# The Influence of Dietary Counseling Over 20 Years on Tracking of Non-High-Density Lipoprotein Cholesterol from Infancy to Young Adulthood

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This 26-year study found that non–high-density lipoprotein cholesterol (non-HDL-C) levels tracked from infancy to young adulthood suggesting early-life non-HDL-C could predict future levels. However, infancy-onset dietary counseling reduced the odds of maintaining at-risk non-HDL-C, highlighting the potential importance of early interventions in preventing cardiovascular risk associated with high pediatric non-HDL-C. *(J Pediatr 2024;264:113776).*

Cardiovascular disease (CVD) risk is established early in life in part due to the longitudinal tracking (or persistence) of contributing risk factors. Lipid risk factors are particularly stable from childhood to adulthood,[1] thereby offering a measure with predictive utility to identify those at heightened CVD risk. Increasingly, attention has focused on non–high-density lipoprotein cholesterol (non-HDL-C)—a comprehensive lipid marker of all atherogenic apolipoprotein B-containing lipoproteins—given its causal role in CVD.[2,3] The clinical utility of non-HDL-C is further underscored by data showing that non-HDL-C levels in youth are at least as effective as low-density lipoprotein cholesterol (a primary lipid treatment target) in predicting future carotid intima-media thickness[4] and are a better predictor for dyslipidemia and nonlipid cardiovascular risk factors (hyperinsulinemia, hyperglycemia)[5] in adulthood. Moreover, non-HDL-C plays a central role in pediatric lipid screening guidelines for the prevention of CVD.[6,7] However, limited studies track non-HDL-C.[8]

Nutrition from birth impacts circulating lipid levels,[7] as evidenced by the Special Turku Coronary Risk Factor Intervention Project (STRIP), where 20-year infancy-onset low-saturated-fat dietary counseling was associated with improved longitudinal non-HDL-C levels and lipid risk profiles.[9] Such interventions could potentially modulate non-HDL-C trajectories, thereby reducing associated CVD risk in adulthood. Yet, their efficacy in disrupting long-term tracking of non-HDL-C levels has not been studied. Therefore, we use STRIP data to examine non-HDL-C tracking from infancy to young adulthood and assess the impact of dietary counseling intervention on this tracking.

| | |
|---|---|
| CVD | Cardiovascular disease |
| GEE | Generalized estimating equation |
| HDL-C | High-density lipoprotein cholesterol |
| Non-HDL-C | Non-high-density lipoprotein cholesterol |
| STRIP | Special Turku Coronary Risk Factor Intervention Project |

## Methods

The STRIP, a randomized controlled trial based in Finland, enrolled infants at 5 months of age from well-baby clinics (Clinical Trial Registration—clinicaltrials.gov. Identifier: NCT00223600). Participants were randomized into either intervention or control groups.[9-11] Detailed design and methods have been described previously[9-11] and are summarized in the **Appendix** (online). To summarize, the intervention group received biannual individualized dietary and subsequently antismoking counseling from 7 months to 20 years of age.[9-11] The control group made biannual visits until age 7 years and annual visits thereafter until age 20 years.[9-11] Of the 1116 enrolled, 551 returned for a follow-up at age 26 years, 6 years after the cessation of dietary counseling.

In the analysis tracking non-HDL-C from age 7 months onwards, we included 798 participants who had non-HDL-C measured at 7 months and at least 1 additional follow-up measurement (conducted annually from 13 months to

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20 years, except at ages 6 and 8 years, and then again at 26 years). Among these, 399 were in the intervention group (46.9% female, 53.1% male), and 399 were in the control group (50.6% female, 49.4% male) (**Figure 1**, online available at www.jpeds.com). We then re-fitted the analyses stratified by different initial ages and lengths of follow-up. For each observed time period, the tracking analysis included only participants with non-HDL-C measurements at both the beginning and end of the time period. For example, when examining the tracking of non-HDL-C from ages 2 to 20 years, only participants with non-HDL-C measurement at both age 2 years and 20 years were included in the analysis. This led to variable participant numbers in each sub-analysis (**Table I**, online available at www.jpeds.com).

Ethics approval for the study was obtained from the Joint Commission on Ethics of Turku University and Central Hospital. In terms of participant consent, written informed consent was provided by the parents until the participants reached 15 years of age and was obtained from the participants themselves at 15, 18, and 26 years of age.

Nonfasting venous blood samples were collected when participants were aged 5 years or younger, with subsequent samples taken after overnight fasting. Serum lipids and lipoproteins were assessed annually from 7 months to 20 years (except age 6 and 8 years) and at 26 years.[9] Total cholesterol was assessed with enzymatic cholesterol esterase–cholesterol oxidase method (cholesterol reagent; Beckman Coulter). HDL-C was analyzed after precipitation of low-density lipoprotein and very-low-density lipoproteins with dextran sulfate–$Mg^{2+}$. Non-HDL-C was calculated as total cholesterol minus HDL-C. Participants were divided into 4 at-risk groups based on non-HDL-C values: ≥3.103 mmol/L (120 mg/dL); ≥3.750 mmol/L (145 mg/dL); ≥4.138 mmol/L (160 mg/dL); and ≥4.913 mmol/L (190 mg/dL).[7]

Statistical analyses were performed using Stata (version 17.0, StataCorp). Participant characteristics are presented as mean (SD) for continuous data or proportions for categorical data. Generalized estimating equations (GEEs) estimated the tracking of non-HDL-C by calculating the stability coefficients for continuous non-HDL-C levels and the tracking coefficients (represented as OR) for at-risk non-HDL-C categories.[12,13] Additional details on GEE modeling are provided in the **Appendix** (online). Stability coefficients of <0.3 were categorized as poor tracking, 0.3 to <0.5 as fair tracking, ≥0.5 to <0.7 as moderate tracking, and ≥0.7 as strong tracking.[14] For stability analyses, non-HDL-C was transformed into age- and sex-specific z scores. Going beyond 2 time-point analyses, GEE has the advantage that all available longitudinal data are used to calculate the tracking estimates. Using GEE, we estimated the overall stability coefficients for non-HDL-C levels and tracking coefficients for at-risk status for non-HDL-C values ≥3.750 mmol/L (145 mg/dL) (considered "high" according to the National Heart, Lung, and Blood Institute cut-offs[7]) from age 7 months to 26 years, and then re-fitted stratified by different initial ages and lengths of follow-up. All tracking analyses were stratified by intervention and adjusted for sex and length to follow-up where appropriate. We used the tracking of at-risk status for non-HDL-C values ≥3.750 mmol/L (145 mg/dL) from age 7 months to subsequent follow-ups to depict the reduced odds (tracking coefficients) for the intervention vs control group. GEE models also were conducted to estimate the tracking coefficients for other at-risk groups (non-HDL-C values: ≥3.103 mmol/L [120 mg/dL]; ≥4.138 mmol/L [160 mg/dL]; ≥4.913 mmol/L [190 mg/dL]). As duration of breastfeeding was known to differ between intervention and control groups in the STRIP, which might directly impact on non-HDL-C levels, we performed sensitivity analyses that considered duration of breastfeeding.

## Results

**Table II** (online) presents non-HDL-C levels and the proportion of participants in control and intervention groups classified in each of the 4 non-HDL-C at-risk groups at each age point. Since the age of 13 months, the intervention group has consistently demonstrated lower levels of non-HDL-C on average compared with the control group. Duration of breastfeeding in the intervention group (mean [SD], 7.1 [3.7] months) was longer compared with the control group (6.7 [4.0] months).

The overall 26-year stability coefficients of non-HDL-C levels, from age 7 months, were 0.42 (95% CI 0.32-0.53) in the intervention group and 0.60 (0.52-0.67) in the control group (*P* value for multiplicative interaction term between group and non-HDL-C tracking interaction = .012). **Figure 2** depicts a matrix of stability coefficients over varying time periods and ages. Generally, stability coefficients in the intervention group (0.34-0.80) were lower than those in the control group (0.58-0.88). **Table III**, online, presents the overall 26-year tracking coefficients of participants in the intervention and control groups according to different-risk non-HDL-C cut-offs. Regardless of the group, tracking coefficients generally increased with the severity of at-risk non-HDL-C values. The difference in tracking coefficients between the control and the intervention groups showed a tendency to diminish with increasing severity of at-risk non-HDL-C values (**Table III**, online available at www.jpeds.com).

**Figure 3** (online) presents a matrix of tracking coefficients (calculated as OR) over varying time periods and ages. For all observed at-risk cut-offs, non-HDL-C tracking coefficients were generally lower in the intervention group than in the control group. Notably, GEE models for some time periods failed to converge or presented very high OR (**Figure 3**, D; online available at www.jpeds.com), likely due to the limited number of participants classified in the greatest at-risk group of non-HDL-C >4.913 mmol/L (190 mg/dL) (**Table II**, online available at www.jpeds.com). In this regard, tracking analyses for non-HDL-C ≥4.138 mmol/L

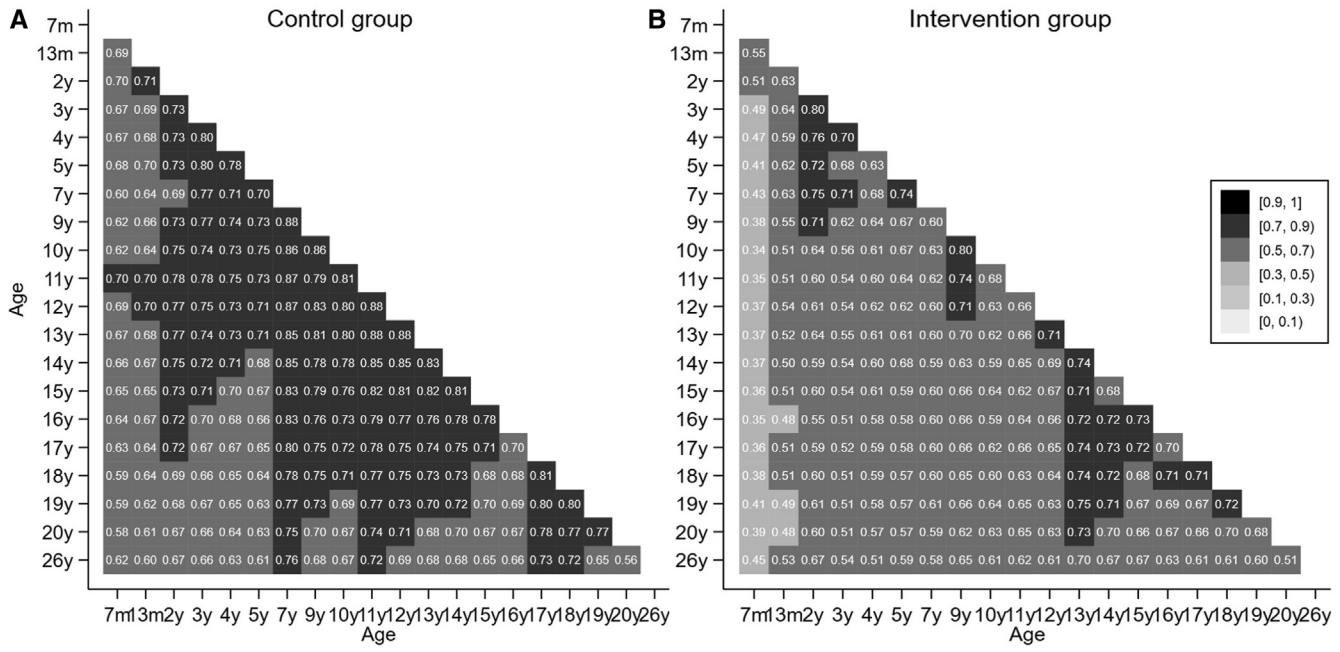



**Figure 2.** Matrix of stability coefficients for non-HDL-C over the time intervals between each 2 age points from infancy to young adulthood, stratified by: **A,** control group, and **B,** intervention group. Values indicate stability coefficients. *Color plates* represent the strength of tracking. "[" indicate more than or equal to the values; "]" indicate less than or equal to the values; ")" indicate less than the values. Non-HDL-C was transformed into age- and sex-specific z scores.

(160 mg/dL) and non-HDL-C ≥4.913 mmol/L (190 mg/dL) were not stratified by breastfeeding duration, as the GEEs failed to converge. In general, participants in the intervention group had a reduced odds of maintaining at-risk status of non-HDL-C ≥3.750 mmol/L (145 mg/dL) over time compared with the control group (**Table IV**).

Estimates ($\beta$, OR) from sensitivity analyses that additionally considered duration of breastfeeding were not markedly different from the primary results. For example, the overall 26-year stability coefficients of non-HDL-C were 0.44 (95% CI 0.33-0.54) in the intervention group and 0.59 (0.52-0.68) in the control group, and there was weak evidence of interaction ($P$ values for multiplicative interaction term between duration of breastfeeding and intervention group = .85). To account for missing data, we refitted our main analyses using multiple imputation combined with inverse probability weighting,[15] yielding similar results to the primary analyses. The imputation considered baseline variables informative of dropout (sex, intervention, body mass index, total cholesterol, energy-adjusted fiber and saturated fatty acid intake, and parental education and occupation status).[16]

## Discussion

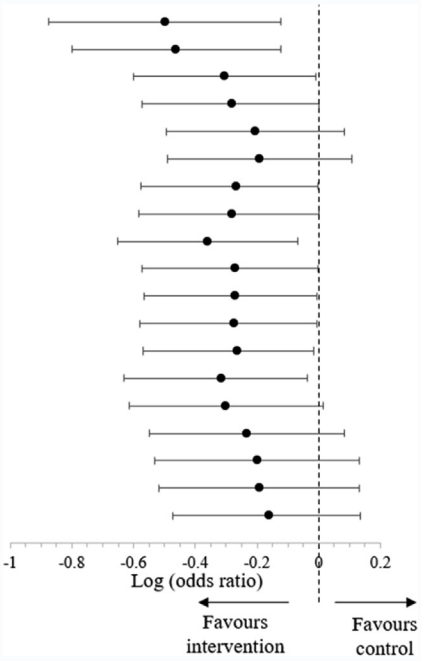
This study is the first to report data on serial non-HDL-C measurements spanning from infancy to young adulthood. We showed that non-HDL-C tracking over the studied time periods and ages was fair to strong. In doing so, our study extends the only previous report on non-HDL-C tracking that showed GEE stability coefficients of 0.56 (95% CI 0.52-0.60) from adolescence to young adulthood.[8] Interestingly, the magnitude of non-HDL-C tracking in the STRIP appeared to be stronger than other risk factors we have previously reported, such as physical activity and cardiorespiratory fitness between adolescence and young adulthood.[16]

Dietary intervention trials[17-19] have emphasized the beneficial effects of a healthy diet,[18,19] and dietary counseling[17] in childhood and adolescence on lipid profiles. However, these trials typically have been of short duration.[17-19] This study provides evidence that infancy-onset dietary counseling can mitigate the likelihood of maintaining at-risk non-HDL-C levels over an extended time while the intervention is ongoing, as well as 6-years' postwithdrawal of the intervention. Given previous findings establishing adult non-HDL-C as a causal factor in CVD,[2,3] and the association of child non-HDL-C with adult carotid intima-media thickness,[20] coronary artery calcification,[21] and cardiovascular events, our results highlight the potential of early dietary interventions. Such interventions may shift individuals on at-risk non-HDL-C trajectories toward healthier profiles in subsequent ages or life stages, thereby delaying or preventing CVD-related complications associated with persistent at-risk non-HDL-C levels.

The STRIP intervention was a comprehensive approach encompassing multiple facets. The main goal of the STRIP intervention was to change dietary "quality"—specifically, replacing saturated with unsaturated fats and reducing cholesterol intake—rather than the "quantity" of fat consumed.[11] In addition, the intervention group also received counseling to prevent smoking and encouragement

**Table IV. Tracking coefficients of at-risk of non-HDL-C ≥3.750 mmol/L (145 mg/dL) from age 7 months to subsequent follow-up surveys in intervention group compared with those in control group**

| Ages | Tracking coefficients (OR) | 95% CI |
|---|---|---|
| 13 mo | 0.3 | 0.1-0.8 |
| 2 y | 0.3 | 0.2-0.8 |
| 3 y | 0.5 | 0.3-0.98 |
| 4 y | 0.5 | 0.3-0.99 |
| 5 y | 0.6 | 0.3-1.2 |
| 7 y | 0.6 | 0.3-1.3 |
| 9 y | 0.5 | 0.3-0.99 |
| 10 y | 0.5 | 0.3-0.99 |
| 11 y | 0.4 | 0.2-0.9 |
| 12 y | 0.5 | 0.3-0.99 |
| 13 y | 0.5 | 0.3-0.98 |
| 14 y | 0.5 | 0.3-0.98 |
| 15 y | 0.5 | 0.3-0.96 |
| 16 y | 0.5 | 0.2-0.9 |
| 17 y | 0.5 | 0.2-1.0 |
| 18 y | 0.6 | 0.3-1.2 |
| 19 y | 0.6 | 0.3-1.4 |
| 20 y | 0.6 | 0.3-1.4 |
| 26 y | 0.7 | 0.3-1.4 |




Age represents the age of the last survey for each observation follow-up period. *Circle* represents log scale of OR (tracking coefficient) in intervention group compared with those in control group. *Error bars* indicate log scale of 95% CIs. For example, in the intervention group, the odds of maintaining at-risk status from 7 months to 26 years was reduced by 30% compared with the odds of maintaining this at-risk status in the control group (OR; 95% CI: 0.7; 0.3-1.4). Model adjusted for sex.

toward physically active lifestyles.[11] Lifestyle factors that individually and collectively contribute to improved lipid/non-HDL-C profiles.[9,22,23] The intervention was proven safe, exhibiting no adverse effects on growth, neurologic or pubertal development, psychosocial well-being, or age at menarche for girls.[24,25] We have shown beneficial effects of the STRIP intervention on various CVD risk factors, such as lipids,[11,26] blood pressure, measures of glucose homeostasis,[9,27] cognitive function,[10] and vascular endothelial function.[28] Our current study builds on this, offering novel insights into how such interventions could potentially modify non-HDL-C trajectories. Taken together, these findings underscore the potential for early and sustained implementation of multifaceted interventions in modifying the long-term risk of CVD. However, this would require long-term commitment from health care professionals, individuals, families, and communities and necessitates legislative policy influencing schools and the food industry.[7] Interestingly, we observed a decrease in the difference in tracking coefficients between the control and intervention groups as the severity of high-risk non-HDL-C values increased. This trend suggests that the STRIP intervention may be less effective in individuals with extremely high non-HDL-C values (≥4.913 mmol/L [190 mg/dL]). For such individuals, a multifaceted approach may prove more beneficial. This might involve augmenting the dietary counseling intervention together with other preventative approaches, such as screening for familial hypercholesterolemia.

Strengths of this study include the 26-year follow-up period and the serial non-HDL-C measurements starting from infancy. Although loss to follow-up poses a limitation, no differences were observed in total cholesterol and HDL-C levels between participants and dropouts.[9]

In conclusion, non-HDL-C levels tend to remain moderately to strongly stable from infancy to young adulthood. Early dietary counseling could help shift individuals at risk due to elevated non-HDL-C levels toward healthier trajectories. These findings reinforce the need for early screening and interventions of non-HDL-C, providing key insights for public health strategies aimed at reducing CVD burden from a very young age. ■

## Declaration of Competing Interest

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