Environment International 211 (2026) 110238

Contents lists available at ScienceDirect

Environment International

journal homepage: www.elsevier.com/locate/envint

Full length article

# Early-life lipid programming: Environmental and biological drivers of neonatal cholesterol

Rossella Alfano [a,*], Tim S. Nawrot [a,b], Charlotte Cosemans [a], Joris Penders [c], Brigitte Reimann [a], Congrong Wang [a], Michelle Plusquin [a]

[a] Centre for Environmental Sciences, Environmental and Molecular Epidemiology, Hasselt University, Agoralaan Building D, 3590 Diepenbeek, Belgium
[b] Department of Public Health & Primary Care, Leuven University, Leuven, Belgium
[c] Limburg Clinical Research Center, Hasselt University, Genk, Belgium

ARTICLE INFO

*Keywords:*
Exposome
Cholesterol
Cord blood
Prenatal

ABSTRACT

*Background:* Cholesterol at birth influences development and long-term health, but its environmental and biological determinants remain understudied.
*Methods:* This study investigates associations between the prenatal exposome and cord blood lipid profiles in 1,732 mother–child pairs from the Belgian ENVIRONAGE cohort. An exposome-wide association study, a deletion/substitution/addition variable selection, and multi-exposure regressions were applied to assess 90 external exposures in relation to cord blood total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), non-HDL-C, and low-density lipoprotein cholesterol (LDL-C). Partial correlations were used to assess relations between 14 internal exposures and cholesterol levels. Mediation of identified internal exposures was tested via an imputation-based approach.
*Results:* Greater sunshine duration and higher atmospheric pressure during pregnancy were inversely associated with TC. Exposure to black carbon, presence of a heating tank in the household, maternal smoking, and primiparity were associated with lower HDL-C, while folic acid supplementation was associated with higher HDL-C. Primiparity was also associated with higher non-HDL-C, and sunshine exposure with lower non-HDL-C. Several biomarkers, including ferritin, thyroid hormones, and inflammatory markers, were correlated with lipid profiles. Homocysteine mediated the effect of atmospheric pressure on TC. Triiodothyronine (fT3), insulin, estradiol, and IL-6 mediated the effect of smoking on HDL-C. fT3 mediated the effect of folic acid on HDL-C, while insulin mediated the effect of primiparity on HDL-C. These findings reveal complex interactions between prenatal environmental exposures, internal biomarkers, and newborn lipid profiles, underscoring the importance of early-life exposome research for preventive health strategies.

## 1. Introduction

At birth, cholesterol plays a critical role in development as a structural component of cell membranes, a precursor of steroid hormones, and a signalling molecule (Schade et al., 2020). Cord blood cholesterol levels have been associated with adverse birth outcomes, including large for gestational age (LGA) (Alfano et al., 2020; Ye et al., 2022), preterm birth (Ghaemi et al., 2014), as well as later childhood outcomes such as obesity (Algaba-Chueca et al., 2022) and emotional and social development (Manczak and Gotlib, 2019). Importantly, cord blood cholesterol has been shown to predict cholesterol levels in childhood, which in turn predict adult levels, suggesting that early-life lipid profiles may serve as early indicators of long-term cardiometabolic risk (Juhola et al., 2011; Taageby Nielsen et al., 2023). Within the Developmental Origins of Health and Disease (DOHaD) framework, such early-life biological markers are increasingly conceptualised as capturing fetal responses to prenatal environments that may influence later disease risk (Barker, 2007; Dai et al., 2020).

Previous research identified factors such as gestational age, parity, birthweight, maternal physical activity, and lipid levels during pregnancy that are associated with neonatal cholesterol levels (Alfano et al., 2020; Collings et al., 2020; Ghaemi et al., 2014; Go et al., 2023; Øyri

* Corresponding author at: Centre for Environmental Sciences, Environmental and Molecular Epidemiology, Hasselt University, Agoralaan gebouw, D BE-3590 Diepenbeek, Belgium.
*E-mail address:* rossella.alfano@uhasselt.be (R. Alfano).

https://doi.org/10.1016/j.envint.2026.110238
Received 21 July 2025; Received in revised form 25 February 2026; Accepted 3 April 2026
Available online 3 April 2026

et al., 2021a). Yet, most studies have focused on single exposures, limiting understanding of the joint relationships between complex prenatal environments and neonatal lipid profiles. The exposome framework provides a complementary approach to systematically assess multiple external exposures and internal biological responses across the life course. The external exposome captures environmental influences, while the internal exposome reflects downstream molecular signatures that may represent proximal biological pathways through which these exposures become biologically embedded (Chadeau-Hyam et al., 2011).

Despite its relevance for studying early-life metabolic programming, comprehensive exposome-wide investigations of cord blood cholesterol are lacking. Therefore, this study addresses this gap by examining the association between the external and internal exposome and cord blood cholesterol. Assessing mediation by internal exposures enables investigation of biological pathways through which prenatal environmental factors may influence neonatal lipid metabolism. Thus, we examined whether internal exposures mediate the observed relationships between external exposures and cord blood cholesterol, thereby providing insight into potential biological mechanisms linking the prenatal environment to early-life metabolic programming.

## 2. Methods

ENVIRonmental influence ON early AGEing (ENVIR*ON*AGE) is a mother–child cohort established in 2010 at the East-Limburg Hospital (Genk, Belgium) with ongoing recruitment of more than 2,000 singletons (Janssen et al., 2017). ENVIR*ON*AGE aims to investigate the interaction between environmental or lifestyle factors (air pollution, nutrition, etc.), molecular markers of ageing (mitochondrial function, telomere length, etc.), and clinical outcomes (cardiovascular function, cognitive performance, etc.) from birth through childhood. The cohort recruits mother–child pairs during delivery. Inclusion criteria are informed consent and the ability to complete questionnaires in Dutch. Medical records of the pregnancy, including obstetric information, are retrieved from the hospital, and lifestyle factors during pregnancy are derived from questionnaires filled out by the mothers after delivery. Demographic and lifestyle characteristics of the ENVIR*ON*AGE birth cohort participants are similar to those of the population in Flanders (Janssen et al., 2017). The study follows the Declaration of Helsinki, and ethical approval was granted by Hasselt University and East-Limburg Hospital (EudraCT B371201216090). For this study, 1,732 participants, recruited between January 2010 and March 2020, were selected based on the availability of at least one cholesterol measurement and one exposome variable.

### 2.1. Biospecimen collection

Umbilical cord blood was collected immediately after delivery using BD Vacutainer® plastic tubes with spray-coated K2EDTA. Samples were centrifuged at 3,200 rpm for 15 min, and plasma was aliquoted and stored for subsequent measurement of cholesterol and internal exposome biomarkers.

### 2.2. Cholesterol

TC, HDL-C, and LDL-C were measured in cord blood plasma using a Cobas 8000 C702 analyzer (Roche, Basel, Switzerland) and expressed in mg/dL. Non HDL-C was calculated as the difference between TC and HDL-C.

### 2.3. Exposome

The exposome was constructed using all the prenatal exposures available within the ENVIR*ON*AGE cohort (Janssen et al., 2017). Exposures were systematically screened according to predefined eligibility criteria (Supplemental Methods), including insufficient completeness, repeated measures, sparse category distributions, or high intercorrelation ($r > 0.90$). After application of these criteria, a total of 104 exposures were retained for the present study and categorized into two domains:

1) external exposome: including 90 factors related to the *in utero* environment. These exposures were selected to capture key dimensions of the prenatal exposome, including: air pollution, built environment, diet, green space, indoor household characteristics, lifestyle, meteorology, socioeconomic, obstetric, and health characteristics, traffic, traffic noise and water disinfectant by-products.
2) internal exposome: including 14 biomarkers that were selected based on their relevance to key physiological systems involved in prenatal development. These biomarkers were measured in cord blood at delivery, reflecting the newborn's internal environment.

#### 2.3.1. External exposome

The external exposome included 90 variables (Table 1). Full details on the characterization and measurement methods of each subdomain of the exposome are provided in the Supplemental Methods. In brief, exposure assessment was conducted using a combination of geographic information systems (GIS), hospital-based medical records, and questionnaire data completed by the mothers after delivery. Specifically, GIS-based exposure data were derived from maternal address information. Maternal addresses were collected through a self-completed maternal questionnaire, capturing details about the address at the time of delivery and the duration of residence at that location. For mothers who changed addresses during pregnancy, additional information was obtained, including previous addresses and the dates of relocation. Exposure estimates were then calculated, accounting for address changes during the relevant periods to ensure accurate assessments.

#### 2.3.2. Internal exposome

Fourteen internal biomarkers (**Excel Table S1**) were measured in cord blood plasma using a Cobas 8000 C702 analyzer (Roche, Basel, Switzerland) at the East-Limburg Hospital (Genk, Belgium). These included markers of the thyroid axis (thyroid-stimulating hormone [TSH], free triiodothyronine [fT3], and free thyroxine [fT4]), kidney function (cystatin C), immune response (interleukin-6 [IL-6]), and metabolic processes (ferritin, homocysteine, glucose, insulin, and gamma-glutamyl transferase [GGT]). Additional biomarkers included those related to vitamin status (vitamin D) and reproductive hormones (estradiol [E2], progesterone, and sex hormone-binding globulin [SHBG]). These biomarkers were considered part of the internal neonatal exposome (herein referred to as the internal exposome), as they captured biological responses to prenatal exposures.

### 2.4. Covariates

Child sex, gestational age (in weeks, estimated based on the last menstrual cycle and, if not available, on the first fetal ultrasound examination), and birthweight (in grams) were obtained from obstetric data. Information on child ethnicity (non-European if the country of origin of 3 or more grandparents was non-European, otherwise European) was obtained from the maternal questionnaire completed at birth. Grandparental origin was used because parental country of origin does not capture ethnicity, and this approach provided greater discriminatory capacity to capture ancestry and migration background. However, this classification may introduce some misclassification, particularly in families with mixed ancestry (e.g., 2 European and 2 non-European grandparents), where the threshold-based definition may not fully capture underlying genetic or sociocultural heterogeneity. Cholesterol levels (TC, HDL-C, and LDL-C) were measured in maternal blood two to three days after delivery using the same method described above for

**Table 1**
Description of the exposures included in the external exposome (N = 90).

| Subdomain | N exposures | Exposures | Exposure assessment |
|---|---|---|---|
| Air pollution | 5 | Average BC, $NO_2$, $PM_{2.5}$, $PM_{10}$, $O_3$ ($mg/m^3$) maternal exposure during pregnancy | GIS linkage to dispersion model |
| Built environment | 8 | Urban, suburban and rural location of the maternal residence during pregnancy. Population density (inhabitants per $km^2$), building density (total built area in $m^2$ per $km^2$), street connectivity (number of road intersections per $km^2$), total length in meters of bus lines and number of bus stops per $km^2$, as well as facility richness and facility density* around 300 m from maternal residential address during pregnancy | GIS linkage to green and European maps |
| Diet | 12 | Any maternal consumption of coffee, tea, and soft drinks (e.g., cola) and frequency* (glasses/week) during pregnancy. Frequency of consumption of eggs, vegetables and fish consumed by the mother during pregnancy sourced from the mother's or family, friends, or neighbours. Any maternal use of non-stick cookware with damaged coatings. | General questionnaire |
| Green space | 6 | Average high, low and total green space and agricultural space and normalized difference vegetation index, and enhanced vegetation index at 300 m buffer from the maternal's residential address during pregnancy | GIS linkage to green local and European maps |
| Indoor household characteristics | 10 | Heating methods (gas, electric, wood, pellet or other kind of stove used for heating, having a central heating tank inside or outside the house), ventilation method (passive, mechanical ventilation system C or D, problems with ventilation), any presence of humidity and mold in the household. | General questionnaire |
| Lifestyle | 12 | Any maternal tobacco active smoking and number of cigarettes smoked during* and before the pregnancy*. Any maternal second hand smoking exposure during the pregnancy. Number of years of smoking*, lifetime number of the total packs of cigarettes*. Any alcohol consumption and number of glasses per week the mother consumed before* and during the pregnancy*. Maternal physical exercise during pregnancy (none or once or more than once per week). | General questionnaire |
| Meteorology | 7 | Average temperature (°C), relative humidity (%), precipitation (mm), pressure (hPA), wind speed (km/h), and sunshine (hours) and average UV maternal exposure during pregnancy | GIS linkage to local weather station data |
| Socioeconomic | 5 | Maternal neighbourhood socioeconomic status during the pregnancy, based on annual household income per statistical sector in the Belgium country for the year of 2014. Number of adults (> 18 years) or children* (< 18 years) in the house, maternal educational achievement (categorized by ISCE-21 into three levels) and maternal occupation (coded according to the ISCO-88) at birth. | GIS linkage to Belgian statistical data, general questionnaire |
| Obstetric and health characteristics | 20 | Maternal age at delivery (in years), season at conception (winter, spring, summer and autumn*), type of delivery (natural or caesarean section), parity (primipara, secondipara or multipara), maternal pre-pregnancy BMI ($Kg/m^2$) and weight gain during pregnancy (Kg), type of conception (natural, hormonal stimulation, IVF, IUI and ICSI), assumption of drugs (e.g., paracetamol and antibiotics), folic acid and vitamins during pregnancy, maternal chronic diseases (e.g., tachycardia, infectious diseases, hypertension, thyroid diseases, asthma and allergies), and diseases during pregnancy (preeclampsia and gestational diabetes) | Obstetric data |
| Traffic | 2 | Distances from maternal residence to the nearest major road (m) with available traffic counts and traffic weighted density in 100-m buffer from maternal residence | GIS linkage to local road maps |

(*continued on next page*)

**Table 1** (*continued*)

| Subdomain | N exposures | Exposures | Exposure assessment |
|---|---|---|---|
| Traffic noise | 1 | Average maternal 24-h residential traffic noise exposure during pregnancy (dB(A)) | GIS linkage to European noise maps |
| Water disinfectant by-products | 2 | The maternal habit of swimming in pools, as possible exposure to disinfectant by-products, and the number of times the mothers swam in pools during pregnancy* | General questionnaire |

* variables (N = 12) excluded from the analysis due to high collinearity (r > 0.99).
GIS = geographic information system; IVF = in vitro fertilization; IUI = intrauterine insemination; ICSI = intracytoplasmic sperm injection; ISCE-21 = international standard classification of education 2021; ISCO-88 = international standard classification of occupations 1988.

cord blood. Additionally, maternal non HDL-C levels were calculated as the difference between TC and HDL-C.

### 2.5. Statistical analysis

Prior to statistical analysis, cholesterol levels, external exposome, internal exposome, and covariates underwent the following preprocessing steps to ensure data quality and suitability for analysis. Categorical variables with more than two levels were transformed into dummy variables. This process generated 35 dummy variables, expanding the pregnancy external exposures from 90 to 125 (**Excel Table S2**). Ordinal categorical variables were retained as continuous to preserve rank information. Continuous variables with > 80% zero observations were dichotomized to reduce zero inflation and improve model stability. Outliers, defined as values exceeding ± 5 standard deviations from the mean, were winsorized, using the *datawizard* R package, to limit the influence of extreme values while retaining observations. Continuous variables were transformed to approximate normality using the *bestNormalize* R package, which selected the optimal transformation based on the Pearson P statistic divided by its degrees of freedom. Transformed variables were subsequently centered to the mean and standardized to unit variance. If normality could not be achieved (Kolmogorov-Smirnov test p-value < 0.05), variables were dichotomized at the median to enable inclusion in downstream analyses. Correlation analyses were conducted using the *polycor* R package. Pearson, polychoric, and polyserial correlations were calculated: (1) between cholesterol measurements and covariates (**Fig. S1**), (2) between external exposome variables (**Fig. S2**), and (3) between internal exposome variables (**Fig. S3**). Heatmaps were generated to visualize these correlations. To identify near-identical or highly redundant variables, pairs with absolute correlation coefficients ($r$) > 0.99 were identified, and one variable from each pair was excluded based on interpretability and data completeness. This process was not applied to dummy variables derived from the same underlying categorical variable. After preprocessing, 113 external exposome variables and all 14 internal biomarkers were retained for analysis. No other variables in the internal exposome or covariates were excluded. A detailed preprocessing workflow is provided in **Fig. S4**.

Descriptive statistics for the study population were presented as mean ± standard deviation for continuous variables and as counts (percentages) for categorical variables. Missing data in the external exposome, internal exposome, and covariates were imputed using the *mice* R package. Twenty imputed datasets were generated, with external exposome, internal exposome, covariates, and outcome variables used as predictors in the imputation models.

#### 2.5.1. Analysis of the external exposome and cord blood cholesterol

The association between the external exposome and each of the cord blood cholesterol variables was examined through a three-step analysis. Briefly, step 1 enabled broad screening, step 2 refined the variable selection based on predictive performance, and step 3 estimated adjusted associations.

(i) In step 1, exposome-wide association studies (ExWAS) were conducted using the *rexposome* R package. ExWAS was used as a hypothesis-free screening approach to efficiently identify candidate exposures associated with cholesterol outcomes across the high-dimensional exposome. Linear regression models were fitted to assess the association between each external exposome variable (independent variable) and cord blood cholesterol measurements (dependent variable) across all 20 imputed datasets. Results were pooled using Rubin's rules for multiple imputation, yielding a single association estimate and a single p-value for each exposome variable. To account for multiple testing, a significance threshold of $p = 6.43 \times 10^{-04}$ was applied, calculated as 0.05 divided by the effective number of independent tests (N = 78), estimated based on the correlation structure of the p-values (Li et al., 2012). The effective number of independent tests approach was used instead of a standard Bonferroni correction to account for correlation among external exposures and to maintain adequate power for hypothesis-free exposome screening. Manhattan plots were generated to visualize the ExWAS results.

(ii) In step 2, exposures identified in the ExWAS were evaluated using the deletion/substitution/addition (DSA) algorithm (Sinisi and van der Laan, 2004), implemented in the *DSA* R package. The DSA algorithm was applied to reduce false-positive findings of ExWAS and identify a parsimonious subset of exposures with strong joint predictive performance. The DSA algorithm aimed to determine the regression model that minimizes the root mean squared error of prediction using 5-fold cross-validation. DSA was applied to the 20 imputed datasets stacked together, with each dataset weighted equally, in order to perform variable selection across imputations while avoiding arbitrary reconciliation of selection results obtained separately within each imputed dataset. Importantly, this stacked approach was used solely for exposure selection. To improve the robustness of the selection procedure, DSA was run 50 times. Exposures with ranks lower than or equal to the best cross-validated cut-off value in more than 10% of the runs (N = 5) were retained.

(iii) Finally, all exposures selected by the DSA algorithm were included in a multi-exposure linear regression model. Multi-exposure regression models were fitted to estimate mutually adjusted associations and account for correlation and confounding between co-occurring environmental exposures. Associations were considered statistically significant at $p < 0.05$. Forest plots were generated to visualize the results.

The combined use of ExWAS and DSA was based on a previous exposome simulation study, which identified ExWAS as the method with the highest sensitivity (96%), albeit with a higher false positive rate (86%), while DSA demonstrated the lowest false positive rate (28%), yet maintained good sensitivity (73%) (Agier et al., 2016). Covariates included in the models were visualized in a directed acyclic graph (DAG, **Fig. S5A**) using dagitty (Textor et al., 2016). All the analyses (ExWAS, DSA, and multi-exposure models) were adjusted for newborn sex and ethnicity. Additionally, analyses of HDL-C, LDL-C, and non HDL-C were adjusted for TC to account for the physiological dependency between lipid subfractions and TC concentration, thereby isolating fraction-specific associations independent of TC.

*Sensitivity analyses*

To assess the robustness of the identified associations, we conducted several sensitivity analyses. Multi-exposure models, adjusted for

newborn sex, ethnicity, all exposures identified by DSA for the specific cholesterol measurement, and TC for the HDL-C and LDL-C models, were stratified by newborn sex to investigate potential sex-specific associations. The analyses were also repeated by excluding preterm births (gestational age < 37 weeks) to account for potential bias related to immature fetal lipid metabolism associated with shorter gestation, and excluding caesarean-section deliveries to account for potential bias due to differences in perinatal stress response and early microbial exposure between delivery modes.

#### 2.5.2. Analysis of the internal exposome and cord blood cholesterol

Since internal exposome biomarkers and cholesterol levels were measured simultaneously in the same biological matrix (cord blood), bidirectional relationships are possible. To account for this, we analyzed the internal exposome separately from the external exposome, the latter being based on prenatal exposures and therefore temporally preceding the outcome. We calculated partial Pearson correlations via the ppcor R package between cord blood cholesterol and internal exposome variables, adjusting for covariates identified in the DAG shown in **Fig. S5B** (newborn sex, ethnicity, birthweight, gestational age, TC for HDL-C, LDL-C, and non HDL-C correlations, and the corresponding maternal cholesterol levels). r values were first calculated within each imputed dataset, transformed to a normally distributed z-score via Fisher's z-transformation, pooled using Rubin's rules, and then retransformed back to the original r-scale coefficient after pooling. We considered significant correlations with a p-value below $8.93 \times 10^{-04}$, which was determined using a Bonferroni correction to account for multiple testing obtained by dividing the threshold of 0.05 by the number of computed correlations (N = 56). This conservative approach was applied given the limited number of internal biomarkers analyzed (N = 14) and the targeted nature of the correlation analysis, providing robust control of the family-wise error rate. This different multiple testing correction strategy was applied compared with the external exposome analysis, which used the effective number of independent tests approach, because the two domains differed in analytical objective and dimensionality.

*Sensitivity analyses*

To assess the robustness of the identified correlations, sensitivity analyses were conducted following the same strategy applied to the external exposome analyses. In detail, analyses were: (i) stratified by newborn sex, (ii) repeated after exclusion of preterm births (gestational age < 37 weeks), and (iii) repeated after exclusion of caesarean-section deliveries.

#### 2.5.3. Mediation analyses

We evaluated the possible mediating effect of internal biomarkers, identified in the internal exposome analyses, in the associations between external exposures, identified in multi-exposure models, and cholesterol levels, via the imputation approach implemented in the *medflex* R package (Vansteelandt et al., 2012). This framework assumes that external exposures may influence cholesterol through intermediate biological pathways reflected by internal biomarkers. However, given the simultaneous measurement of internal biomarkers and cholesterol in cord blood, temporality cannot be established and bidirectional relationships are possible. Consequently, these mediation analyses should be interpreted as exploratory and hypothesis-generating, providing insight into potential biological pathways rather than confirming causal mechanisms. Mediation analyses were performed using the 20 imputed datasets and estimates were pooled using Rubin's rules via the *mitools* R package. The total effect (TE) was decomposed into the natural indirect effect (NIE) and the natural direct effect (NDE). We considered NIE to be significant if 95% confidence intervals (95% CI) did not include 0. The 95% CI threshold was determined using a Bonferroni correction to account for multiple testing, calculated as 0.05 divided by the total number of mediation analyses performed (N = 59). Based on the DAGs displayed in **Fig. S5**, mediation analyses were adjusted for sex and ethnicity. Since gestational age, birthweight, and the corresponding maternal cholesterol measurements are mediators in the pathway between external exposures and cholesterol, but also potential confounders for the association between internal exposures and cholesterol, they were included as covariates in the imputation step but not in the final regression model.

## 3. Results

The study population included 1,732 mother–child pairs (Table 2). A minority of the children were of non-European ethnicity (17.2%). Sex distribution was nearly equal (49.1% were girls), with a mean gestational age of 39.2 weeks and an average birthweight of 3,422.1 g. For cord blood, the mean TC was 66.0 mg/dL, HDL-C was 30.0 mg/dL, LDL-C was 22.3 mg/dL, and non HDL-C was 36.0 mg/dL (Table 2 and **Fig. S1**). The mean maternal TC was 219.2 mg/dL, LDL-C was 115.8 mg/dL, HDL-C was 64.9 mg/dL, and non HDL-C was 154.2 mg/dL. Cord blood cholesterol levels were significantly higher in girls than in boys (*t*-test p-value < 0.01, **Excel Table S3**). Maternal and cord blood cholesterol levels were strongly correlated within mothers (absolute *r* range = 0–0.94) and newborns (absolute *r* range = 0.20–0.82) but weakly correlated between them (absolute *r* range = 0.02–0.12) (**Fig. S1**). External exposome variables had a mean *r* of 0.11 (absolute *r* range = 0–0.99), with correlations being stronger within exposome subdomains (**Fig. S2**), while internal exposome variables had a mean *r* of 0.10 (absolute *r* range = 0–0.48) (**Fig. S3**).

### 3.1. External exposome

First, ExWAS analyses identified 14 unique exposures associated with cord blood cholesterol (p-values < $6.43 \times 10^{-04}$) among the 113 external exposures considered in the study (Fig. 1, **Excel Table S4**). Two exposures, exposure to sunshine and atmospheric pressure during pregnancy, were associated with TC (Fig. 1**A**). Eleven exposures were associated with HDL-C (Fig. 1**B**): black carbon (BC) and particulate matter smaller than 2.5 μm ($PM_{2.5}$) exposure during pregnancy, presence of a central heating tank inside the house, maternal age at delivery, maternal smoking during pregnancy, maternal smoking status prior to pregnancy (non-smokers and ex-smokers), sunshine exposure during pregnancy, folic acid supplementation during pregnancy, primiparity and secundiparity. No exposure was significantly associated with LDL-C (Fig. 1**C**). 14 exposures were associated with non HDL-C (Fig. 1**D**): BC, nitrogen dioxide ($NO_2$), ozone ($O_3$) and $PM_{2.5}$ exposures during pregnancy, presence of a central heating tank inside the house, maternal age at delivery, maternal smoking during pregnancy, maternal smoking status prior to pregnancy (non-smoker and ex-smoker), exposure to sunshine and atmospheric pressure during pregnancy, folic acid supplementation during pregnancy, primiparity and secundiparity.

In step 2, DSA selected 9 exposures (**Excel Table S5**). All exposures

**Table 2**
Descriptive characteristics of the study population (N = 1,732).

| Characteristics | N (%) or mean ± SD | % missings |
|---|---|---|
| sex, *girls* | 828 (49.1) | 2.7 |
| gestational age, *weeks* | 39.2 ± 1.3 | 2.7 |
| birthweight, *grams* | 3422.1 ± 466.7 | 2.0 |
| ethnicity, *non-European* | 279 (17.2) | 6.3 |
| cord blood TC (mg/dL) | 66.0 (18.8) | 0.5 |
| cord blood HDL-C (mg/dL) | 29.1 (10.2) | 0.2 |
| cord blood LDL-C (mg/dL) | 22.3 (9.3) | 1.4 |
| cord blood non HDL-C (mg/dL) | 36.0 (14.3) | 0.7 |
| maternal blood TC (mg/dL) | 219.2 (52.4) | 14.8 |
| maternal blood HDL-C (mg/dL) | 64.9 (17.6) | 14.9 |
| maternal blood LDL-C (mg/dL) | 115.8 (42.1) | 14.8 |
| maternal blood non HDL-C (mg/dL) | 154.2 (49.3) | 14.9 |

The table shows descriptive statistics for unimputed and untransformed data. BMI = body mass index; HDL-C = high-density cholesterol; LDL-C = low-density cholesterol; SD = standard deviation; TC = total cholesterol.

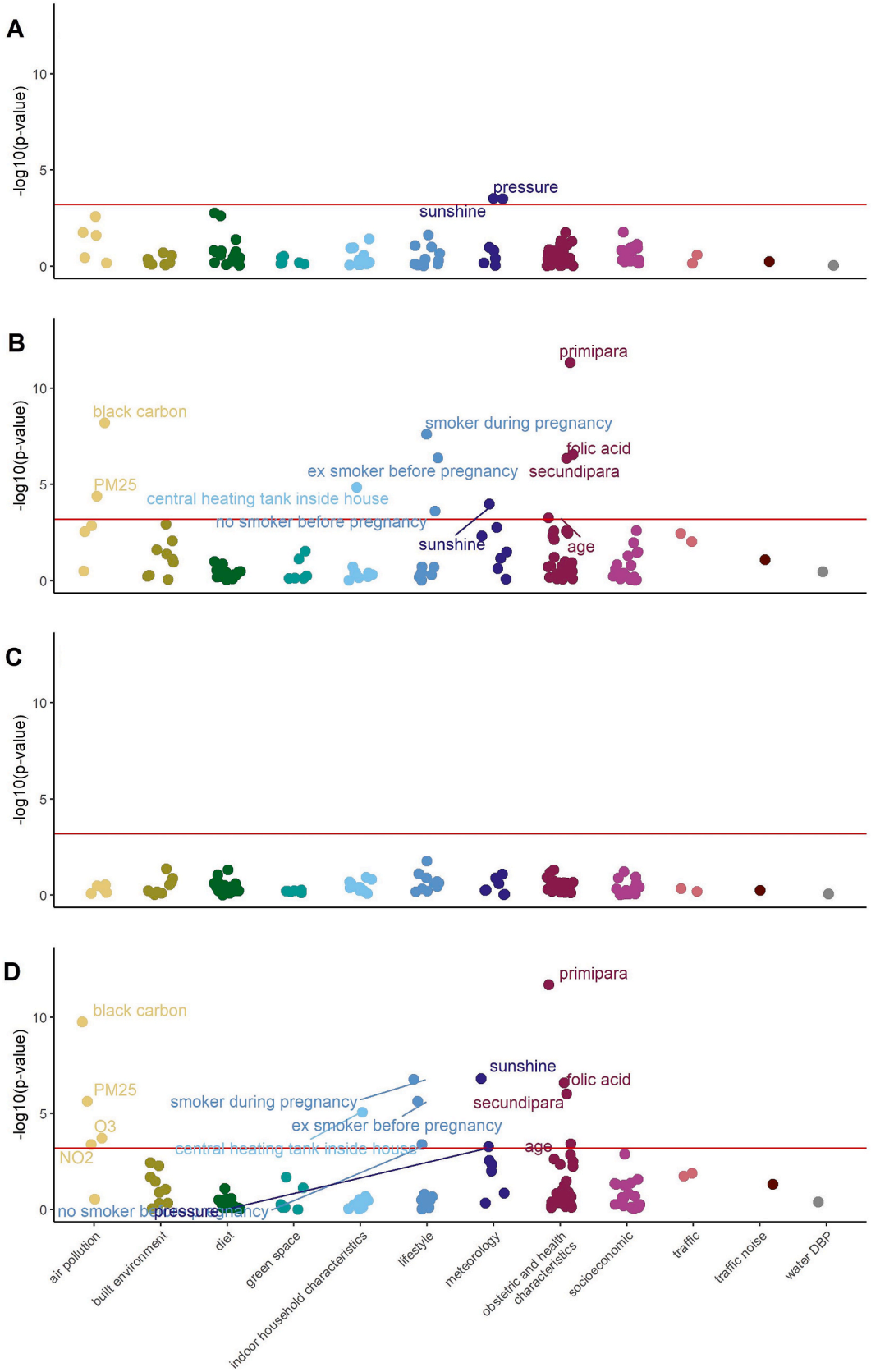


**Fig. 1. Manhattan plots of exposome-wide association studies (ExWAS) for cord blood cholesterol.** Manhattan plots illustrating the ExWAS results for cord blood: (**A**) total cholesterol (TC), (**B**) high-density lipoprotein cholesterol (HDL-C), (**C**) low-density lipoprotein cholesterol (LDL-C) and (**D**) non HDL-C. Each point represents the $-\log_{10}$ p-value of the association between an exposure and cholesterol, grouped by exposure subdomain along the x-axis. The horizontal line indicates the significance threshold (p-value = 6.43 x $10^{-04}$), calculated as 0.05 divided by the effective number of tests (n = 78). Labelled points represent exposures significantly associated with cholesterol levels. All models are adjusted for newborn ethnicity, sex, and TC for HDL-C and LDL-C models.

identified by ExWAS of TC were also retained by the DSA algorithm. Six out of the 11 exposures identified by ExWAS of HDL-C were retained by DSA: BC exposure during pregnancy, presence of a central heating tank inside the house, maternal smoking during pregnancy, maternal non-smoking status prior to pregnancy, folic acid supplementation during pregnancy, and primiparity. Two out of 14 exposures identified by ExWAS of non HDL-C were retained by DSA: sunshine exposure during pregnancy and primiparity.

Associations of the exposures retained by DSA with cholesterol were tested in step 3 via multi-exposure linear models. In multi-exposure linear models, a 1 SD increase in exposure to sunshine and atmospheric pressure during pregnancy was associated with a decrease of −1.31 (95% CI: −2.23, −0.38) and −1.27 (95% CI: −2.18, −0.36) mg/dL of TC, respectively (Fig. 2**A**). 5 out of the 6 exposures selected by DSA for HDL-C were associated with HDL-C (Fig. 2**B**). A 1 SD increase in BC exposure during pregnancy, presence of a central heating tank inside the house, maternal smoking during pregnancy, and primiparity was associated with a decrease of −0.75 (95% CI: −1.11, −0.39), −1.37 (95% CI: −2.08, −0.65), −2.50 (95% CI: −3.74, −1.26), and −2.64 (95% CI: −3.32, −1.95) mg/dL of HDL-C, respectively, while folic acid supplementation during pregnancy was associated with an increase of 1.88 (95% CI: 1.17, 2.60) mg/dL of HDL-C. Finally, primiparity was associated with an increase of 2.80 (95% CI: 2.04, 3.56) mg/dL of non HDL-C and exposure to sunshine with a decrease of −1.06 (95% CI: −1.45,-0.68) mg/dL of non HDL-C (Fig. 2**C**).

Sensitivity analyses of the multi-exposure models revealed that some of the associations were more pronounced in boys, while further stratification excluding preterm births or caesarean-section deliveries did not materially alter the main results (Fig. S6). For TC, inverse associations with atmospheric pressure and sunshine exposure were observed in boys but not in girls. In boys, atmospheric pressure (β = -1.23, 95% CI: −2.16, −0.31) and sunshine exposure (β = -1.24, 95% CI: −2.18, −0.31) were associated with lower TC. In contrast, in girls, effect estimates were similar in direction but weaker and not statistically significant (for atmospheric pressure: β = -1.17, 95% CI: −2.46, 0.12; and for sunshine: β = -1.14, 95% CI: −2.49, 0.21). For HDL-C, smoking during pregnancy was inversely associated in boys (β = -2.55, 95% CI: −3.80, −1.31), but not in girls (β = -1.43, 95% CI: −3.17, 0.31). Folic acid supplementation was positively associated with HDL-C in boys (β = 1.43, 95% CI: 0.70, 2.15) but not in girls (β = 0.60, 95% CI: −0.36, 1.56).

### 3.2. Internal exposome

Thirteen out of the 14 internal exposome variables were correlated with cholesterol levels (p-values < 8.93 x $10^{-04}$). Nine biomarkers were positively associated with TC: cystatin C ($r$ = 0.13), ferritin ($r$ = 0.16), fT4 (r = 0.12), GGT ($r$ = 0.21), homocysteine ($r$ = 0.15), progesterone ($r$ = 0.16), SHBG ($r$ = 0.10), TSH ($r$ = 0.08), and vitamin D ($r$ = 0.10). Five biomarkers were correlated with HDL-C: insulin (r = 0.13) and GGT ($r$ = 0.11) positively, IL-6 ($r$ = -0.12), fT3 ($r$ = -0.15), and E2 ($r$ = -0.11) negatively. Ferritin ($r$ = -0.18) and fT4 ($r$ =-0.09) were negatively correlated with LDL-C. Eight biomarkers were correlated with non HDL-C: fT3 ($r$ = 0.15), IL-6 ($r$ = 0.17), E2 ($r$ = 0.13), progesterone ($r$ = 0.10)

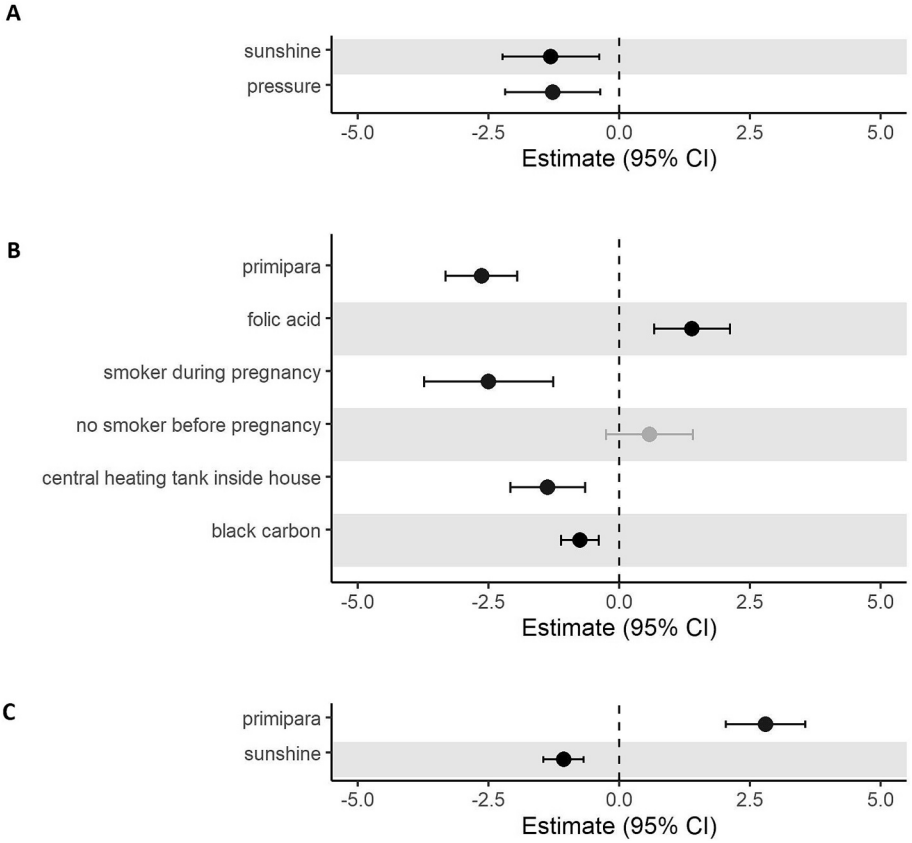


**Fig. 2. Forest plots of multi-exposure model associations with cord blood cholesterol.** Forest plots showing the regression estimates and 95% confidence intervals (95% CI) for associations of exposures and cord blood (**A**) total cholesterol (TC), (**B**) high-density lipoprotein cholesterol (HDL-C) and (**C**) non HDL-C in multi-exposure models. Estimates represent the change in cholesterol levels (mg/dL) per standard deviation increase in exposure (for continuous variables) or per specific exposure category (for categorical variables). Only exposures commonly identified in the Exposome-Wide Association Studies (ExWASs) and the deletion, substitution and addition (DSA) are shown. Grayed estimates and 95% CI indicate non-significant associations (p-value > 0.05). P-values are not adjusted for multiple comparisons. All models are adjusted for newborn ethnicity, sex, and TC for the HDL-C and non HDL-C model along with all the exposures identified for the specific cholesterol measurement.

and SHBG ($r$ = 0.09) positively, and ferritin ($r$ = -0.12), fT4 ($r$ = -0.08) and insulin ($r$ = -0.12) negatively (Fig. 3).

Sensitivity analyses supported the primary findings, with the direction of correlations remaining consistent across models. However, the correlations between TSH and TC, and between fT4 and LDL-C and non-HDL-C did not remain significant in boys. Similarly, correlations between vitamin D and TC, and GGT and HDL-C in girls lost significance. Additionally, when excluding caesarean-section deliveries, the correlation between fT3 and non-HDL-C was no longer significant (**Fig. S7**).

We tested a total of 59 mediation paths (full results shown in **Excel Table S6**). In mediation analysis, 4 internal exposures were found to mediate the effect of 3 external exposures on TC and HDL-C. In detail, the effect of maternal exposure to pressure during pregnancy on TC (TE = -1.44) was mediated by cord blood homocysteine (NIE = -0.34, 95% CI: −0.66, -0.01). The effect of folic acid supplementation during pregnancy on HDL-C (TE = 1.87) was mediated by fT3 (NIE = 0.40, 95% CI: 0.03, 0.77); the effect of maternal smoking during pregnancy on HDL-C (TE = -2.95) was mediated by insulin (NIE = -0.62, 95% CI: −1.18, -0.07), fT3 (NIE = -0.73, 95% CI: −1.31, -0.15), E2 (NIE = -0.61, 95% CI: −1.14, -0.07) and IL-6 (NIE = -0.64, 95% CI: −1.20, -0.07); and the effect of primiparity on HDL-C (TE = -2.38) was mediated by insulin (NIE = -0.39, 95% CI: −0.74, -0.04) (Fig. 4).

## 4. Discussion

Our findings offer valuable insights into the factors influencing offspring lipid profiles at birth and reveal possible mechanisms underlying cardiometabolic health risks in later life.

Since cholesterol levels at birth are predictive of cholesterol levels later in life (Juhola et al. 2011), understanding the determinants of early-life lipid profiles is crucial for guiding preventive strategies. Although up to 59% of the variability in cholesterol levels is heritable, a substantial modifiable fraction remains (Cadby et al. 2020), leaving significant scope for preventive strategies. A recent study estimated that lifelong reduction of LDL-C by 40 mg/dL could result in a 49% decrease in coronary heart disease (Dron et al. 2023), highlighting the potential long-term benefits of early interventions.

While cholesterol levels at birth are not routinely measured, previous literature suggests that they are low at delivery (Øyri et al. 2021a; Strandkjær et al. 2022) and reach adult levels between two and six months of life (Øyri et al., 2021b; Taageby Nielsen et al. 2023). Consistent with these findings, our study also showed that cord blood cholesterol levels were lower than maternal levels at delivery. While some correlation is expected due to the heritability of lipid traits (Cadby et al. 2020; Teslovich et al. 2010; Willer et al. 2013) and placental cholesterol transfer, the relatively low correlation we observed between cord blood and maternal cholesterol levels supports the hypothesis that distinct mechanisms govern cholesterol regulation in newborns as pregnancy progresses (Horne et al. 2019; Øyri et al. 2021a). This suggests that cord blood cholesterol levels are not merely a reflection of maternal levels but are influenced by unique fetal physiological processes.

Cord blood HDL-C was the cholesterol measure associated with most external exposures, followed by TC and non HDL-C, while no exposure was associated with LDL-C. This may be attributed to the predominance of HDL-C in cord blood (Stadler et al. 2021), in contrast to adults, in which LDL-C is the most abundant lipoprotein (Goldstein and Brown 1987). However, this observation may also reflect functional differences in lipoprotein metabolism between newborns and adults. For instance, proteomic studies have revealed distinct apolipoprotein concentrations in cord blood HDL-C compared to adults, indicating potential attenuation of anti-oxidative activity in newborns (Sreckovic et al. 2013; Stadler et al. 2021).

Parity emerged as a key external factor, associated with both HDL-C and non HDL-C. Previous studies have reported lower levels of cord blood TC in children born to multiparous compared to nulliparous mothers (Go et al., 2023). Similarly, we found that newborns of first-time mothers had lower HDL-C and higher non-HDL-C levels compared to those born to multiparous mothers. This association may reflect maternal physiological changes due to previous pregnancies, such as fluctuations in oestrogen, inflammation markers and insulin (Ezeigwe et al. 2022; Kazzi et al. 2022; Motevalizadeh et al. 2024),

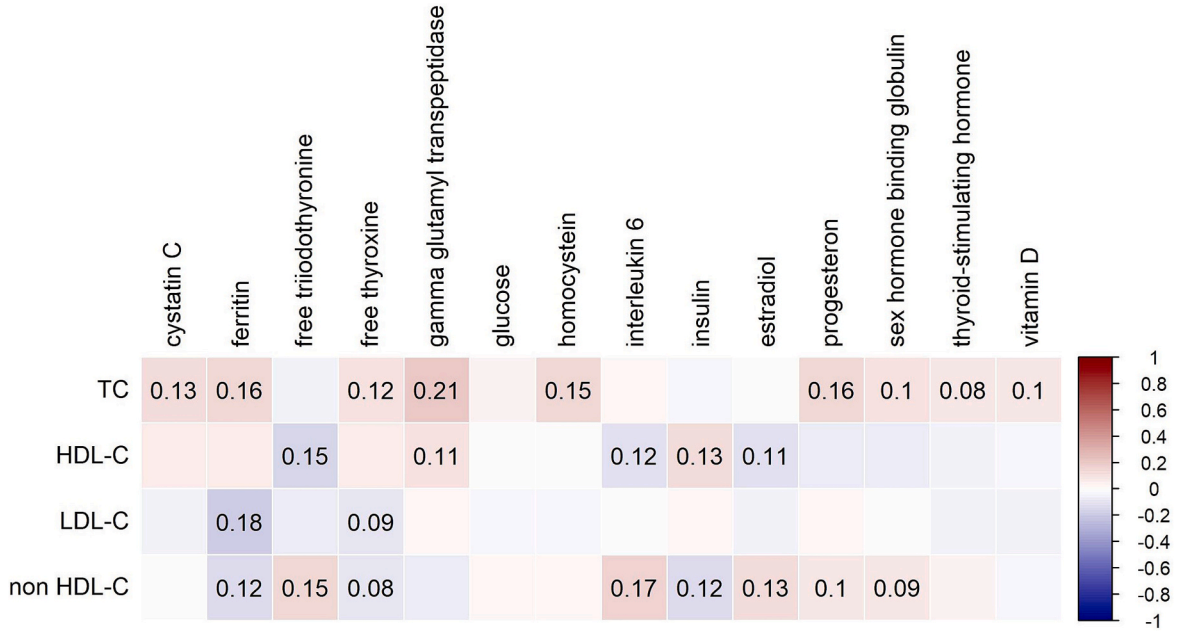


**Fig. 3. Heatmap of internal exposome correlations with cord blood cholesterol.** Heatmap illustrating Pearson partial correlation coefficients between the 14 internal exposome variables and cord blood cholesterol levels. Correlations are adjusted for newborn sex, ethnicity, birthweight, gestational age, total cholesterol (TC) for correlations involving high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C) and non HDL-C, and corresponding maternal cholesterol levels. Numbers indicate correlation coefficients and are reported if p-value was below the threshold of 8.93 x 10e-04, calculated by dividing 0.05 for the number of correlation computed (N = 56).

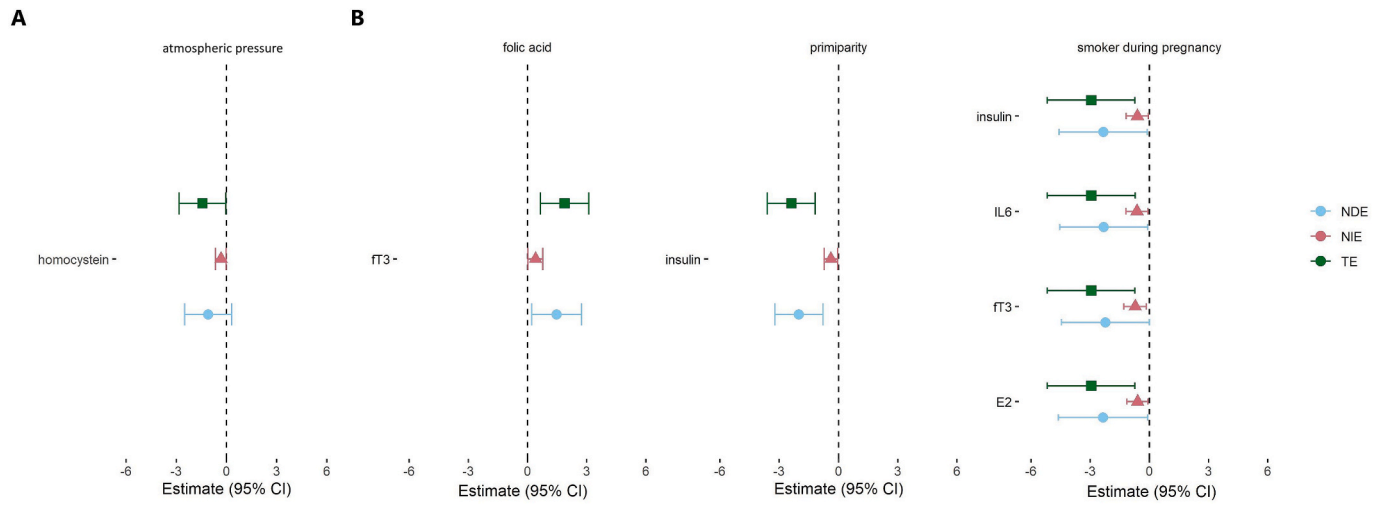


**Fig. 4. Forrest plots of mediation analyses.** Forest plots showing the estimates and 95% confidence intervals (95% CI) of total effects (TE), natural indirect effects (NIE) and natural direct effects (NDE) of external exposures identified by multi-exposures models on cord blood (A) total cholesterol (TC) and (B) high-density lipoprotein cholesterol (HDL-C) via internal exposures identified by correlations analyses. Only internal exposures demonstrating NIEs with 99% CI excluding the null value are presented. Mediation analyses were adjusted for sex and ethnicity and TC (for analysis of HDL-C). Gestational age, birthweight and corresponding maternal cholesterol levels were included as covariates in the imputation step but not in the final regression model, since they can be considered mediators in the pathway between external exposures and cholesterol, but also potential confounders for the association between internal exposures and cholesterol.

which may influence lipid metabolism. In line with this hypothesis, we identified insulin as a mediator in the relationship between primiparity and HDL-C. Furthermore, family lifestyle adaptations related to pregnancy and child-rearing could also play a role.

Two meteorological factors were associated with cholesterol levels. Atmospheric pressure during pregnancy was inversely associated with TC. While studies investigating atmospheric pressure directly and lipid metabolism in pregnancy are limited, studies of high-altitude pregnancy, which is characterised by chronic hypobaric hypoxia, demonstrate that reduced oxygen availability can alter placental function and fetal metabolism (Määttä et al. 2018). These adaptations involve coordinated changes in hematological parameters, oxygen transport capacity, and vascular function (Rangel et al. 2026), providing biological plausibility for pressure-related influences on neonatal lipid profiles. In addition, we identified homocysteine as a potential mediator of this association, suggesting involvement of endothelial or oxidative stress-related pathways. These novel findings, while intriguing, require further validation. Exposure to sunshine during pregnancy was also inversely associated with TC and non HDL-C. A plausible explanation for this association could be a competitive mechanism involving increased vitamin D synthesis in the skin, which shares a precursor (7-dehydrocholesterol) with cholesterol (Kuan et al. 2013). However, we did not observe mediation via vitamin D in our analyses. Alternative mechanisms, independent of vitamin D, such as systemic inflammation or oxidative stress, may be relevant (Slominski et al. 2024). Furthermore, while associations with lipids were identified for sunshine exposure, similar relationships were not observed for ultraviolet radiation indices or seasonality metrics. This suggests that sunshine duration, also referred to as photoperiod, may capture broader environmental and behavioural correlates, including circadian regulation during pregnancy (Méndez et al. 2023). In addition to sunshine, several actionable factors were identified. Some had previously documented effects on cholesterol, such as maternal smoking, which we found associated with lower HDL-C in cord blood, in accordance with a previous study (Işcan et al. 1997). Multiple mechanisms can underlie this association. We identified IL-6, fT3, insulin, and E2 as potential mediators of the effect of smoking during pregnancy on offspring cholesterol. Notably, similar to our study in children, a previous mediation study in adults identified inflammation mediating the effect of smoking on remnant cholesterol (Lai et al. 2024). Other factors, including folic acid supplementation, exposure to BC during pregnancy, and having a central heating tank inside the house, have been less studied at birth in relation to cholesterol. Our results indicated that folic acid supplementation was positively associated with higher HDL-C levels, suggesting potential benefits for newborn lipid profiles beyond neural tube defect prevention (Force 2023). This finding warrants further investigation to explore the broader metabolic effects of folic acid supplementation during pregnancy. Conversely, BC and central heating tank presence inside the house were associated with lower HDL-C. Indoor placement of combustion-based heating systems may increase exposure to indoor air pollutants, including particulate matter and combustion-related by-products. Both outdoor and indoor air pollution have been linked to systemic inflammation and oxidative stress, processes known to influence lipoprotein metabolism in adults (Li et al., 2025; Wang et al., 2023; Zhang et al., 2023). It is therefore plausible that such exposures during pregnancy may already be associated with lipid metabolism at birth.

Surprisingly, maternal BMI, diet, or physical exercise were not identified as crucial factors influencing cholesterol levels at birth in our analysis. Measurement error may have contributed to null findings for self-reported exposures such as diet and physical activity. For maternal BMI, which was objectively measured, a previous study similarly reported no association with cord blood lipid concentrations (Geraghty et al. 2017), and another reported higher cord blood total cholesterol among offspring of obese mothers (Beneventi et al. 2025). These inconsistencies suggest that the relationship between maternal adiposity and neonatal lipid levels may be context-dependent. It is plausible that maternal BMI does not uniformly translate into altered neonatal cholesterol concentrations. Additionally, the relatively narrow distribution of maternal BMI in our cohort may have limited statistical power to detect subtle associations. Finally, it is also possible that maternal adiposity has limited direct influence on conventional cord blood cholesterol measures at birth.

Girls had higher levels of cholesterol than boys, consistent with previous literature (Felzer-Kim et al. 2020). However, sex-stratified analyses revealed that the effects of folic acid supplementation, sunshine exposure, atmospheric pressure, and maternal smoking during pregnancy on cholesterol levels were specific to boys, suggesting a potential sex-specific vulnerability to these exposures. This observation aligns with previous studies in children showing sex-modified effects of perfluoroalkyl substances on cholesterol levels (Blomberg et al. 2021),

further supporting the notion that sex is an important modifier of environmental influences on lipid metabolism.

To explore biological mechanisms, we examined correlations with other cord blood biochemical markers. All, except glucose, were correlated with cholesterol. Mediation analyses showed that several of these internal biomarkers act as mediators of the effects of identified external exposures on cholesterol. Interpretation of this result should be cautious since reverse causality is possible. For example, fT3 was found to have a mediation effect in the association between smoking and folic acid supplementation and cholesterol levels; however, a Mendelian randomization study indicated that cholesterol is influencing thyroid hormones and not the opposite (Tan et al. 2025).

Our findings should be interpreted as hypothesis-generating rather than demonstrating clinically meaningful physiological effects at birth. Although several exposures were statistically associated with cord blood cholesterol, the absolute magnitude of most associations was small to modest, generally ranging between approximately 1–3 mg/dL per 1 SD increase in exposure. For example, primiparity was associated with a 2.6 mg/dL reduction in HDL-C, corresponding to approximately 0.25 SDs within the neonatal lipid distribution, indicating a modest shift in relative terms. Clinical dyslipidemia thresholds are not defined at birth, as lipid risk classification begins in early childhood. For contextual comparison only, epidemiologic evidence from adult populations indicates that HDL-C differences on the order of approximately 1 SD (≈10-15 mg/dL) are associated with substantial gradients in coronary heart disease risk in large pooled analyses (Di Angelantonio et al. 2009). Therefore, while the exposure-related differences observed here are unlikely to be clinically meaningful in the neonatal period, they may still be relevant from a developmental programming perspective. Lipid levels track from early life into adulthood, and even modest early-life variations may contribute to long-term cardiometabolic risk trajectories (Juhola et al. 2011). In addition, exposures were operationalised as whole-pregnancy averages within the exposome framework, precluding identification of trimester-specific windows of susceptibility that may differentially influence fetal lipid metabolism. Future hypothesis-driven studies incorporating temporally resolved exposure assessments, more sophisticated analytical approaches (e.g., distributed lag nonlinear model), and longitudinal follow-up are needed to determine whether these modest early-life differences translate into meaningful cardiometabolic risk trajectories.

This study has several strengths. The study population had a large sample size (N = 1,732). We employed a sequential analysis strategy, using ExWAS and DSA to identify relevant exposures, which were then integrated into a multi-exposure model, to utilize the complementary strengths and mitigate the inherent limitations of individual statistical methods. Using an agnostic approach, including a wide range of exposures (N = 104), offered the opportunity for novel discoveries. Sensitivity analyses, excluding preterm and caesarean section deliveries, confirmed the robustness of our findings.

However, there are limitations to acknowledge. Previous studies identified specific genetic associations with infant cord blood lipids (Huang et al. 2024), but in this study, we lacked genotype data to assess genetic influences. Although our focus was on environmental determinants, the absence of genetic information may have resulted in residual confounding for some associations, particularly given the heritability of lipid traits. Given the cross-sectional nature of our anlysis, we cannot exclude reverse causality for some exposures, such as those within the internal newborn exposome. Furthermore, some exposures derived from geographic information systems or maternal questionnaires (e.g., air pollution, physical activity, diet) are subject to measurement error. Such non-differential misclassification would likely bias effect estimates towards the null, potentially underestimating true associations. In our mediation analyses, we included only internal exposures that were significantly associated with cholesterol. While this approach helped focus on relevant pathways, it may have led to the omission of potential mediators. We acknowledge our findings are not causal, but they provide a basis for future studies to explore mechanisms underlying these associations. Future studies employing longitudinal designs and Mendelian randomization approaches could further elucidate causal relationships. Finally, apart from parity and smoking, which were previously associated with cord blood cholesterol levels (Işcan et al., 1997; Go, H.; Hashimoto, K.; Maeda, H.; Ogasawara, K.; Kume, Y.; Murata, T.; Sato, A.; Ogata, Y.; Shinoki, K.; Nishigori, H.; Ikeda-Araki, A.; Fujimori, K.; Yasumura, S.; Hosoya, M.; Kamijima, M.; Yamazaki, S.; Ohya, Y.; Kishi, R.; Yaegashi, N.; Hashimoto, K.; Mori, C.; Ito, S.; Yamagata, Z.; Nakayama, T.; Sobue, T.; Shima, M.; Nakamura, H.; Suganuma, N.; Kusuhara, K.; Katoh, T.; the Japan, E.; Children's Study, G. Cord blood triglyceride and total cholesterol in preterm and term neonates: reference values and associated factors from the Japan Environment and Children's Study. European Journal of Pediatrics, 2023), the associations we identified were novel. Therefore, validation in external populations is needed.

Cord blood cholesterol levels are associated with different exposures, including lifestyle, environmental exposures, and internal biomarkers. Some of the identified internal exposures may also serve as mediators in the relationships between cholesterol and external exposures. As cardiometabolic health is determined from early life onwards, the insights gained from this research contribute to the primary prevention of cholesterol-related diseases in children.

## Funding

The ENVIRONAGE birth cohort is supported by the Methusalem Fund, the Research Foundation Flanders (FWO, grant numbers 1516112 N, G.0873.11.N.10), and Kom op Tegen Kanker. FWO financially supported RA (grant number 1296523 N) and CC (1249025N).

## CRediT authorship contribution statement

**Rossella Alfano:** Writing – original draft, Formal analysis, Conceptualization. **Tim S. Nawrot:** Writing – review & editing, Supervision, Funding acquisition. **Charlotte Cosemans:** Writing – review & editing. **Joris Penders:** Writing – review & editing, Resources. **Brigitte Reimann:** Writing – review & editing, Data curation. **Congrong Wang:** Writing – review & editing, Data curation. **Michelle Plusquin:** Writing – original draft, Supervision, Conceptualization.

## Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

## Acknowledgments

The authors are extremely grateful to the participating women and neonates, as well as the staff of the maternity ward, midwives, and the staff of the clinical laboratory of East-Limburg Hospital in Genk.

## Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.envint.2026.110238.

## Data availability

Data will be made available on request.

## References

Agier, L., Portengen, L., Chadeau-Hyam, M., Basagaña, X., Giorgis-Allemand, L., Siroux, V., Robinson, O., Vlaanderen, J., González, J.R., Nieuwenhuijsen, M.J.,

Vineis, P., Vrijheid, M., Slama, R., Vermeulen, R., 2016. A Systematic comparison of linear regression-based statistical methods to assess exposome-health associations. Environ. Health Perspect. 124, 1848–1856.
Alfano, R., Chadeau-Hyam, M., Ghantous, A., Keski-Rahkonen, P., Chatzi, L., Perez, A.E., Herceg, Z., Kogevinas, M., de Kok, T.M., Nawrot, T.S., Novoloaca, A., Patel, C.J., Pizzi, C., Robinot, N., Rusconi, F., Scalbert, A., Sunyer, J., Vermeulen, R., Vrijheid, M., Vineis, P., Robinson, O., Plusquin, M., 2020. A multi-omic analysis of birthweight in newborn cord blood reveals new underlying mechanisms related to cholesterol metabolism. Metabolism 110, 154292.
Algaba-Chueca, F., Maymó-Masip, E., Ballesteros, M., Guarque, A., Majali-Martínez, A., Freixes, O., Amigó, N., Fernández-Veledo, S., Vendrell, J., Megía, A., 2022. Cord Blood Advanced Lipoprotein Testing reveals an Interaction between Gestational Diabetes and Birth-Weight and Suggests a New Early Biomarker of Infant Obesity. Biomedicines 10.
Barker, D.J., 2007. The origins of the developmental origins theory. J. Intern. Med. 261, 412–417.
Beneventi, F., Bellingeri, C., De Maggio, I., Spada, C., Pandolfi, M.P., Smaghi, A.B., Cortese, M., Ligari, E., Alpini, C., Spinillo, A., 2025. Maternal and Cord Blood Lipids in Pregnant Women with Obesity and their Impact on Neonatal and Placental Biometric Features. Obes. Sci. Pract. 11, e70053.
Blomberg, A.J., Shih, Y.-H., Messerlian, C., Jørgensen, L.H., Weihe, P., Grandjean, P., 2021. Early-life associations between per- and polyfluoroalkyl substances and serum lipids in a longitudinal birth cohort. Environ. Res. 200, 111400.
Cadby, G., Melton, P.E., McCarthy, N.S., Giles, C., Mellett, N.A., Huynh, K., Hung, J., Beilby, J., Dubé, M.P., Watts, G.F., Blangero, J., Meikle, P.J., Moses, E.K., 2020. Heritability of 596 lipid species and genetic correlation with cardiovascular traits in the Busselton Family Heart Study. J. Lipid Res. 61, 537–545.
Chadeau-Hyam, M., Athersuch, T.J., Keun, H.C., De Iorio, M., Ebbels, T.M., Jenab, M., Sacerdote, C., Bruce, S.J., Holmes, E., Vineis, P., 2011. Meeting-in-the-middle using metabolic profiling - a strategy for the identification of intermediate biomarkers in cohort studies. Biomarkers 16, 83–88.
Collings, P.J., Farrar, D., Gibson, J., West, J., Barber, S.E., Wright, J., 2020. Maternal Physical activity and Neonatal Cord Blood Lipid Levels: Findings from a prospective Pregnancy Cohort. J. Phys. Act. Health 17, 236–241.
Dai, Y., Huo, X., Cheng, Z., Faas, M.M., Xu, X., 2020. Early-life exposure to widespread environmental toxicants and maternal-fetal health risk: a focus on metabolomic biomarkers. Sci. Total Environ. 739, 139626.
Di Angelantonio, E., Sarwar, N., Perry, P., Kaptoge, S., Ray, K.K., Thompson, A., Wood, A.M., Lewington, S., Sattar, N., Packard, C.J., Collins, R., Thompson, S.G., Danesh, J., 2009. Major lipids, apolipoproteins, and risk of vascular disease. J. Am. Med. Assoc. 302, 1993–2000.
Dron, J.S., Patel, A.P., Zhang, Y., Jurgens, S.J., Maamari, D.J., Wang, M., Boerwinkle, E., Morrison, A.C., de Vries, P.S., Fornage, M., Hou, L., Lloyd-Jones, D.M., Psaty, B.M., Tracy, R.P., Bis, J.C., Vasan, R.S., Levy, D., Heard-Costa, N., Rich, S.S., Guo, X., Taylor, K.D., Gibbs, R.A., Rotter, J.I., Willer, C.J., Oelsner, E.C., Moran, A.E., Peloso, G.M., Natarajan, P., Khera, A.V., 2023. Association of Rare Protein-Truncating DNA Variants in APOB or PCSK9 with Low-density Lipoprotein Cholesterol Level and risk of Coronary Heart Disease. JAMA Cardiol. 8, 258–267.
Ezeigwe, A., Ogunmoroti, O., Minhas, A.S., Rodriguez, C.P., Kazzi, B., Fashanu, O.E., Osibogun, O., Kovell, L.C., Harrington, C.M., Michos, E.D., 2022. Association between parity and markers of inflammation: the multi-ethnic study of atherosclerosis. Front. Cardiovasc. Med. 9, 922367.
Felzer-Kim, I.T., Visker, J.R., Ferguson, D.P., Hauck, J.L., 2020. Infant blood lipids: a systematic review of predictive value and influential factors. Expert Rev. Cardiovasc. Ther. 18, 381–394.
Force, U.P.S.T., 2023. Folic Acid Supplementation to prevent Neural Tube Defects: US Preventive Services Task Force Reaffirmation Recommendation Statement. J. Am. Med. Assoc. 330, 454–459.
Geraghty, A.A., Alberdi, G., O'Sullivan, E.J., O'Brien, E.C., Crosbie, B., Twomey, P.J., McAuliffe, F.M., 2017. Maternal and fetal blood lipid concentrations during pregnancy differ by maternal body mass index: findings from the ROLO study. BMC Pregnancy Childbirth 17, 360.
Ghaemi, S., Najafi, R., Kelishadi, R., 2014. Cord blood lipoprotein profile in term, preterm, and late preterm newborns. J. Res. Med. Sci. 19, 1038–1040.
Goldstein, J.L., Brown, M.S., 1987. Regulation of low-density lipoprotein receptors: implications for pathogenesis and therapy of hypercholesterolemia and atherosclerosis. Circulation 76, 504–507.
Horne, H., Holme, A.M., Roland, M.C.P., Holm, M.B., Haugen, G., Henriksen, T., Michelsen, T.M., 2019. Maternal-fetal cholesterol transfer in human term pregnancies. Placenta 87, 23–29.
Huang, S., Liu, S., Huang, M., He, J.-R., Wang, C., Wang, T., Feng, X., Kuang, Y., Lu, J., Gu, Y., Xia, X., Lin, S., Shen, S., Liu, X., Lu, M., Yuan, M., Xiao, W., Zhou, F., Wei, D., Chen, N., Wei, X., Zhang, L., Xie, J., Tu, S., Liu, H., Chan, F., He, J., Liu, P., Guo, Y., Li, Y., Deng, L., Liu, W., Shao, D., Yang, Y., Li, Y., Zhou, W., Fu, Q., Xia, H., Qiu, X., 2024. Born in Guangzhou Cohort Study. G. The Born in Guangzhou Cohort Study enables generational genetic discoveries, Nature.
Işcan, A., Yiğitoğlu, M.R., Ece, A., Ari, Z., Akyildiz, M., 1997. The effect of cigarette smoking during pregnancy on cord blood lipid, lipoprotein and apolipoprotein levels. Jpn. Heart J. 38, 497–502.
Janssen, B.G., Madhloum, N., Gyselaers, W., Bijnens, E., Clemente, D.B., Cox, B., Hogervorst, J., Luyten, L., Martens, D.S., Peusens, M., Plusquin, M., Provost, E.B., Roels, H.A., Saenen, N.D., Tsamou, M., Vriens, A., Winckelmans, E., Vrijens, K., Nawrot, T.S., 2017. Cohort Profile: the ENVIRonmental influence on early AGEing (ENVIRONAGE): a birth cohort study. Int. J. Epidemiol. 46, 1386–1387M.
Juhola, J., Magnussen, C.G., Viikari, J.S., Kähönen, M., Hutri-Kähönen, N., Jula, A., Lehtimäki, T., Åkerblom, H.K., Pietikäinen, M., Laitinen, T., Jokinen, E., Taittonen, L., Raitakari, O.T., Juonala, M., 2011. Tracking of serum lipid levels, blood pressure, and body mass index from childhood to adulthood: the Cardiovascular Risk in Young Finns Study. J. Pediatr. 159, 584–590.
Kazzi, B., Ogunmoroti, O., Rodriguez, C.P., Zhao, D., Minhas, A.S., Osibogun, O., Subramanya, V., Allison, M.A., Ouyang, P., Michos, E.D., 2022. Parity history and later Life sex Hormone Levels in the Multi-Ethnic Study of Atherosclerosis (MESA). Can. J. Cardiol. 38, 1893–1900.
Kuan, V., Martineau, A.R., Griffiths, C.J., Hyppönen, E., Walton, R., 2013. DHCR7 mutations linked to higher vitamin D status allowed early human migration to northern latitudes. BMC Evol. Biol. 13, 144.
Lai, T., Su, Z., Tian, G., Sun, J., Hu, K., 2024. The mediating role of inflammation in the association between cotinine levels and remnant cholesterol: a cross-sectional study. Lipids Health Dis. 23, 385.
Li, M.X., Yeung, J.M., Cherny, S.S., Sham, P.C., 2012. Evaluating the effective numbers of independent tests and significant p-value thresholds in commercial genotyping arrays and public imputation reference datasets. Hum. Genet. 131, 747–756.
Li, Y., Li, J., Cidan, W., Yin, L., Nima, Q., Li, X., 2025. Association between indoor air pollution exposure and dyslipidemia among chinese adults: the role of leisure-time physical activity. Ecotoxicol. Environ. Saf. 305, 119170.
Määttä, J., Sissala, N., Dimova, E.Y., Serpi, R., Moore, L.G., Koivunen, P., 2018. Hypoxia causes reductions in birth weight by altering maternal glucose and lipid metabolism. Sci. Rep. 8, 13583.
Manczak, E.M., Gotlib, I.H., 2019. Lipid Profiles at Birth Predict Teacher-rated Child Emotional and Social Development 5 Years later. Psychol. Sci. 30, 1780–1789.
Méndez, N., Corvalan, F., Halabi, D., Ehrenfeld, P., Maldonado, R., Vergara, K., Seron-Ferre, M., Torres-Farfan, C., 2023. From gestational chronodisruption to noncommunicable diseases: Pathophysiological mechanisms of programming of adult diseases, and the potential therapeutic role of melatonin. J. Pineal Res. 75, e12908.
Motevalizadeh, E., Díaz-López, A., Martín, F., Basora, J., Arija, V., 2024. Association of Parity with Insulin Resistance Early in Pregnant Women: ECLIPSES Study. J. Clin. Endocrinol. Metabol. 109, 730–739.
Go, H.; Hashimoto, K.; Maeda, H.; Ogasawara, K.; Kume, Y.; Murata, T.; Sato, A.; Ogata, Y.; Shinoki, K.; Nishigori, H.; Ikeda-Araki, A.; Fujimori, K.; Yasumura, S.; Hosoya, M.; Kamijima, M.; Yamazaki, S.; Ohya, Y.; Kishi, R.; Yaegashi, N.; Hashimoto, K.; Mori, C.; Ito, S.; Yamagata, Z.; Nakayama, T.; Sobue, T.; Shima, M.; Nakamura, H.; Suganuma, N.; Kusuhara, K.; Katoh, T.; the Japan, E.; Children's Study, G. Cord blood triglyceride and total cholesterol in preterm and term neonates: reference values and associated factors from the Japan Environment and Children's Study. European Journal of Pediatrics 2023;182:4547-4556.
Øyri, L.K.L.; Bogsrud, M.P.; Christensen, J.J.; Ulven, S.M.; Brantsæter, A.L.; Retterstøl, K.; Brekke, H.K.; Michelsen, T.M.; Henriksen, T.; Roeters van Lennep, J.E.; Magnus, P.; Veierød, M.B.; Holven, K.B. Novel associations between parental and newborn cord blood metabolic profiles in the Norwegian Mother, Father and Child Cohort Study. BMC Medicine 2021a;19:91.
Øyri, L.K.L., Bogsrud, M.P., Kristiansen, A.L., Myhre, J.B., Astrup, H., Retterstøl, K., Brekke, H.K., Roeters van Lennep, J.E., Andersen, L.F., Holven, K.B., 2021b. Cholesterol at ages 6, 12 and 24 months: Tracking and associations with diet and maternal cholesterol in the Infant Cholesterol Study. Atherosclerosis 326, 11–16.
Rangel, V.S., Castillo, J.M., Duarte, J.D., Barreto, M.A., Ramirez, A.V., Daza, J.A., 2026. Ruiz-Sternberg Á, M. A narrative review. Int J Gynaecol Obstet, Understanding pre-eclampsia and fetal growth restriction at high altitude.
Schade, D.S., Shey, L., Eaton, R.P., 2020. Cholesterol Review: a Metabolically Important Molecule. Endocr. Pract. 26, 1514–1523.
Sinisi, S.E., van der Laan, M.J., 2004. Deletion/substitution/addition algorithm in learning with applications in genomics. Stat. Appl. Genet. Mol. Biol. 3, Article18.
Slominski, R.M., Chen, J.Y., Raman, C., Slominski, A.T., 2024. Photo-neuro-immuno-endocrinology: how the ultraviolet radiation regulates the body, brain, and immune system. PNAS 121, e2308374121.
Sreckovic, I., Birner-Gruenberger, R., Obrist, B., Stojakovic, T., Scharnagl, H., Holzer, M., Scholler, M., Philipose, S., Marsche, G., Lang, U., Desoye, G., Wadsack, C., 2013. Distinct composition of human fetal HDL attenuates its anti-oxidative capacity. Biochim. Biophys. Acta 1831, 737–746.
Stadler, J.T., Wadsack, C., Marsche, G., 2021. Fetal High-Density Lipoproteins: Current Knowledge on Particle Metabolism, Composition and Function in Health and Disease. Biomedicines 9.
Strandkjær, N., Hansen, M.K., Nielsen, S.T., Frikke-Schmidt, R., Tybjærg-Hansen, A., Nordestgaard, B.G., Tabor, A., Bundgaard, H., Iversen, K., Kamstrup, P.R., 2022. Lipoprotein(a) Levels at Birth and in Early Childhood: the COMPARE Study. J. Clin. Endocrinol. Metab. 107, 324–335.
Taageby Nielsen, S., Mohr Lytsen, R., Strandkjær, N., Juul Rasmussen, I., Sillesen, A.-S., Vøgg, R.O.B., Axelsson Raja, A., Nordestgaard, B.G., Kamstrup, P.R., Iversen, K., Bundgaard, H., Tybjærg-Hansen, A., Frikke-Schmidt, R., 2023. Significance of lipids, lipoproteins, and apolipoproteins during the first 14–16 months of life. Eur. Heart J. 44, 4408–4418.
Tan, M.Y., Zhang, P., Wu, S., Zhu, S.X., Gao, M., 2025. Association between non-high-density lipoprotein cholesterol to high-density lipoprotein cholesterol ratio and serum thyroid function measures: recent Findings from NHANES 2007-2012 and Mendelian randomization. Front Endocrinol (lausanne) 16, 1467254.
Teslovich, T.M.; Musunuru, K.; Smith, A.V.; Edmondson, A.C.; Stylianou, I.M.; Koseki, M.; Pirruccello, J.P.; Ripatti, S.; Chasman, D.I.; Willer, C.J.; Johansen, C.T.; Fouchier, S.W.; Isaacs, A.; Peloso, G.M.; Barbalic, M.; Ricketts, S.L.; Bis, J.C.; Aulchenko, Y.S.; Thorleifsson, G.; Feitosa, M.F.; Chambers, J.; Orho-Melander, M.; Melander, O.; Johnson, T.; Li, X.; Guo, X.; Li, M.; Shin Cho, Y.; Jin Go, M.; Jin Kim, Y.; Lee, J.-Y.; Park, T.; Kim, K.; Sim, X.; Twee-Hee Ong, R.; Croteau-Chonka, D.C.; Lange, L.A.; Smith, J.D.; Song, K.; Hua Zhao, J.; Yuan, X.; Luan, J.a.; Lamina, C.; Ziegler, A.;

Zhang, W.; Zee, R.Y.L.; Wright, A.F.; Witteman, J.C.M.; Wilson, J.F.; Willemsen, G.; Wichmann, H.E.; Whitfield, J.B.; Waterworth, D.M.; Wareham, N.J.; Waeber, G.; Vollenweider, P.; Voight, B.F.; Vitart, V.; Uitterlinden, A.G.; Uda, M.; Tuomilehto, J.; Thompson, J.R.; Tanaka, T.; Surakka, I.; Stringham, H.M.; Spector, T.D.; Soranzo, N.; Smit, J.H.; Sinisalo, J.; Silander, K.; Sijbrands, E.J.G.; Scuteri, A.; Scott, J.; Schlessinger, D.; Sanna, S.; Salomaa, V.; Saharinen, J.; Sabatti, C.; Ruokonen, A.; Rudan, I.; Rose, L.M.; Roberts, R.; Rieder, M.; Psaty, B.M.; Pramstaller, P.P.; Pichler, I.; Perola, M.; Penninx, B.W.J.H.; Pedersen, N.L.; Pattaro, C.; Parker, A.N.; Pare, G.; Oostra, B.A.; O'Donnell, C.J.; Nieminen, M.S.; Nickerson, D.A.; Montgomery, G.W.; Meitinger, T.; McPherson, R.; McCarthy, M.I.; McArdle, W.; Masson, D.; Martin, N.G.; Marroni, F.; Mangino, M.; Magnusson, P.K.E.; Lucas, G.; Luben, R.; Loos, R.J.F.; Lokki, M.-L.; Lettre, G.; Langenberg, C.; Launer, L.J.; Lakatta, E.G.; Laaksonen, R.; Kyvik, K.O.; Kronenberg, F.; König, I.R.; Khaw, K.-T.; Kaprio, J.; Kaplan, L.M.; Johansson, Å.; Jarvelin, M.-R.; Cecile J. W. Janssens, A.; Ingelsson, E.; Igl, W.; Kees Hovingh, G.; Hottenga, J.-J.; Hofman, A.; Hicks, A.A.; Hengstenberg, C.; Heid, I.M.; Hayward, C.; Havulinna, A.S.; Hastie, N.D.; Harris, T.B.; Haritunians, T.; Hall, A.S.; Gyllensten, U.; Guiducci, C.; Groop, L.C.; Gonzalez, E.; Gieger, C.; Freimer, N.B.; Ferrucci, L.; Erdmann, J.; Elliott, P.; Ejebe, K.G.; Döring, A.; Dominiczak, A.F.; Demissie, S.; Deloukas, P.; de Geus, E.J.C.; de Faire, U.; Crawford, G.; Collins, F.S.; Chen, Y.-d.I.; Caulfield, M.J.; Campbell, H.; Burtt, N.P.; Bonnycastle, L.L.; Boomsma, D.I.; Boekholdt, S.M.; Bergman, R.N.; Barroso, I.; Bandinelli, S.; Ballantyne, C.M.; Assimes, T.L.; Quertermous, T.; Altshuler, D.; Seielstad, M.; Wong, T.Y.; Tai, E.S.; Feranil, A.B.; Kuzawa, C.W.; Adair, L.S.; Taylor Jr, H.A.; Borecki, I.B.; Gabriel, S.B.; Wilson, J.G.; Holm, H.; Thorsteinsdottir, U.; Gudnason, V.; Krauss, R.M.; Mohlke, K. L.; Ordovas, J.M.; Munroe, P.B.; Kooner, J.S.; Tall, A.R.; Hegele, R.A.; Kastelein, J.J. P.; Schadt, E.E.; Rotter, J.I.; Boerwinkle, E.; Strachan, D.P.; Mooser, V.; Stefansson, K.; Reilly, M.P.; Samani, N.J.; Schunkert, H.; Cupples, L.A.; Sandhu, M.S.; Ridker, P. M.; Rader, D.J.; van Duijn, C.M.; Peltonen, L.; Abecasis, G.R.; Boehnke, M.; Kathiresan, S. Biological, clinical and population relevance of 95 loci for blood lipids. Nature 2010;466:707-713.

Textor, J., van der Zander, B., Gilthorpe, M.S., Liśkiewicz, M., Ellison, G.T.H., 2016. Robust causal inference using directed acyclic graphs: the R package 'dagitty'. Int. J. Epidemiol. 45, 1887–1894.

Vansteelandt, S., Bekaert, M., Lange, T., 2012. Imputation strategies for the estimation of natural direct and indirect effects. Epidemiologic Methods 1, 27.

Wang, C., Meng, X.-C., Huang, C., Wang, J., Liao, Y.-H., Huang, Y., Liu, R., 2023. Association between ambient air pollutants and lipid profile: a systematic review and meta-analysis. Ecotoxicol. Environ. Saf. 262, 115140.

Willer, C.J.; Schmidt, E.M.; Sengupta, S.; Peloso, G.M.; Gustafsson, S.; Kanoni, S.; Ganna, A.; Chen, J.; Buchkovich, M.L.; Mora, S.; Beckmann, J.S.; Bragg-Gresham, J.L.; Chang, H.-Y.; Demirkan, A.; Den Hertog, H.M.; Do, R.; Donnelly, L.A.; Ehret, G.B.; Esko, T.; Feitosa, M.F.; Ferreira, T.; Fischer, K.; Fontanillas, P.; Fraser, R.M.; Freitag, D.F.; Gurdasani, D.; Heikkilä, K.; Hyppönen, E.; Isaacs, A.; Jackson, A.U.; Johansson, Å.; Johnson, T.; Kaakinen, M.; Kettunen, J.; Kleber, M.E.; Li, X.; Luan, J.a.; Lyytikäinen, L.-P.; Magnusson, P.K.E.; Mangino, M.; Mihailov, E.; Montasser, M.E.; Müller-Nurasyid, M.; Nolte, I.M.; O'Connell, J.R.; Palmer, C.D.; Perola, M.; Petersen, A.-K.; Sanna, S.; Saxena, R.; Service, S.K.; Shah, S.; Shungin, D.; Sidore, C.; Song, C.; Strawbridge, R.J.; Surakka, I.; Tanaka, T.; Teslovich, T.M.; Thorleifsson, G.; Van den Herik, E.G.; Voight, B.F.; Volcik, K.A.; Waite, L.L.; Wong, A.; Wu, Y.; Zhang, W.; Absher, D.; Asiki, G.; Barroso, I.; Been, L.F.; Bolton, J.L.; Bonnycastle, L.L.; Brambilla, P.; Burnett, M.S.; Cesana, G.; Dimitriou, M.; Doney, A.S.F.; Döring, A.; Elliott, P.; Epstein, S.E.; Eyjolfsson, G.I.; Gigante, B.; Goodarzi, M.O.; Grallert, H.; Gravito, M.L.; Groves, C.J.; Hallmans, G.; Hartikainen, A.-L.; Hayward, C.; Hernandez, D.; Hicks, A. A.; Holm, H.; Hung, Y.-J.; Illig, T.; Jones, M.R.; Kaleebu, P.; Kastelein, J.J.P.; Khaw, K.-T.; Kim, E.; Klopp, N.; Komulainen, P.; Kumari, M.; Langenberg, C.; Lehtimäki, T.; Lin, S.-Y.; Lindström, J.; Loos, R.J.F.; Mach, F.; McArdle, W.L.; Meisinger, C.; Mitchell, B.D.; Müller, G.; Nagaraja, R.; Narisu, N.; Nieminen, T.V.M.; Nsubuga, R.N.; Olafsson, I.; Ong, K.K.; Palotie, A.; Papamarkou, T.; Pomilla, C.; Pouta, A.; Rader, D. J.; Reilly, M.P.; Ridker, P.M.; Rivadeneira, F.; Rudan, I.; Ruokonen, A.; Samani, N.; Scharnagl, H.; Seeley, J.; Silander, K.; Stancáková, A.; Stirrups, K.; Swift, A.J.; Tiret, L.; Uitterlinden, A.G.; van Pelt, L.J.; Vedantam, S.; Wainwright, N.; Wijmenga, C.; Wild, S.H.; Willemsen, G.; Wilsgaard, T.; Wilson, J.F.; Young, E.H.; Zhao, J.H.; Adair, L.S.; Arveiler, D.; Assimes, T.L.; Bandinelli, S.; Bennett, F.; Bochud, M.; Boehm, B.O.; Boomsma, D.I.; Borecki, I.B.; Bornstein, S.R.; Bovet, P.; Burnier, M.; Campbell, H.; Chakravarti, A.; Chambers, J.C.; Chen, Y.-D.I.; Collins, F.S.; Cooper, R.S.; Danesh, J.; Dedoussis, G.; de Faire, U.; Feranil, A.B.; Ferrières, J.; Ferrucci, L.; Freimer, N.B.; Gieger, C.; Groop, L.C.; Gudnason, V.; Gyllensten, U.; Hamsten, A.; Harris, T.B.; Hingorani, A.; Hirschhorn, J.N.; Hofman, A.; Hovingh, G.K.; Hsiung, C.A.; Humphries, S.E.; Hunt, S.C.; Hveem, K.; Iribarren, C.; Järvelin, M.-R.; Jula, A.; Kähönen, M.; Kaprio, J.; Kesäniemi, A.; Kivimaki, M.; Kooner, J.S.; Koudstaal, P.J.; Krauss, R.M.; Kuh, D.; Kuusisto, J.; Kyvik, K.O.; Laakso, M.; Lakka, T.A.; Lind, L.; Lindgren, C.M.; Martin, N.G.; März, W.; McCarthy, M.I.; McKenzie, C.A.; Meneton, P.; Metspalu, A.; Moilanen, L.; Morris, A.D.; Munroe, P.B.; Njølstad, I.; Pedersen, N. L.; Power, C.; Pramstaller, P.P.; Price, J.F.; Psaty, B.M.; Quertermous, T.; Rauramaa, R.; Saleheen, D.; Salomaa, V.; Sanghera, D.K.; Saramies, J.; Schwarz, P.E.H.; Sheu, W.H.H.; Shuldiner, A.R.; Siegbahn, A.; Spector, T.D.; Stefansson, K.; Strachan, D.P.; Tayo, B.O.; Tremoli, E.; Tuomilehto, J.; Uusitupa, M.; van Duijn, C.M.; Vollenweider, P.; Wallentin, L.; Wareham, N.J.; Whitfield, J.B.; Wolffenbuttel, B.H.R.; Ordovas, J. M.; Boerwinkle, E.; Palmer, C.N.A.; Thorsteinsdottir, U.; Chasman, D.I.; Rotter, J.I.; Franks, P.W.; Ripatti, S.; Cupples, L.A.; Sandhu, M.S.; Rich, S.S.; Boehnke, M.; Deloukas, P.; Kathiresan, S.; Mohlke, K.L.; Ingelsson, E.; Abecasis, G.R.; Global Lipids Genetics, C. Discovery and refinement of loci associated with lipid levels. Nature Genetics 2013;45:1274-1283.

Ye, Q.Q., Kong, S.M., Yin, X., Gao, C., Lu, M.S., Ramakrishnan, R., Guo, C., Yao, W., Zeng, J.Y., Kuang, Y.S., Lu, J.H., He, J.R., Qiu, X., 2022. Associations of cord blood lipids with childhood adiposity at the age of three years: a prospective birth cohort study. Metabolites 12.

Zhang, Y., Shi, J., Ma, Y., Yu, N., Zheng, P., Chen, Z., Wang, T., Jia, G., 2023. Association between air pollution and lipid profiles. Toxics 11.