ^[48-55].

Variables that can Potentially Impact on the Development of the Immature Kidney

Intrauterine variables

It is currently very much perceived that the in utero environment can straightforwardly impact organ structure and improvement. Henceforth, it is likely that the components that lead to the incitement of preterm conveyance (unconstrained or helped) can conceivably affect on nephrogenesis and/or render the kidneys defenseless against unexpected labor and resulting pathology. In the following segments we portray a portion of the regular variables/conditions connected with preterm birth and how these components can unfavorably affect on the advancement of the fetal kidney ^[56-60].

Intrauterine disease and irritation (chorioamnionitis)

Intrauterine disease (specifically, chorioamnionitis) is generally recognized as a noteworthy benefactor to unexpected labor ^[13], particularly in births preceding 32 weeks growth ^[61-70]. A late study by Ogge et al. ^[71], found that ceaseless chorioamnionitis was included in 34% of the untimely conveyances identifying with preterm work with in place layers and 39% of preterm work with film crack. Chorioamnionitis is characterized as irritation of the chorion and amnion, brought on by a bacterial contamination which commonly rises from the vagina ^[3]. Essentially, chorioamnionitis can prompt fetal incendiary reaction disorder (FIRS) ^[72], and this has been appeared to antagonistically impact neonatal organ improvement. The impact of introduction to aggravation in utero on the fetal kidney has as of late been inspected in fetal sheep [73.74]. In the study by Galinsky et al. [73], there was a 20% lessening in nephron number, with no impact on body weight, when chorioamnionitis was affected in late incubation, utilizing an intense intra-amniotic bolus measurement of lipopolysaccharide (LPS, which starts an incendiary reaction like that saw with chorioamnionitis). Curiously, notwithstanding, when fetal sheep were presented to a lower dosage of LPS over an interminable period, amid the period in incubation when nephrogenesis is quickly continuous, there were no recognizable adverse impacts on nephrogenesis ^[74]. Henceforth, it gives the idea that with interminable low measurements introduction that the kidney might have the capacity to adjust, to counteract antagonistic impacts on nephron development. The differentiating discoveries from these two studiesdemonstrate that the planning, term and degree of disease/irritation are essential variables while surveying the effect of chorioamnionitis on the creating kidney.

Maternal diabetes

Presentation to intrauterine maternal diabetes can altogether impact fetal development all through growth and lead to an early onset to preterm birth; this is of concern given the late ascent in Type 1 and sort 2 and/or gestational diabetes ^[75,76]. A typical result of intrauterine presentation to maternal diabetes is macrosomia, specifically unbalanced macrosomia ^[77]. Macrosomia oftenleads to misrepresented fetal development, whereby the infant is conceived with a birth weight that is high for gestational age ^[78]. This expansion in body weight is an aftereffect of over the top measures of glucose and different supplements crossing the placenta prompting an increment in fetal body development. Interestingly, when maternal diabetes (both Type 1 and Type 2) is extreme, this can prompt IUGR in the baby ^[76,79]; the effects of IUGR on the kidney are portrayed later. With the expanded pervasiveness of maternal diabetes there have been various late studies taking a gander at the impacts on the fetal kidney. In a study led in preterm and term babies destined to Pima Indian mother, presentation to maternal diabetes (Type 2 diabetes) amid pregnancy prompted a higher discharge of egg whites (3.8 times higher) when contrasted with newborn children of pre-diabetic and non-diabetic moms; in this manner demonstrative of renal harm in posterity presented to diabetes in utero ^[80-90].

Postnatal Nutrition

Late studies highlight the significance of postnatal nourishment on the development and capacity of the kidney in IUGR and preterm newborn children. Positively, when nephrogenesis is progressing there are typically solid straight relationships between's nephron number and kidney size. Weakened development after birth (additional uterine development confinement; EUGR) regularly happens amid the postnatal period in preterm babies ^[91-95]; henceforth, it is likely that hindered body development in the prompt time frame after birth will antagonistically influence kidney development and nephron gift in the preterm newborn child. In this manner, there is the potential for enhanced postnatal nourishment to decidedly affect on the quantity of nephrons shaped. In backing of this thought, in a late investigation of preterm kids (conceived <30 weeks incubation) glomerular filtration rate was altogether diminished (suggestive of lessened nephron blessing) at 7 years old, in those that were either intra or additional uterine development confined ^[96-100]. Vitally, the additional uterine development confined kids were found to have altogether bring down protein-vitality admission amid their first week of life when contrasted with IUGR or suitably developed youngsters. What's more, watched that expending protein-rich recipe, contrasted with simply bosom milk, amid the early postnatal period brought on a noteworthy increment in kidney size.

CONCLUSION

This survey highlights the numerous variables connected with the etiology of preterm birth and in the postnatal environment that can possibly affect on the juvenile kidney of the preterm newborn child. Keeping in mind the end goal to enhance long haul renal wellbeing in subjects conceived preterm, it is currently essential in

future studies, to create interventional methodologies that alleviate the antagonistic effect of the intrauterine and additional uterine environment on the youthful kidney. At this stage, there is no unmistakable marker of the reasons for the glomerular variations from the norm connected with preterm birth. Deliberately controlled creature studies can explain the reasons for the glomerular anomalies and this is an essential territory of future exploration. With respect to renal harm, this survey highlights various medicines, ordinarily utilized as a part of the neonatal emergency unit can prompt renal weakness. Consequently, it is the test for the neonatologist, when choosing to utilize these pharmaceuticals, to find out whether the advantages exceed the dangers.

REFERENCES

- 1. Rodriguez-Barbero A, López-Novoa JM, Arévalo M (1997) Involvement of platelet-activating factor in gentamicin nephrotoxicity in rats.Exp Nephrol 5: 47-54.
- 2. Giapros VI, Andronikou SK, Cholevas VI, Papadopoulou ZL (2003) Renal function and effect of aminoglycoside therapy during the first ten days of life.Pediatr Nephrol 18: 46-52.
- 3. Kent AL, Maxwell LE, Koina ME, Falk MC, Willenborg D, et al. (2007) Renal glomeruli and tubular injury following indomethacin, ibuprofen, and gentamicin exposure in a neonatal rat model.Pediatr Res 62: 307-312.
- 4. Gilbert T, Lelievre-Pegorier M, Merlet-Benichou C (1990) Immediate and long-term renal effects of fetal exposure to gentamicin.Pediatr Nephrol 4: 445-450.
- 5. Kurki T, Eronen M, Lumme R, Ylikorkala O (1991) A randomized double-dummy comparison between indomethacin and nylidrin in threatened preterm labor.Obstet Gynecol 78: 1093-1097.
- 6. Pomeranz A, Korzets Z, Dolfin Z, Eliakim A, Bernheim J, et al. (1996) Acute renal failure in the neonate induced by the administration of indomethacin as a tocolytic agent.Nephrol Dial Transplant 11: 1139-1141.
- 7. Kent AL, Douglas-Denton R, Shadbolt B, Dahlstrom JE, Maxwell LE, et al. (2009) Indomethacin, ibuprofen and gentamicin administered during late stages of glomerulogenesis do not reduce glomerular number at 14 days of age in the neonatal rat. Pediatric Nephrology.24:1143-1149.
- 8. Vanderheyden T, Kumar S, Fisk NM (2003) Fetal renal impairment.Semin Neonatol 8: 279-289.
- 9. Nyberg DA, Mc Gahan JP, Pretorius DH, Pilu G (2002) Diagnostic imaging of fetal anomalies. Lippincott Williams & Wilkins, Philadelphia.
- 10. Klaassen I, Neuhaus TJ, Mueller-Wiefel DE, Kemper MJ (2007) Antenatal oligohydramnios of renal origin: longterm outcome.Nephrol Dial Transplant 22: 432-439.
- 11. Casey BM, McIntire DD, Bloom SL, Lucas MJ, Santos R, et al. (2000) Pregnancy outcomes after antepartum diagnosis of oligohydramnios at or beyond 34 weeks' gestation. Am J Obstet Gynecol 182: 909-912.
- 12. Hinchliffe SA, Lynch MR, Sargent PH, Howard CV, Van Velzen D (1992) The effect of intrauterine growth retardation on the development of renal nephrons.Br J Obstet Gynaecol 99: 296-301.
- 13. Mañalich R, Reyes L, Herrera M, Melendi C, Fundora I (2000) Relationship between weight at birth and the number and size of renal glomeruli in humans: a histomorphometric study.Kidney Int 58: 770-773.
- 14. Fiscella K1 (1996) Racial disparities in preterm births. The role of urogenital infections. Public Health Rep 111: 104-113.
- 15. Schieve LA, Handler A (1996) Preterm delivery and perinatal death among black and white infants in a Chicagoarea perinatal registry.0bstet Gynecol 88: 356-363.
- 16. de Onis M, Blössner M, Villar J (1998) Levels and patterns of intrauterine growth retardation in developing countries. Eur J Clin Nutr 52 Suppl 1: S5-15.
- 17. Painter RC, Roseboom TJ, Bleker OP (2005) Prenatal exposure to the Dutch famine and disease in later life: an overview.Reprod Toxicol 20: 345-352.
- 18. Godfrey K, Robinson S, Barker DJ, Osmond C, Cox V (1996) Maternal nutrition in early and late pregnancy in relation to placental and fetal growth.BMJ 312: 410-414.
- 19. Langley SC, Jackson AA (1994) Increased systolic blood pressure in adult rats induced by fetal exposure to maternal low protein diets.Clin Sci (Lond) 86: 217-222.
- 20. Merlet-Bénichou C, Gilbert T, Muffat-Joly M, Lelièvre-Pégorier M, Leroy B (1994) Intrauterine growth retardation leads to a permanent nephron deficit in the rat.Pediatr Nephrol 8: 175-180.
- 21. Desai M, Crowther NJ, Lucas A, Hales CN (1996) Organ-selective growth in the offspring of protein-restricted mothers.Br J Nutr 76: 591-603.

- 22. Zimanyi MA, Denton KM, Forbes JM, Thallas-Bonke V, Thomas MC, et al. (2006) A developmental nephron deficit in rats is associated with increased susceptibility to a secondary renal injury due to advanced glycation end-products. Diabetologia.49:801-810.
- 23. Lazzaroni F, Bonassi S, Magnani M, Calvi A, Repetto E, et al. (1993) Moderate maternal drinking and outcome of pregnancy.Eur J Epidemiol 9: 599-606.
- 24. Burguet A, Kaminski M, Abraham-Lerat L, Schaal JP, Cambonie G, et al. (2004) The complex relationship between smoking in pregnancy and very preterm delivery. Results of the Epipage study.BJOG 111: 258-265.
- 25. Sokol RJ, Janisse JJ, Louis JM, Bailey BN, Ager J, et al. (2007) Extreme prematurity: an alcohol-related birth effect. Alcohol Clin Exp Res 31: 1031-1037.
- 26. Jaddoe VW, de Ridder MA, van den Elzen AP, Hofman A, Uiterwaal CS, et al. (2008) Maternal smoking in pregnancy is associated with cholesterol development in the offspring: A 27-years follow-up study. Atherosclerosis 196: 42-48.
- 27. O'Leary CM, Nassar N, Kurinczuk JJ, Bower C (2009) The effect of maternal alcohol consumption on fetal growth and preterm birth.BJOG 116: 390-400.
- 28. Maccani MA, Avissar-Whiting M, Banister CE, McGonnigal B, Padbury JF, et al. (2010) Maternal cigarette smoking during pregnancy is associated with downregulation of miR-16, miR-, and miR-146a in the placenta. Epigenetics 5: 583-589.
- 29. Dejmek J, Solansk y I, Podrazilova K, Sram RJ (2002) The exposure of nonsmoking and smoking mothers to environmental tobacco smoke during different gestational phases and fetal growth. Environ Health Perspect.110:601-606.
- 30. Henriksen T, Clausen T (2002) The fetal origins hypothesis: placental insufficiency and inheritance versus maternal malnutrition in well-nourished populations. Acta Obstet Gynecol Scand 81: 112-114.
- 31. Schreuder M, Delemarre-van de Waal H, van Wijk A (2006) Consequences of intrauterine growth restriction for the kidney.Kidney Blood Press Res 29: 108-125.
- 32. Baschat AA, Cosmi E, Bilardo CM, Wolf H, Berg C, et al. (2007) Predictors of neonatal outcome in early-onset placental dysfunction.Obstet Gynecol 109: 253-261.
- Ananth CV, Smulian JC, Srinivas N, Getahun D, Salihu HM (2005) Risk of infant mortality among twins in relation to placental abruption: contributions of preterm birth and restricted fetal growth. Twin Res Hum Genet 8: 524-531.
- 34. Lyell DJ, Lambert-Messerlian GM, Giudice LC (2003) Prenatal screening, epidemiology, diagnosis, and management of preeclampsia.Clin Lab Med 23: 413-442.
- 35. Duley L1 (2009) The global impact of pre-eclampsia and eclampsia.Semin Perinatol 33: 130-137.
- 36. Sibai B, Dekker G, Kupferminc M (2005) Pre-eclampsia.Lancet 365: 785-799.
- 37. Churchill D, Perry IJ, Beevers DG (1997) Ambulatory blood pressure in pregnancy and fetal growth.Lancet 349: 7-10.
- 38. Walker BR, McConnachie A, Noon JP, Webb DJ, Watt GC (1998) Contribution of parental blood pressures to association between low birth weight and adult high blood pressure: cross sectional study.BMJ 316: 834-837.
- 39. Sibai BM1 (2003) Diagnosis and management of gestational hypertension and preeclampsia.Obstet Gynecol 102: 181-192.
- 40. López-Jaramillo P, García RG, López M (2005) Preventing pregnancy-induced hypertension: are there regional differences for this global problem?J Hypertens 23: 1121-1129.
- 41. Smith AP, Ong S, Smith NC, Campbell D (2001) A prospective longitudinal study of growth velocity in twin pregnancy.Ultrasound Obstet Gynecol 18: 485-487.
- 42. Keith L, Oleszczuk JJ (1999) latrogenic multiple birth, multiple pregnancy and assisted reproductive technologies.Int J Gynaecol Obstet 64: 11-25.
- 43. Fauser BC, Devroey P, Macklon NS (2005) Multiple birth resulting from ovarian stimulation for subfertility treatment.Lancet 365: 1807-1816.
- 44. Lynch A, McDuffie R, Stephens J, Murphy J, Faber K, et al. (2003) The contribution of assisted conception, chorionicity and other risk factors to very low birthweight in a twin cohort.BJOG 110: 405-410.

- 45. Chang YL, Chang SD, Chao AS, Wang CN, Wang TH, et al. (2011) The relationships of umbilical venous volume flow, birthweight and placental share in monochorionic twin pregnancies with and without selective intrauterine growth restriction. Twin Res Hum Genet.14:192-197.
- 46. Breathnach FM, Malone FD (2012) Fetal growth disorders in twin gestations. Semin Perinatol 36: 175-181.
- 47. Lucas SR, Costa Silva VL, Miraglia SM, Zaladek Gil F (1997) Functional and morphometric evaluation of offspring kidney after intrauterine undernutrition. Pediatr Nephrol.11:719-723
- 48. Louey S, Cock ML, Stevenson KM, Harding R (2000) Placental insufficiency and fetal growth restriction lead to postnatal hypotension and altered postnatal growth in sheep.Pediatr Res 48: 808-814.
- 49. Ozaki T, Nishina H, Hanson MA, Poston L (2001) Dietary restriction in pregnant rats causes gender-related hypertension and vascular dysfunction in offspring.J Physiol 530: 141-152.
- 50. Mitchell EK, Louey S, Cock ML, Harding R, Black MJ (2004) Nephron endowment and filtration surface area in the kidney after growth restriction of fetal sheep.Pediatr Res 55: 769-773.
- Moritz KM, Mazzuca MQ, Siebel AL, Mibus A, Arena D, et al. (2009) Uteroplacental insufficiency causes a nephron deficit, modest renal insufficiency but no hypertension with ageing in female rats. J Physiol.587:2635-2646.
- 52. Hamid R, Mufti G, Wani SA, Ali I, Bhat NA, et al. (2015) Importance of the Early Management of Omphalocele Minor. J Neonatal Biol 4:169.
- 53. Kaneshi Y, Sudo A, Cho K, Satomi T, Uchida M, et al. (2015) Avoidance of Tracheostomy in a Newborn of Congenital Central Hypoventilation Syndrome. J Neonatal Biol 4:170.
- 54. Divekar A, Seshia MM, Kesselman M (2015) Non-Restrictive Ductal Patency in Management of Cardiac Failure in Congenital Diaphragmatic Hernia Non-Invasive Biventricular Assist. J Neonatal Biol 4:171.
- 55. Laarman ARC, Ris JM, den Burger JCG, Veldkamp AI, Swart EL, et al. (2015) Pain Treatment of Newborns: Paracetamol Rectal Versus Intravenous Administration, A Randomised Open Clinical Trial. J Neonatal Biol 4:172.
- 56. Kiran B, Rajesh SM, Baliga BS (2015) Laryngomalacia in Neonates: A Review and the Surgical Management or Severe Cases . J Neonatal Biol 4:173.
- 57. Maisonneuve E, Whalen S, Conté C, Carbonne B, Guellec I (2015) First Case of Congenital Myeloproliferative Disorder in a Newborn Diagnosed With Noonan Syndrome. J Neonatal Biol 4:174.
- 58. Agarwal P, Arora H, Abdulhamid I, Asmar B, Natarajan G, Chawla S (2015) Pulmonary Hemorrhage in an Infant with Coronavirus Infection. J Neonatal Biol 4:175.
- 59. Navaei AH, Alvarez D, Rajegowda B, Khanna S (2015) Term Infant with Echogenic Fetal Lung Mass on Prenatal Sonogram. J Neonatal Biol 4:176.
- 60. kyoon Hwang J, kyung Park H, cheol Jun S, Lee HJ (2015) Spinnaker Sail Sign Accompanied with Pneumopericardium and Pneumoperitoneum. J Neonatal Biol 4:177.
- 61. Uzuki Y, Cho K, Honda S, Fujisawa S, Taketomi A, et al. (2015) Atypical Double-Bubble in MRI of a Fetus with Double Atresia Involving Esophagus and Jejunum. J Neonatal Biol 4:178.
- 62. Yamada N, Kodama Y, Kaneko M, Sameshima H, Ikenoue T (2015) Impact of Skin Lesions on Morbidity and Mortality in Extremely Premature Infants in One Tertiary Center in Southern Japan. J Neonatal Biol 4:179.
- 63. Gutiérrez-Padilla JA, De León JCB, Meza-Anguiano A, Rodríguez FA, Castañeda PC, et al. (2015) Propofol for Procedural Anaesthesia During Laser Treatment of Retinopathy of Prematurity in the Neonatal Intensive Care Unit (NICU). J Neonatal Biol 4:180.
- 64. You F, Weng L, Zhang Y, Cheng G, Qin Q, et al. (2015) Evaluation of Cardiac Reserve Function in Gestational Hypertension. J Neonatal Biol 4: 181.
- 65. Canals FJ, Vizcaíno C, Ferrández MJ, Serrano MI, Vázquez C, Quiles JL, et al. (2015) Minimally Invasive Surfactant Therapy: New Age. J Neonatal Biol 4:182.
- Tomaszewska E, Dobrowolski P, Hułas-Stasiak M, Tomczyk A (2015) Maternal Nutrition with B Hydroxy B -Methylbutyrate as Strong Determinants of the Development of Newborn Offspring in Pigs. J Neonatal Biol 4:183.
- 67. Abiodun MT, Oluwafemi RO, Fabunmi O, Ajimuda T (2015) Cervical Teratoma and Cystic Hygroma in Nigerian Infants: Case Studies of Two Differential Diagnoses of Neonatal Neck Mass and Review of the Literature. J Neonatal Biol 4:184.

- 68. Jose R, Kumari P, Vijayaselvi R, Beck MM (2015) Audit to Assess the Implementation of Early First Feeding in Newborns after Caesarean Delivery . J Neonatal Biol 2015 4:185.
- 69. Al-Saloos H (2015) Case of Laryngeal Atresia, Tracheal Agenesis, Tracheo-Esophageal Fistula, Double Outlet Right Ventricle and Persistent Left Superior Vena Cava. J Neonatal Biol 4:186.
- 70. Manfredini VA, Cerini C, Giovanettoni C, Brazzoduro EA, Rezzonico RM (2015) Metabolic Bone Disease of Prematurity: A Review of Minerals Supplementation and Disease Monitoring. J Neonatal Biol 2015 4:187.
- 71. Imaizumi Y, Hayakawa K (2015) Perinatal Mortality Rates and Risk Factors for Mortality among Zygotic Twins and Singletons in Japan, 1995-2008. J Neonatal Biol 4:188.
- 72. Uchenna E, Nwabueze AI, Kingsley NI (2015) Neonatal Polycythemia Secondary to Twin to Twin Transfusion Syndrome- A Case Report. J Neonatal Biol 4:189.
- 73. Ndu IK, Edelu BO, Uwaezuoke S, Chinawa JC, Ubesie A (2015) Maternal Risk Factors Associated with Low Birth Weight Neonates: A Multi-Centre, Cross-Sectional Study in a Developing Country. J Neonatal Biol 4:190.
- 74. Kotiya P, Zhu X (2015) Effects of Early and Late Parenteral Nutrition on Clinical Outcomes in Very Low Birth Weight Preterm Infants: A Systematic Review and Meta-analysis. J Neonatal Biol 4:191.
- 75. Raban MS, Muller SJ, Harrison MC (2015) An Unusual Case of Congenital Syngnathia. J Neonatal Biol 4:192.
- 76. Vargas NSO, Ceccon MEJ, CiceroFalcao M, De Carvalho WB (2015) Prognostic Markers of Neonatal Outcomes in Full Term Neonates Suffering from Perinatal Asphyxia. J Neonatal Biol 4:193.
- 77. Shiono, Takei, Yamada, Tachibana, Cho Minakami, et al. (2015) A Case of Prenatal Presentation with Double Aortic Arch. J Neonatal Biol 4:194.
- 78. Sunil Munakomi. (2015) Vein of Galen Aneurysmal Malformation. J Neonatal Biol 4:195.
- 79. Bülbül A, Dursun M, Yildirmak Y, Akyol B, Zübarioglu U, et al. (2015) Spontaneous Remission in Congenital Leukemia AML-M1 with Pericardial Effusion. J Neonatal Biol 4:196.
- Clelia T, Irene B, Lucio PC (2015) Hair Collar Sign and Viral Infection in Pregnancy: Two Clinical Cases. J Neonatal Biol 4:197
- 81. Kobata K, Nabetani M, Yutaka N, Hiroyuki, Sanno (2015) Experiences of Therapeutic Hypothermia Therapy on Six Cases with Persistent: Pulmonary Hypertension and Moderate to Severe Hypoxic Ischemic Encephalopathy using Inhaled Nitric Oxide Therapy. J Neonatal Biol 4:198.
- 82. Dorbani A, Bairi A, Ouakid ML, Tahraoui A (2015) Endocrine and Immunological Alterations under Early Nasal Obstruction in Rats Wistar. J Neonatal Biol 4:199.
- 83. Kondo T, Honda S, Minato M, Fujisawa S, Miyagi H, et al., (2015) A Preoperative Diagnostic Challenge of a Long Overlapping Upper Pouch with Distal Tracheoesophageal Fistula. J Neonatal Biol 4:200.
- 84. Renoldner B, Hofer N, Resch B (2015) Early-Onset Neonatal Sepsis: Group B Streptococcal Compared to E. coli Disease. J Neonatal Biol 4:201.
- 85. Garg PM, Garg PP, Lal CV (2015) Necrotizing Enterocolitis (NEC): A Devastating Disease of Prematurity. J Neonatal Biol 4:202.
- 86. Zhang H, Ling Y (2015) Head to Shoulder Interval in 92 Cases Normal Birth with Good Baby Condition by Two Step Shoulder Delivery Method. J Neonatal Biol 4:203.
- 87. Wiberg-Itzel E, Josephson H, Wiberg N, Olson L, Winbladh B, et al. (2015) Lactic Dehydrogenase in Umbilical Cord Blood in Healthy Infants after Different Modes of Delivery. J Neonatal Biol 4:204.
- 88. Shenoy J, Reddy V, Baliga KN (2014) Serum Lipid Profile in Preterm and Term Appropriate for Gestational Age Indian Newborns: A Hospital Based Comparative Study. J Neonatal Biol 3:156.
- 89. Manohar PS, Tukaram MV, Satish PU, Satish PS, Sarjerao PU, et al. (2014) "Human Milk Component (HMC) Therapy": A Novel Treatment for Gut Maturation of Preterm Infants. J Neonatal Biol 3:157.
- Matturri L, Pusiol T, Lavezzi AM (2014) Proposal of the Acronym "SIUDS" for Unexplained Stillbirths, Like "SIDS". J Neonatal Biol 3:165.
- 91. Imaizumi Y, Hayakawa K (2014) Stillbirth Rates and Risk Factors for Stillbirths among Zygotic Twins in Japan, 1995-2008. J Neonatal Biol 3:164.
- 92. Herruzo R, Ruiz G, Rubio M, Cruz–Troca JJ, Mora E, et al. (2014) Controlling an Outbreak of Pseudomonas aeruginosa in a Neonatal Intensive Care Unit: Multivariate Analysis of Risk Factors through a Case-Case-Control Study. J Neonatal Biol 3:163.

- 93. Oluwayemi I O, Ogundaren E O, Olatunya O S (2014) Care of the Newborn in Developing Countries . J Neonatal Biol 3:162.
- 94. Sharma D, Pandita A, Murki S, Pratap T, Madhavi V (2014) Suspected Smith-Lemli-Opitz-Syndrome: A Very Rare Syndrome. J Neonatal Biol 3:161. D
- 95. May L E, Suminski R R (2014) Amount of Physical Activity in Pregnancy and Infant Heart Outcomes. J Neonatal Biol 3:160.
- 96. Sobaih B H, Al-Mandeel H (2014) Early and Late Onset Neonatal Sepsis in Very Low Birth Weight Infants in a Tertiary Center in Saudi Arabia. J Neonatal Biol 3:159.
- 97. Vijayalakshmi S, Patil R, Datta SS (2014) Community-based Study on Newborn Care Practices and its Determinants in Rural Pondicherry, India. J Neonatal Biol 3:158.
- 98. Manohar PS, Tukaram MV, Satish PU, Satish PS, Sarjerao PU, et al. (2014) "Human Milk Component (HMC) Therapy": A Novel Treatment for Gut Maturation of Preterm Infants. J Neonatal Biol 3:157.
- 99. Prasad M, Miller M, Bhutada A, Rastogi S (2014) Citrulline: Is it Ready for Primetime. Its Uses and Limitations in Neonatal Medicine. J Neonatal Biol. 3:147.
- 100. Chhugani M, Sarkar S (2014) Therapeutic Touch Modalities and Premature Neonate's Health Outcome: A Literature Review. J Neonatal Biol 3:148.