RESEARCH PAPER

Comparison of Umbilical Cord Lipid Profile in Preterm and Term Neonates in Relation to Gestational Age and Birth Weight

Dr. Israr Alam[1], Dr. Bindu T Nair[2*], Dr. Meenakshi Bothra[3], Dr. Praneta Swarup[4]

[1]*Junior Resident, Sharda School of Medical Sciences & Research, Sharda Hospital, Sharda University, Gr. Noida*
[2*] *Professor, Sharda School of Medical Sciences & Research, Sharda Hospital, Sharda University, Gr. Noida*
[3]*Professor, Sharda School of Medical Sciences & Research, Sharda Hospital, Sharda University, Gr. Noida*
[4]*Associate Professor, Sharda School of Medical Sciences & Research, Sharda Hospital, Sharda University, Gr. Noida*

ABSTRACT

Background: Foetal lipid metabolism is critical for growth and development, and disturbances during intrauterine life may predispose individuals to cardiometabolic disease later in life. Umbilical cord blood lipid profiling provides insight into foetal nutritional status and placental lipid transfer.

Objectives: To compare umbilical cord lipid profiles between preterm and term neonates and to evaluate their association with gestational age and birth weight.

Methods: This observational study was conducted at a tertiary-care teaching hospital in North India over 18 months. Cord blood samples from 174 neonates (87 each of preterm and, 87 term neonates) were analysed for total cholesterol (TC), triglycerides (TG), high-density lipoprotein (HDL), low-density lipoprotein (LDL), and very-low-density lipoprotein (VLDL). Neonates were categorized by gestational age and birth weight (AGA/SGA; LBW/NBW). Statistical analysis was performed using SPSS v25.

Results: Preterm neonates had significantly higher TC, TG, HDL, and VLDL levels than term neonates ($p<0.01$), while LDL differences were not significant. SGA neonates, both preterm and term, demonstrated dyslipidaemia characterized by higher TG and lower HDL levels compared with AGA counterparts. All lipid parameters were significantly higher in low-birth-weight neonates. No significant gender-based differences were observed.

Conclusion: Cord blood lipid profiles vary significantly with gestational age and birth weight. Preterm and SGA neonates exhibit a more atherogenic lipid pattern, supporting the foetal origins of adult disease hypothesis. Early identification of at-risk neonates may enable targeted preventive strategies.

Keywords: Umbilical cord blood, lipid profile, preterm neonates, small for gestational age, Barker hypothesis

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INTRODUCTION

Foetal lipid metabolism undergoes progressive adaptation throughout gestation to meet the increasing demands of cellular growth, membrane synthesis, and energy storage. During normal pregnancy, maternal lipid concentrations rise, particularly in the third trimester, facilitating placental transfer of cholesterol and triglycerides essential for foetal development. Umbilical cord blood lipid profile thus reflects intrauterine metabolic status and provides an early window into foetal programming of cardiometabolic health [1,2].

Preterm birth interrupts late gestational metabolic maturation, while intrauterine growth restriction exposes the foetus to chronic nutritional stress. Both conditions are associated with altered hepatic lipid synthesis, lipoprotein metabolism, and enzymatic activity. Recent studies have demonstrated significantly higher cord blood concentrations of total cholesterol, triglycerides, and very-low-density lipoprotein (VLDL) in preterm neonates compared to term neonates, suggesting immaturity of lipid clearance mechanisms [3–5]. Similarly, small-for-gestational-age (SGA) neonates exhibit dyslipidaemic patterns characterized by hypertriglyceridemia and reduced high-density lipoprotein (HDL) levels, reflecting adverse metabolic adaptations in utero [6,7].

Birth weight independently influences neonatal lipid profiles, with low-birth-weight neonates showing elevated atherogenic lipoproteins irrespective of gestational age [8]. These early lipid alterations are consistent with the developmental origins of health and disease hypothesis, which proposes that adverse intrauterine environments predispose individuals to long-term cardiovascular and metabolic disorders [9].

The present study was undertaken to compare umbilical cord blood lipid profiles between preterm and term neonates in relation to gestational age and birth weight, with the aim of identifying early metabolic markers that may predict future cardiometabolic risk.

**Author for Correspondence:* Dr Bindu T Nair

MATERIAL AND METHODS

This was a hospital-based observational study conducted in the Department of Paediatrics of a tertiary care hospital in North India, from April 2024 to October 2025.A total of 174 live-born neonates with gestational age between 32 and 42 weeks and a 1-minute Apgar score >7 were included. Neonates with congenital anomalies, perinatal asphyxia, respiratory distress, large-for-gestational-age status, or born to mothers with metabolic or chronic illnesses were excluded.

Umbilical cord blood was collected from the placental end immediately after delivery. Lipid profile estimation (TC, TG, HDL, LDL, VLDL) was performed using the VITROS 5600 autoanalyzer. Anthropometric measurements were recorded, and gestational age was assessed using the last menstrual period and confirmed by the modified New Ballard score.

Data were analysed using IBM SPSS Statistics version 25(V25) Normality of each variable was assessed by using the Kolmogorov- Simirnov test and Shapiro-Wilk test. Quantitative data was expressed by mean, standard deviation or median with interquartile range difference between means of two groups was tested by unpaired t test or Mann Whitney U test. Qualitative data was expressed in percentage and difference between the proportions was tested by chi square test or Fisher's exact test. 'P' value less than 0.05 was considered statistically significant.

OBSERVATION AND RESULTS

A total of 174 healthy neonates (87 neonates each in term and preterm group) born with gestational age between 32 to 42 weeks of gestational age born were included in the study. Demographic and clinical characteristics of the study population is shown in Table 1.

Table 1: Distribution of Study Neonates by Demographic and Clinical Characteristics (n = 174)

Variable	Category	Number (n)	Percentage (%)
Gender	Male	82	47.1
	Female	92	52.9
Gestational Age	Preterm	87	50.0
	Term	87	50.0
Birth weight for GA	AGA	102	58.6
	SGA	72	41.4
Preterm subgroup	AGA	53	60.9
	SGA	34	39.1
Term subgroup	AGA	49	56.3
	SGA	38	43.7
Birth Weight	LBW	109	62.6
	NBW	65	37.4

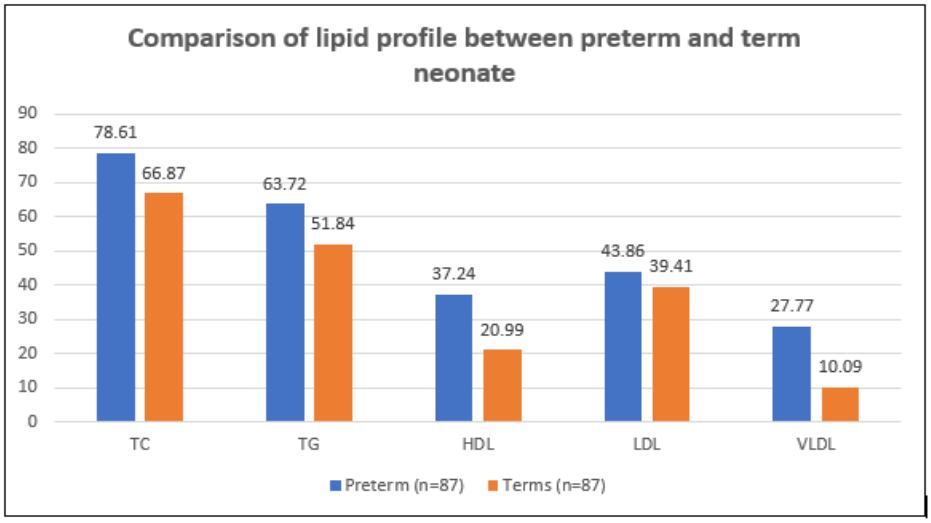

Figure 1: Comparison of lipid profile between preterm and term neonate

It was observed that preterm neonates had significantly higher mean levels of total cholesterol, triglycerides (TG), high-density lipoprotein (HDL), and very low-density lipoprotein (VLDL) compared with term neonates, with p values < 0.01 for all parameters (Figure 1).

TABLE 2 : Comparison of Cord Blood Lipid Profile Across Gestational Age, Growth Status, Birth Weight

Lipid Parameter	Preterm (n=87)	Term (n=87)	*p* value	Preterm AGA (n=53)	Preterm SGA (n=34)	*p* value	Term AGA (n=49)	Term SGA (n=38)	*p* value
Total Cholesterol	78.61 ± 15.86	66.87 ± 21.41	<0.01	75.57 ± 14.52	83.35 ± 16.90	0.02	65.31 ± 25.22	68.89 ± 15.24	0.44
Triglycerides	63.72 ± 23.15	51.84 ± 22.13	<0.01	61.62 ± 19.97	67.00 ± 27.40	0.29	47.39 ± 23.94	57.58 ± 18.30	0.03
HDL Cholesterol	37.24 ± 15.83	20.99 ± 5.27	<0.01	41.26 ± 15.56	30.97 ± 14.32	<0.01	22.08 ± 4.99	19.58 ± 5.36	0.02
LDL Cholesterol	43.86 ± 14.70	39.41 ± 19.66	0.09	44.68 ± 12.65	42.59 ± 17.56	0.52	37.51 ± 21.67	41.87 ± 16.67	0.30
VLDL Cholesterol	27.77 ± 16.61	10.09 ± 5.49	<0.01	31.62 ± 16.17	21.76 ± 15.68	<0.01	8.90 ± 5.73	11.63 ± 4.80	0.02

Footnote:
Comparisons between two groups were performed using unpaired t-test or Mann–Whitney U test, as appropriate. A p value <0.05 was considered statistically significant.

The mean total cholesterol level in preterm neonates was 78.61 ± 15.86 mg/dL, whereas in term neonates it was 66.87 ± 21.41 mg/dL, indicating a statistically significant elevation in the preterm group. Similarly, the mean

triglyceride level was 63.72 ± 23.15 mg/dL in preterm neonates compared to 51.84 ± 22.13 mg/dL in term neonates, again showing a highly significant difference ($p < 0.01$) **(Table 2).**

Preterm neonates showed significantly higher levels of TC, TG, HDL, and VLDL compared to term neonates ($p < 0.01$), while the difference in LDL was not significant ($p = 0.09$), indicating an overall higher lipid profile in preterm newborns **(Table 2).**

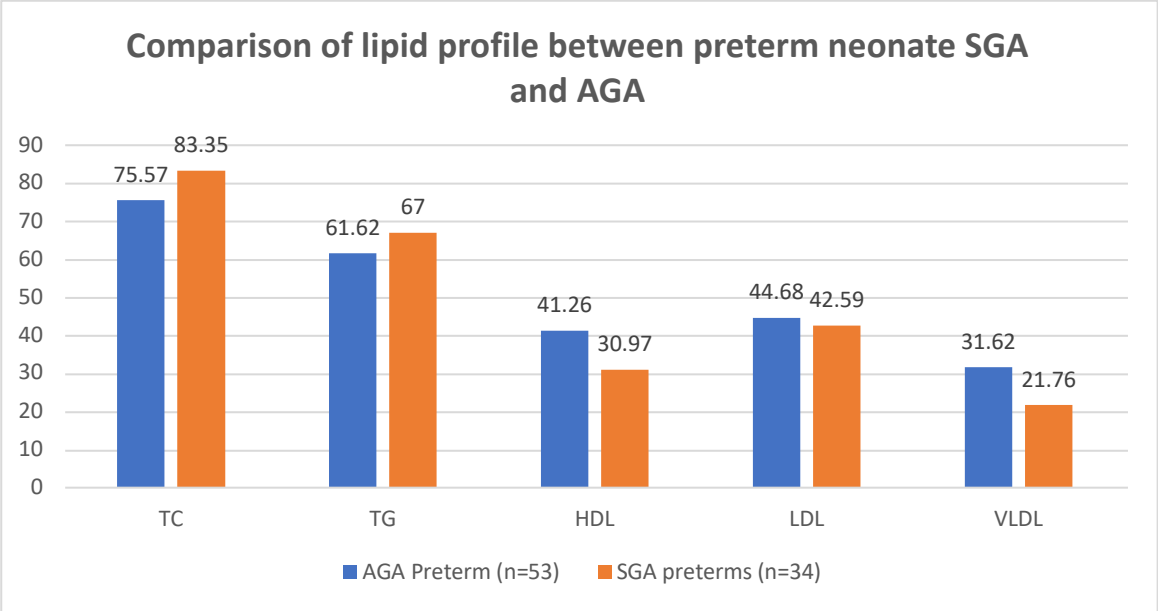

Figure 2: Comparison of lipid profile between preterm SGA and AGA neonate

It was observed that SGA preterm neonates had significantly higher mean total cholesterol levels (83.35 ± 16.90 mg/dL) compared to AGA preterm neonates (75.57 ± 14.52 mg/dL), with the difference being statistically significant ($p = 0.02$).The mean triglyceride (TG) level was slightly higher in SGA preterm (67.00 ± 27.40 mg/dL) than in AGA preterm (61.62 ± 19.97 mg/dL), but this difference did not reach statistical significance ($p = 0.29$). In preterm neonates, SGA babies had significantly higher total cholesterol ($p = 0.02$) and lower HDL and VLDL levels ($p < 0.01$ each) compared to AGA preterm. Differences in triglycerides and LDL were not statistically significant **(Figure 2, Table 2)**

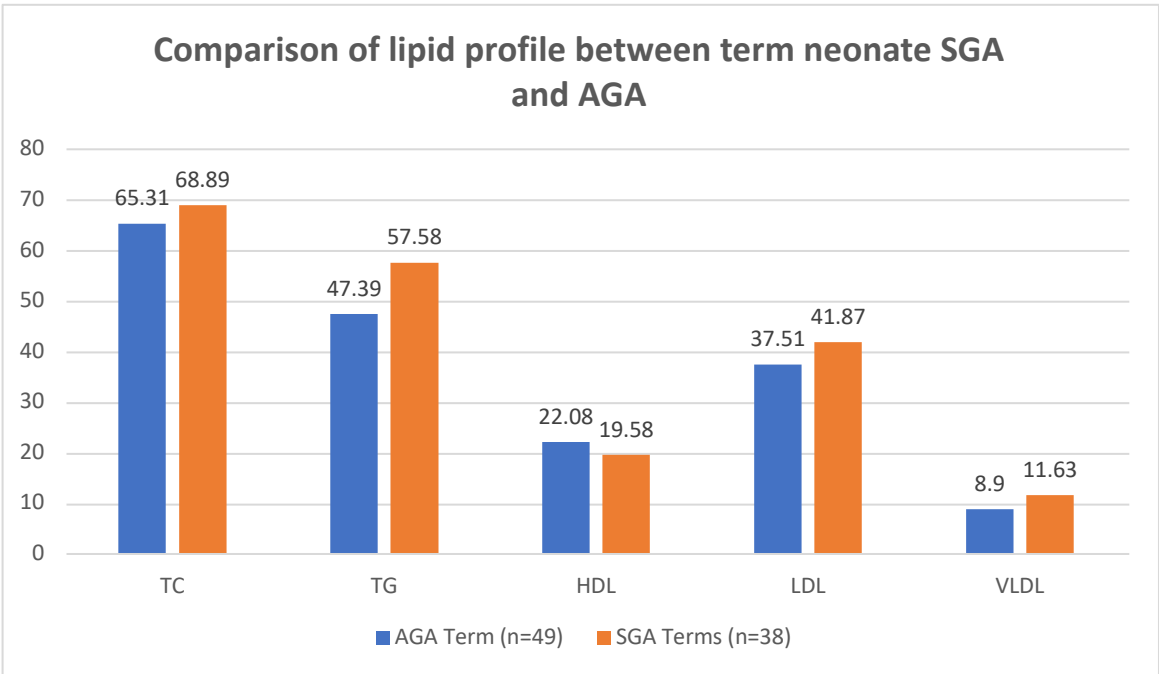

Figure 3: Comparison of lipid profile between term SGA and AGA neonate

The mean total cholesterol level was marginally higher in SGA term neonates (68.89 ± 15.24 mg/dL) compared to AGA term neonates (65.31 ± 25.22 mg/dL), but the difference was not statistically significant (p = 0.44). A significant difference was observed in triglyceride (TG) levels, with SGA term neonates showing higher mean values (57.58 ± 18.30 mg/dL) compared to AGA term neonates (47.39 ± 23.94 mg/dL, p = 0.03). Among term neonates, SGA babies showed significantly higher triglyceride and VLDL levels (p = 0.03 and 0.02, respectively) and lower HDL levels (p = 0.02) compared to AGA term neonates. Differences in total cholesterol and LDL were not statistically significant **(Figure 3, Table 2)**

DISCUSSION

The present study evaluated umbilical cord blood lipid profiles in neonates and demonstrated significant variations in lipid parameters in relation to gestational age and birth weight. These findings reinforce the concept that foetal maturity and growth patterns play a pivotal role in determining neonatal lipid metabolism at birth.

Preterm neonates in the present study exhibited significantly higher mean levels of total cholesterol, triglycerides, HDL cholesterol, and VLDL cholesterol compared to term neonates, while LDL levels did not differ significantly. Similar observations have been reported in recent neonatal studies by Ghaemi et al, Mishra et al and Daniel et al, which attribute elevated lipid levels in preterm infants to immature hepatic enzyme systems, reduced lipoprotein lipase activity, and impaired peripheral utilization of lipids [3,4,10]. Interruption of normal third-trimester metabolic adaptations may further contribute to the accumulation of circulating lipids in preterm neonates [1].

The significantly higher triglyceride and VLDL levels observed in preterm neonates suggest reduced clearance of triglyceride-rich lipoproteins. Experimental data indicate that lipoprotein lipase activity matures late in gestation, explaining the persistence of elevated triglycerides in preterm infants [11]. The higher HDL cholesterol levels noted in preterm neonates may represent a compensatory mechanism aimed at cholesterol transport and antioxidative protection during early life, as HDL plays a critical role in reverse cholesterol transport and endothelial protection [12].

Among preterm neonates, those who were SGA demonstrated significantly higher total cholesterol levels and lower HDL and VLDL levels compared to AGA counterparts. These findings indicate that intrauterine growth restriction exerts an additional adverse effect on lipid metabolism beyond prematurity alone. Chronic foetal undernutrition has been shown to upregulate hepatic cholesterol synthesis while impairing lipoprotein remodelling, resulting in dysregulated lipid profiles detectable at birth [6,13]. The Barker hypothesis postulates that undernutrition during critical periods of foetal development results in long-term changes in lipid metabolism, insulin sensitivity, and vascular function [14]. The observed higher triglyceride and VLDL levels and lower HDL levels in SGA neonates in the present study provide further evidence in favour of this hypothesis. These lipid alterations may persist into childhood and adulthood, increasing the risk of atherosclerosis, metabolic syndrome, and coronary artery disease [15].

Reduced HDL levels in SGA preterm neonates are of particular concern, as impaired HDL-mediated cholesterol efflux has been associated with early endothelial dysfunction and increased cardiovascular risk later in life [16].

In term neonates, SGA infants exhibited significantly higher triglyceride and VLDL levels and lower HDL levels compared to AGA neonates, while differences in total cholesterol and LDL were not statistically significant. These findings are consistent with recent studies by Zamojska et al and Zhang et al , reporting dyslipidaemia in term SGA neonates, predominantly characterized by hypertriglyceridemia and reduced HDL cholesterol [7,17]. Such lipid alterations support the foetal programming hypothesis, whereby adverse intrauterine environments induce permanent metabolic changes that predispose individuals to insulin resistance, dyslipidaemia, and cardiovascular disease in adulthood [9,18].

Low-birth-weight neonates in the present study demonstrated significantly higher levels of total cholesterol, triglycerides, LDL, HDL, and VLDL compared to normal-birth-weight neonates. Similar associations between low birth weight and adverse lipid profiles have been confirmed in recent systematic reviews and cohort studies [8,19]. Elevated LDL and VLDL levels are of particular clinical relevance, as these lipoproteins are strongly atherogenic and have been linked to early vascular changes in growth-restricted infants [20]. Although HDL levels were also higher in low-birth-weight neonates, emerging evidence suggests that HDL functionality may be impaired despite higher concentrations, limiting its cardioprotective role [21].

No significant gender-based differences in lipid parameters were observed in the present study, consistent with previous reports indicating that sexual dimorphism in lipid metabolism emerges later in life under the influence of pubertal hormonal changes rather than during the neonatal period [10,22].

The findings of the present study have important clinical implications. Early identification of dyslipidaemia in preterm, SGA, and low-birth-weight neonates may allow targeted nutritional interventions, optimized growth monitoring, and long-term follow-up to mitigate future cardiometabolic risk. Cord blood lipid profiling represents a simple, non-invasive tool that may serve as an early biomarker of altered metabolic programming.

Limitations of study: The present study was from a single-centre with limited sample size. There was a lack of maternal lipid profiling; absence of longitudinal follow-up to assess long-term outcomes.

**Author for Correspondence:* Dr Bindu T Nair

CONCLUSION

Umbilical cord lipid profile varies significantly with gestational age and birth weight. Preterm and SGA neonates exhibit a more atherogenic lipid pattern at birth, supporting the foetal origins of adult disease hypothesis. Cord blood lipid profiling may serve as an early marker for identifying neonates at risk for future cardiometabolic disorders.

REFERENCES

1. Herrera E, Ortega-Senovilla H. Lipid metabolism during pregnancy and its implications for fetal development. Placenta. 2021;104:152-60. DOI: 10.2174/1389201015666140330192345
2. Koletzko B, Godfrey KM, Poston L, Szajewska H, van Goudoever JB, de Waard M, et al. Nutrition during pregnancy, lactation and early childhood and its implications for long-term health. Lancet Child Adolesc Health. 2021;5:408-17. DOI: 10.1159/000496471
3. Nghiem-Rao TH, Dahlgren AF, Kalluri D, Cao Y, Simpson PM, Patel SB. Influence of gestational age and birth weight in neonatal cholesterol response to total parenteral nutrition.
4. J Clin Lipidol. 2016 Apr 28;10(4):891-897.e1. doi: 10.1016/j.jacl.2016.03.005.
5. Yadav S, Mishra S, Singh A. Cord blood lipid profile in preterm and term neonates and its association with gestational age. J Neonatal Perinatal Med. 2022;15:527-34.
6. Li X, Zhang M, Wang Y. Alterations in cord blood lipid profile among preterm infants. BMC Pediatr. 2022;22:614.
7. Adam-Raileanu A, Miron I, Lupu A, Bozomitu L, Sasaran MO, Russu R, et al. Fetal Growth Restriction and Its Metabolism-Related Long-Term Outcomes-Underlying Mechanisms and Clinical Implications. Nutrients. 2025 Jan 31;17(3):555. doi: 10.3390/nu17030555.
8. Zamojska J, Niewiadomska-Jarosik K, Kierzkowska B, Gruca M, Wosiak A, Smolewska E. Lipid Profile in Children Born Small for Gestational Age. Nutrients. 2023;15(22):4781. doi: 10.3390/nu15224781.
9. Chen B, Chen Y, Wang Y, Xin Q, Ma D. The association between rapid growth and lipid profile: a systematic review and meta-analysis. Front Endocrinol (Lausanne). 2024 Mar 21;15:1353334. doi: 10.3389/fendo.2024.1353334.
10. Barker DJ. The developmental origins of adult disease. J Am Coll Nutr. 2004 Dec;23(6 Suppl):588S-595S. doi: 10.1080/07315724.2004.10719428.
11. Daniels SR, Pratt CA, Hayman LL. Reduction of risk for cardiovascular disease in children and adolescents. Circulation. 2011 Oct 11;124(15):1673-86. doi: 10.1161/CIRCULATIONAHA.110.016170.
12. Herrera E, Lasunción MA, Gomez-Coronado D, Aranda P, López-Luna P, Maier I. Role of lipoprotein lipase activity on lipoprotein metabolism and the fate of circulating triglycerides in pregnancy. Am J Obstet Gynecol. 1988 Jun;158(6 Pt 2):1575-83. doi: 10.1016/0002-9378(88)90193-7.
13. Casula M, Colpani O, Xie S, Catapano AL, Baragetti A. HDL in Atherosclerotic Cardiovascular Disease: In Search of a Role. Cells. 2021 Jul 23;10(8):1869. doi: 10.3390/cells10081869.
14. Bock J, Wainstock T. Fetal programming of lipid metabolism and cardiometabolic risk. Nutrients. 2023;15:1184.
15. Barker DJ. The fetal and infant origins of adult disease. BMJ. 1990 Nov 17;301(6761):1111. doi: 10.1136/bmj.301.6761.1111.
16. Eriksson JG, Forsén T, Tuomilehto J, Osmond C, Barker DJ. Early growth and coronary heart disease in later life: longitudinal study. BMJ. 2001 Apr 21;322(7292):949-53. doi: 10.1136/bmj.322.7292.949.
17. Rosenson RS, Brewer HB Jr, Ansell BJ, Barter P, Chapman MJ, Heinecke JW, Kontush A, Tall AR, Webb NR. Dysfunctional HDL and atherosclerotic cardiovascular disease. Nat Rev Cardiol. 2016 Jan;13(1):48-60. doi: 10.1038/nrcardio.2015.124.
18. Zhang Y, Liu H, Chen X. Lipid profile abnormalities in term small-for-gestational-age neonates. J Pediatr Endocrinol Metab. 2022;35:1345-1352.
19. Singhal A. Early nutrition and long-term cardiovascular health. Nutr Rev. 2006 May;64(5 Pt 2):S44-9; discussion S72-91. doi: 10.1301/nr.2006.may.s44-s49.
20. Jańczewska I, Wierzba J, Jańczewska A, Szczurek-Gierczak M, Domżalska-Popadiuk I. Prematurity and Low Birth Weight and Their Impact on Childhood Growth Patterns and the Risk of Long-Term Cardiovascular Sequelae. Children (Basel). 2023 Sep 25;10(10):1599. doi: 10.3390/children10101599.
21. McGill HC Jr, McMahan CA, Gidding SS. Preventing heart disease in the 21st century: implications of the Pathobiological Determinants of Atherosclerosis in Youth (PDAY) study. Circulation. 2008 Mar 4;117(9):1216-27. doi: 10.1161/CIRCULATIONAHA.107.717033. 21. Agouridis AP, Banach M, Mikhailidis DP. Dysfunctional high-density lipoprotein: not only quantity but first of all quality? Arch Med Sci. 2015 Mar 16;11(1):230-1. doi: 10.5114/aoms.2015.49816.
22. World Health Organization. Guidelines on neonatal nutrition and long-term health outcomes. Geneva: WHO; 2024.(Last accessed on 24 Jan, 2026).