Date: 2025-08-26

RE: Post-publication concern regarding "Longitudinal Data From the KETO-CTA Study: Plaque Predicts Plaque, ApoB Does Not" (Soto-Mota *et al.* JACC: Advances, 2025; doi:10.1016/j.jacadv.2025.101686)

Dear Editors,

I am submitting a post-publication concern regarding the article cited above. Because the findings could inform lipid-management decisions, accurate statistical reporting is essential. An independent re-analysis (code and figure checks) identifies material issues that may mislead readers. I respectfully request editorial assessment and correction.

Each concern is documented with evidence and reproduced checks in the attached Appendix (with code in the repository).

Critical issues

- **Figure mislabeling and incorrect IQR shading (C1–C3)**: Figure 1B's y-axis is mislabeled (0–10 % vs the correct 0–20 %), halving both absolute plaque burden and within-person change; Figures 1A–1B IQR bands do not match the sample IQR; Figures 2E–2F y-axis mislabeled; should be –1 to 6 (not –1 to 4).
- **Claims exceed reported models (C4)**: The manuscript asserts no association between ApoB/LDL-C (baseline, change, or "exposure") and plaque change (including TPS), yet several required models—particularly Δ-TPS vs LDL-C/ApoB/exposure—are not reported.
- **Linear-model assumptions violated (C5)**: Residual diagnostics show non-linearity, heteroskedasticity, and non-normality in the change-score regressions, contradicting the manuscript's claim that assumptions were met.

Major issues (M1–M3)

(i) **Model strategy & multiplicity**: departure from standard practice[1,2], univariable change-scores used instead of baseline-adjusted follow-up (ANCOVA)[3,4]; absence of a prespecified adjustment set/multiplicity plan; predictive wording for associative findings.
(ii) **Bayes-factor analysis**: Added post-hoc, employs an extra-wide r-scale[5] without standard sensitivity analysis[6], and overstates BF of 6–10 as posterior probability[7].
(iii) **Age "mediation"**: Age is a confounder; treating it as a mediator and conditioning on CAC (a downstream variable) is not mediation[8].

Additional items (O-series)—including the lifetime-exposure unit mismatch and the non-standard percent-change metric—are detailed in the Appendix.

Requested editorial actions

1. **Figure corrections**: Replot Figures 1A–1B and 2E–2F with correct y-axes and IQR shading; verify all figure captions against the underlying data.
2. **Re-analysis & full reporting**: Refit baseline-adjusted (ANCOVA) models for every claimed association, supply coefficients/CIs/diagnostics, run Bayes-factor prior-sensitivity, and address the additional modeling and data issues detailed in the Appendix (M1–M3, O-series).
3. **Independent statistical review**: Given the scope of figure and modelling issues, I respectfully request that the journal obtain an external statistical review and, depending on the findings, issue the appropriate notice (e.g., Author Correction or stronger).

Materials provided: Appendix (PDF, attached); reproducibility repository (code/figures/README): https://github.com/your-org/keto-cta-review

I have no conflicts of interest related to this work and am available to provide any additional information.

Sincerely,

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Appendix

Summary Table of Critical and Major Issues

ID	Area	Core issue	Severity	Ref.
C1–C3	Figures	Incorrect y-axis (Fig 1B, 2E–F); incorrect IQR shading (1A–B)	Critical	C1, C2, C3
C4	Results claims	Claims exceed reported models; Δ-TPS models missing	Critical	C4
C5	Model diagnostics	Linear-model assumptions violated	Critical	C5
M1	Model strategy & multiplicity	Univariable Δ focus; no multiplicity; predictive wording	Major	M1
M2	Methods (Bayes)	Multiple BF issues (r-scale, no sensitivity, misinterpretation)	Major	M2
M3	Age "mediation" analysis	Age is a confounder; CAC downstream of exposure	Major	M3

Data provenance. Individual-level plaque metrics used for reproductions were obtained from the Citizen Science Foundation keto-CTA repository (accessed Aug 23, 2025).[9]

Critical (C1–C5)

C1 and C2 reference panels from *Figure 1 Individual Change in Plaque Volume* of the published article (p. 6); left-hand images are published panels, right-hand images are reproductions.

C1 — Figure 1B: y-axis mislabeled

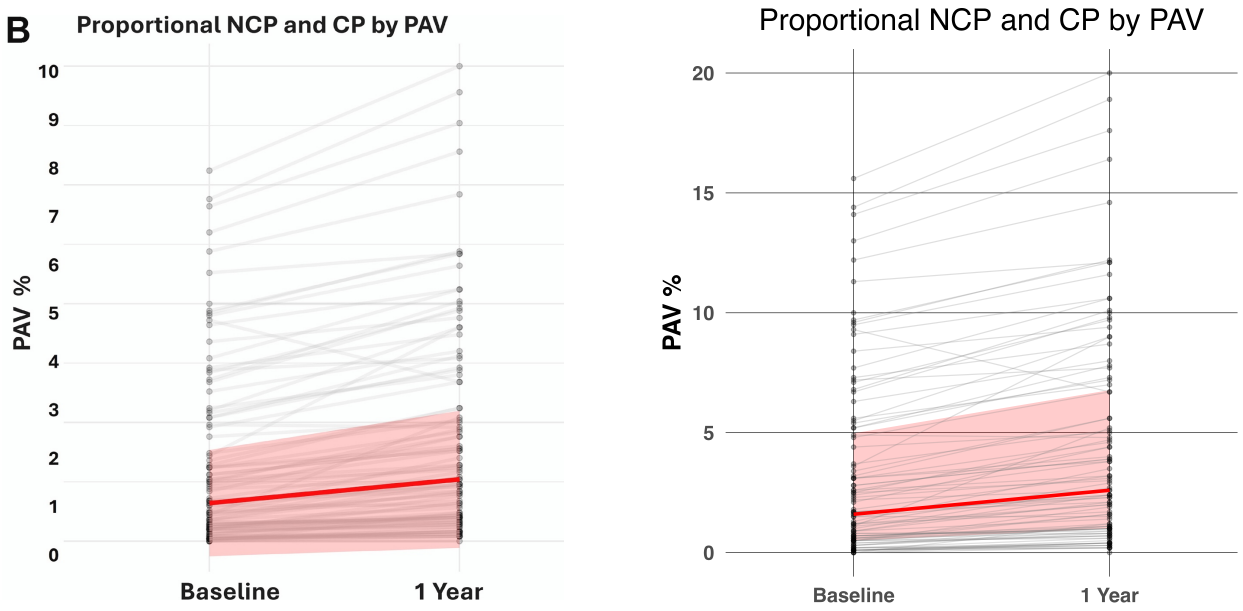

Figure 1: Left: published (axis 0–10%; IQR band mismatched). Right: reproduced (axis 0–20%; IQR ≈ 0.3%–1.7%).

The published panel's y-axis label is inconsistent with the plotted scale. This reproduction uses the correct 0–20% range. The underlying data are unchanged.

Requested correction. Update the Figure 1B y-axis label to **0–20%**.

C2 — Figures 1A & 1B: shaded IQR band incorrect; caption unclear

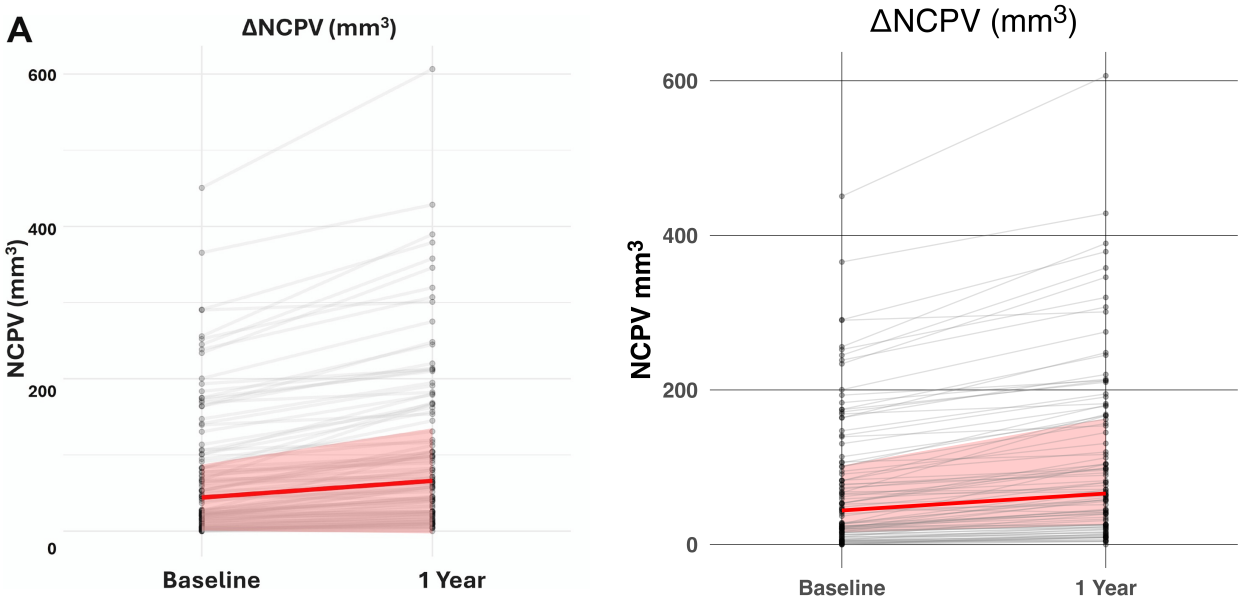

Figure 2: Left: published (IQR shading does not match the sample IQR). Right: reproduced (median $\approx$ 18.9 mm^3; IQR $\approx$ 9.3–47.0 mm^3).

The shaded band is described as the interquartile range (IQR) of **change** (median 0.8%, IQR 0.3%–1.7%). If so, the band should lie entirely **above 0%** (the 25th percentile is 0.3%). In the published panels the band is anchored at 0% in Figure 1A and dips **below 0%** in Figure 1B, which is inconsistent with the reported quantiles and cannot occur if the band truly represents the IQR of paired change. In both panels, the published shaded band differs materially from the reproduced band that plots the IQR of *paired* within-person change: the published band is anchored at (1A) or dips below (1B) 0%, whereas the reproduced band lies entirely above 0% and matches the stated quantiles. This indicates the published shading is not the IQR of paired change.

Requested correction.

1. Re-compute and plot the **interquartile range of paired within-person change** from baseline to 1 year (25th–75th percentiles).
2. Revise the caption for both panels to read: "The red line shows the median paired change from baseline to 1 year; the shaded band shows the interquartile range (25th–75th percentiles) of paired change from baseline to 1 year."

C3 — Figures 2D–2F: y-axis mislabeled

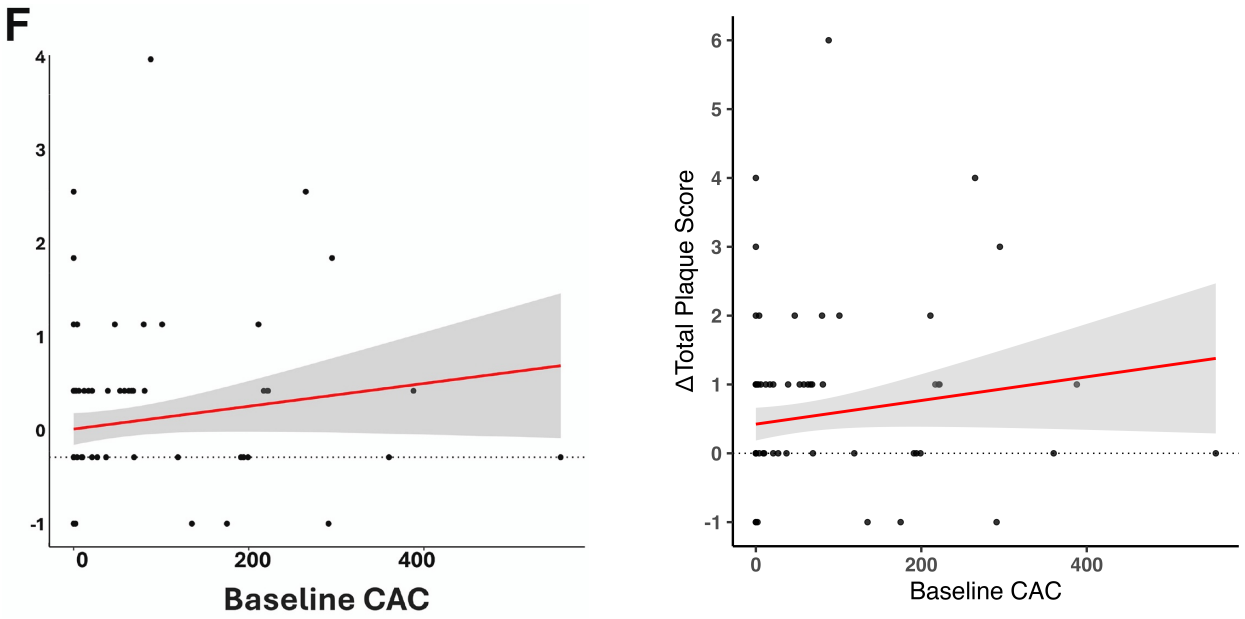

Figure 3: Left: published (y-axis **labeled** −1–4 while the plotted range/ticks correspond to **−1–6**). Right: reproduced (axis −1–6); OLS $lm(y \sim x)$ line with SE band.

Similar to Figure 1B, Panel 2F published panel's y-axis label is inconsistent with the plotted scale. The label indicates (-1)–4, but the scale used is (-1)–6 (all data are shown). The same mislabeling is visible in panels **2D** and **2E** (not shown). This can mislead readers about the plotted domain and relative spread.

Requested correction.
Correct the y-axis **label** to reflect the actual plotted range in each panel (e.g., (-1)–6).

Panels 2D and 2E exhibit the same label–range mismatch; 2F is shown as the example.

C4 — Conclusions exceed support from reported models

Quoted claims (paper): "In lean metabolically healthy people on KD, neither total exposure nor changes in baseline levels of ApoB and LDL-C were associated with changes in plaque." (p. 1)

"… no association between NCPV vs LDL-C or ApoB and **TPS** vs LDL-C or ApoB." (p. 6)

"changes in and baseline levels of ApoB were not associated with changes in NCPV or TPS as measured by CCTA." (p. 8)

These statements bundle multiple non-equivalent associations: change vs. exposure vs. baseline level as predictors, and NCPV and TPS as separate outcomes, each requiring its own prespecified model; several of those models are not reported. The audit table below maps each claim to the model it implies and whether it was reported.

Claim → model → reported? (audit checklist):

Abstract/Results component	Required model (example)	Reported in paper?
Δ-plaque vs ΔLDL-C	Δ-NCPV ~ Δ-LDL-C	Not reported
Δ-plaque vs LDL-C **exposure**	Δ-NCPV ~ LDL-C exposure	Not reported
Δ-plaque vs LDL-C **baseline**	Δ-NCPV ~ LDL-C baseline	Not reported
Δ-plaque vs ΔApoB	Δ-NCPV ~ Δ-ApoB	Reported
Δ-plaque vs ApoB **exposure**	Δ-NCPV ~ ApoB exposure	Not reported
Δ-plaque vs ApoB **baseline**	Δ-NCPV ~ ApoB baseline	Reported
Δ-TPS vs LDL-C / ApoB / exposure	Δ-TPS ~ (LDL-C or ApoB or exposure)	**Not reported**

A review of the paper's Model Results table confirms these omissions: the Model Results table lists selected Δ-NCPV regressions but **does not** include any Δ-TPS regressions or models using LDL-C baseline or ApoB exposure as independent variables. They are also not present in any supplementary material.

TABLE 3 Model Results

	β	*P* Value	R^2	BF
Estimated LDL-C exposure				
$NCPV_{final}$ ~ $LDL\text{-}C_{exp}$	0.00	0.88	−0.01	(01) 9.9
PAV_{final} ~ $LDL\text{-}C_{exp}$	0.00	0.73	−0.01	(01) 9.1
ApoB				
ΔNCPV ~ ΔApoB	0.01	0.91	−0.01	(01) >10.0
ΔNCPV ~ ApoB	0.06	0.33	−0.00	(01) 6.3
Plaque Metrics				
ΔNCPV ~ CAC_{bl}	0.18	<0.001	0.33	(10) >10.0[a]
ΔNCPV ~ $NCPV_{bl}$	0.25	<0.001	0.49	(10) >10.0[a]
ΔNCPV ~ PAV_{bl}	5.5	<0.001	0.43	(10) >10.0[a]
ΔNCPV ~ TPS_{bl}	7.4	<0.001	0.37	(10) >10.0[a]
ΔNCPV ~ CAC_{bl}* ΔApoB				
CAC_{bl}	0.18	<0.001	0.33	N/A
ΔApoB	0.08	0.20	N/A	N/A
CAC_{bl}: ΔApoB	−0.00	0.23	N/A	N/A
Saturated fat				
ΔNCPV ~ Saturated fat intake	−0.05	0.77	−0.01	(01) 9.8
ApoB ~ Saturated fat intake	−0.03	0.90	−0.01	(01) >10.0
Age mediation analysis				
$NCPV_{final}$ ~ Age	3.0	0.004	0.07	(01) 0.3
$NCPV_{final}$ ~ Age + $Life\text{-}LDL\text{-}C_{exp}$	1.93 | 0.01	0.13 | 0.16	0.07	(01) 0.3 | 0.3
$NCPV_{final}$ ~ Age + $Life\text{-}LDL\text{-}C_{exp}$ + CAC_{bl}	0.02 | 0.01 | 0.73	0.98 | 0.15 | <0.001	0.47	(01) 2.7 | 3.1 | >10.0[a]

[a]Models on CAC are provided for the alternative hypothesis (10). All other models are provided for the null hypothesis (01).

β = estimate (slope magnitude); ΔNCPV = change in noncalcified plaque volume; ApoB = apolipoprotein B; ApoB = ApoB on a ketogenic diet; ΔApoB = ApoB change during the study; BF = Bayes factor; CAC_{bl} = CAC at baseline; $LDL\text{-}C_{exp}$ = LDL-C exposure while on a ketogenic diet (mean 5.7 y); $Life\text{-}LDL\text{-}C_{exp}$ = LDL-C exposure over life course to date; $NCPV_{final}$ = noncalcified plaque volume at the end of the study; PAV = percent atheroma volume; R^2 = squared correlation coefficient (explained variability); TPS_{final} = total plaque score at the end of the study; other abbreviations as in Table 1.

Figure 4: C4a. "Model Results" / from Table 3 excerpt — no ΔTPS models are reported.

Requested fix: Provide fitted models (coefficients, CIs, diagnostics) for all claimed relationships—especially **Δ-TPS** vs LDL-C/ApoB/exposure—or amend the conclusions.

C5 — Model assumptions: diagnostics indicate violations

Scope. Only plaque metrics were available to audit (CAC, NCPV, TPS, PAV; see **Data provenance**), so lipid/demographic covariates (ApoB, LDL-C, age, sex, BP) were not available. Four univariable change-score models were tested: $\Delta\text{NCPV} \sim \text{CAC}_{bl}$, $\Delta\text{NCPV} \sim \text{NCPV}_{bl}$, $\Delta\text{NCPV} \sim \text{PAV}_{bl}$, $\Delta\text{NCPV} \sim \text{TPS}_{bl}$.

Quoted claim (paper). "All linear model assumptions were corroborated with the R function `performance::check_model`." (p. 3)

Objective diagnostics. Linearity (Ramsey RESET), constant variance (Breusch–Pagan), residual normality (Shapiro–Wilk); $\alpha = 0.05$.

Model	β	Linearity	Constant Variance	Residual Normality
ΔNCPV ~ CAC_{bl}	β = 0.18 p = <0.001	**Violation** p = 0.031	**Violation** p = 0.001	**Violation** p = <0.001
ΔNCPV ~ NCPV_{bl}	β = 0.25 p = <0.001	**OK** p = 0.198	**Violation** p = <0.001	**Violation** p = <0.001
ΔNCPV ~ PAV_{bl}	β = 5.48 p = <0.001	**Borderline** p = 0.050	**Violation** p = <0.001	**Violation** p = <0.001
ΔNCPV ~ TPS_{bl}	β = 7.37 p = <0.001	**OK** p = 0.132	**Violation** p = <0.001	**Violation** p = 0.001

Figure 5: C5a. Objective tests for four ΔNCPV models — each fails ≥2 assumptions.

Results of diagnostics. Breusch–Pagan rejected homoscedasticity in **all four** models ($p < 0.001$); Shapiro–Wilk rejected residual normality in **all four** (p 0.001); RESET indicated misspecification for $\Delta\text{NCPV} \sim \text{CAC}_{bl}$ ($p = 0.031$) and was borderline for $\Delta\text{NCPV} \sim \text{PAV}_{bl}$ ($p = 0.050$). **Each model fails 2 assumption checks.** Accordingly, the blanket statement that "all assumptions were corroborated" is **not supported** for these models.

Generalizability. These checks cover plaque-only change-score regressions because of data availability. Given the similar functional form and residual structure, **similar violations are plausible** for other reported change-score regressions (e.g., with ApoB/LDL-C), but cannot be verified without those covariates.

Author response (post-publication). In response to a letter, the authors stated that they reran all models using robust linear regression (`MASS::rlm`) and that estimates were "consistent" with the published results[10].

Interpretation. M-estimator "robust regression" (e.g., `MASS::rlm`) downweights outliers/heavy tails but does **not** remedy **nonlinearity**, **heteroskedasticity**, or **non-normal residuals**; it also does not supply standard p-values by default. Appropriate fixes require model specification (e.g., splines/transformations) and, where variance is non-constant, **heteroskedasticity-consistent (HC/sandwich) SEs**. Robust regression alone does not address the violations indicated here.[11,12]

Requested fix. Report assumption checks for **all** fitted models; where violations occur, fit appropriately specified models (e.g., baseline-adjusted follow-up with nonlinear terms; transformations where justified; HC/sandwich SEs) rather than relying solely on robust regression, and include diagnostics with the corrected analyses.

Major (M1–M3)

M1 — Model strategy, multiplicity, and claim wording

> "Linear models on the primary (NCPV) and secondary outcomes were univariable" (p. 3)

Evidence (paper). Methods describe univariable linear models; Table 3 and accompanying text report numerous univariable regressions across outcomes/exposures. The narrative alternates between "predicts/predictor" and "associated with," with a single late "exploratory" caveat. No baseline-adjusted follow-up models (ANCOVA) or prespecified covariate set are reported.

Issue (affects interpretation).

- Primary reliance on univariable change-score models invites confounding (e.g., age, sex) and mathematical coupling with baseline[3,4]; Δ compounds measurement error and reduces power. Baseline imbalance and regression-to-the-mean can inflate, attenuate, or reverse associations.
- The approach departs from standard practice[1,2] for progression analyses , where follow-up is modeled as a function of baseline and covariates (ANCOVA) to obtain clinically interpretable effects at a given starting plaque burden.
- Multiplicity is not addressed despite many tests; without a plan, familywise/type-I error is inflated and false positives are expected.
- "Predicts/predictor" implies prognostic (prediction)[13] standards—multivariable specification, internal validation, calibration/discrimination—which are not presented. If claims are associational, wording should reflect association rather than prediction.

Requested action.

1. **Clarify analytic aim**: state consistently whether the analyses are predictive or associative in the Title, Abstract, Methods, and Results.
2. **If association is the aim**:
   - Prespecify a primary adjustment set (baseline NCPV/TPS/CAC, age, sex).
   - Report baseline-adjusted follow-up models (ANCOVA) as the main analysis with effect estimates + 95 % CIs; move univariable Δ-models to the Supplement.
   - Provide full diagnostics (linearity, residuals, influence; see C5).
   - Add a post-hoc power (precision) analysis so readers can judge whether the study could detect clinically meaningful ApoB effects (e.g., 5 mm^3 NCPV or 0.5 % PAV change).
3. **If prediction is the aim**:
   - Present a multivariable prognostic model with internal validation (bootstrap or k-fold).
   - Report calibration and discrimination; do not rely on univariable screens as evidence.
4. **Multiplicity control**: specify a strategy (e.g., Holm or FDR) across related tests and emphasise effect estimates with uncertainty rather than dichotomous "significance."

M2 — Bayes-factor analysis: extra-wide r-scale, no sensitivity, misinterpreted evidence

Quoted claims (paper).

> "the addition of Bayesian inference adds credence to finding that there is no association between NCPV vs LDL-C or ApoB and TPS vs LDL-C or ApoB." (p. 6)

> "Bayes factors were calculated using BayesFactor::regressionBF with default settings and an rscale value of 0.8 to contrast a moderately informative prior with a conservative distribution width" (p. 3)

> "these data suggest it is 6 to 10 times more likely that the hypothesis of no association between these variables (the null) is true as compared to the alternative." (p. 6)

Issue (multiple)

- The paper's sole rationale for adding a Bayes-factor analysis is that it "adds credence to finding there is no association", is circular: a BF has evidential meaning only when the prior and analysis plan are fixed *a priori*.
- `regressionBF` run with `rscale = 0.8`, called "moderately informative."
  - Package defaults: "medium" = 0.354, "wide" = 0.5, "ultrawide" = 0.707; **0.8 is wider than "ultrawide."**[5]
- The same extra-wide r-scale is applied to every model without justification
- No prior-sensitivity analysis (a standard recommendation for Bayes-factor reporting[6]) and no indication the BF test was pre-specified.
- Stating that a BF of 6 to 10 makes the null "6–10 times more likely confuses a Bayes factor (likelihood ratio) with posterior probability. Such a claim requires explicit prior odds.[7]

Requested action

1. Re-compute Bayes factors with default r-scales (0.354, 0.5, 0.707) **and** (r = 0.8); report all values.
2. Justify the prior for each model or adopt a standard scale.
3. Indicate whether the BF analysis (and r-scale choice) was pre-specified; if post-hoc, label it exploratory and provide the rationale for adding it
4. Revise manuscript text to avoid equating BF with the probability the null is true unless prior odds are specified.

M3 — Age mediation analysis: causal language and improper adjustment

Age mediation analysis		
$NCPV_{final}$ ~ Age	3.0	0.004
$NCPV_{final}$ ~ Age + Life-LDL-C_{exp}	1.93 | 0.01	0.13 | 0.16
$NCPV_{final}$ ~ Age + Life-LDL-C_{exp} + CAC_{bl}	0.02 | 0.01 | 0.73	0.98 | 0.15 | <0.001

Figure 6: M2a. Table 3 (excerpt) from the published article showing the sequential models ("univariable," "+ age," "+ baseline CAC").

Quoted claim (paper): "There was no association between LDL-C exposure while on a KD (mean 5.7 years) and NCPV or TPS (Figure 2G, Table 3). Estimated lifetime LDL-C exposure was only a significant predictor of final NCPV in the univariable analysis but lost significance when age was included as a covariate (Table 3). Both age and lifetime LDL-C exposure lost significance when baseline CAC was included in the model (Table 3)." (p. 4)

Issue (Major).
Age is a **confounder** (not caused by LDL), and baseline **CAC** is plausibly **downstream** of lifetime LDL exposure. Conditioning on CAC when asking about the *total* effect of lifetime LDL on NCPV blocks the LDL → CAC → NCPV pathway and yields only a **direct effect**; it does not constitute a mediation analysis[8]. No mediator model or **indirect effect with CI** was reported. Given that the paper's lifetime-LDL metric embeds age (see M3), collinearity is expected and "loss of significance" is not evidence of no association.

Requested fix.
Specify the causal estimand with a DAG. If mediation is intended: fit **Mediator ~ Exposure + Age** and **Outcome ~ Exposure + Mediator + Age** and report **indirect/direct/total** effects with CIs. If the target is the **total effect** of lifetime LDL on NCPV, do **not** adjust for baseline CAC; report models with/without age (confounder), with diagnostics and collinearity checks.

Other — Additional analysis & reporting issues

ID	Sev.	Location	Issue (concise)	Ref.	Requested action
O1	Other	Methods	Percent change reported as **ratio of medians**, not per-participant	§O1	Provide per-subject % change (or both) with summary + CI
O2	Other	Measurements	"Lifetime LDL-C exposure" mixes time units (days + years)	§O2	Recalculate exposure with a single time unit
O3	Other	Data/QC	**Inconsistent durations** distort exposure denominators	§O3	Audit durations; rerun exposure-based analyses
O4	Other	Table 1	**Impossible IQR** for total cholesterol (301–337 mg/dL; median = 338 mg/dL)	§O4	Correct IQR; verify all baseline stats & units

O1 — Percent-change metric: ratio of medians, not per-subject

> "The median change in NCPV was 18.9 mm3 (IQR: 9.3-47.0 mm3) and the median change in PAV was 0.8% (IQR: 0.3%-1.7%). Compared to baseline, these represent a **43% and 50% change**, respectively." (p. 4)

Issue

The paper's "43%" (NCPV) and "50 %" (PAV) values use a **non-standard percent-change metric**, a *ratio of medians*:

$$\frac{\text{median}(\Delta)}{\text{median}(\text{baseline})} \times 100\%.$$

This definition differs from the customary per-participant percent change and can under- or overstate the typical effect (see table below).

Outcome	Ratio-of-medians	Median % change (per subject)	Mean % change
NCPV	43 %	49.2 %	81.4 %
PAV	50 %	47.3 %	80.7 %

(participants with baseline = 0 excluded from % change calculations)

Requested action

1. Report per-subject percent change

$$\%\Delta X_i = \frac{X_{1\text{y},i} - X_{bl,i}}{X_{bl,i}} \times 100$$

with median, IQR, and 95 % CI.

2. Clarify that the published 43 % / 50 % are ratios of medians.
3. State how zero baselines were handled.

O2 — Exposure metric: construction and units inconsistent

Quoted method (paper). "LDL-C exposure on a KD was calculated by summing the products of the reported days on a KD prior to study commencement and baseline LDL-C on a KD plus the study follow-up days by their final LDL-C. Estimated **lifelong** LDL-C additionally included the product of **age** upon commencing a KD and pre-KD LDL-C." (p. 3)

$$\mathrm{LDL}_{\text{KD-exp}} = \mathit{Days}_{\mathrm{KD}} \cdot \mathrm{LDL}_{\text{baseline}} + \mathit{Days}_{\mathrm{FU}} \cdot \mathrm{LDL}_{\text{final}}$$

Unit: $\mathrm{mg} \cdot \mathrm{day/dL}$

$$\mathrm{LifeLDL}_{\text{exp}} = \mathrm{LDL}_{\text{KD-exp}} + \mathit{Age}_{\text{KD-start}} \cdot \mathrm{LDL}_{\text{preKD}}$$

Unit (as written): dimensionally inconsistent

Issue

- **Dimensional inconsistency:** the "lifetime" sum mixes **days** and **years**; these are not commensurate.
- **Scale distortion:** the pre-KD term is ~**1/365** the magnitude of the day-based terms unless age is converted; if converted to days, the term becomes a near-linear **age surrogate** times one LDL value.
- **Interpretability:** model coefficients are not in a single unit ($\mathrm{mg} \cdot \mathrm{day/dL} + \mathrm{mg} \cdot \mathrm{year/dL}$), hindering effect-size meaning and comparisons.

Requested fix. Use a single time unit (e.g., convert all time to **days** and label units), report a sensitivity **dropping the pre-KD term**, and consider also reporting the **time-weighted mean LDL**:

$$\overline{\mathrm{LDL}} = \frac{\sum_i \mathrm{LDL}_i \, \Delta t_i}{\sum_i \Delta t_i}$$

alongside total duration.

O3 — Exposure durations inconsistent across study

Issue

- Table 1 KD duration: **1,642.7 days (~4.5 y)**.
- Table 3 caption: "LDL-C exposure … mean **5.7 y**."
- Abstract median KD duration: **1,302 days**

Discrepant durations feed into exposure metrics and bias effect estimates.

Requested action

1. Audit raw duration variables; reconcile KD start date, follow-up days, and exposure windows.

2. Re-calculate exposure metrics (e.g., time-weighted mean LDL) and rerun analyses with corrected durations.
3. Report both mean and median durations in consistent units (days or years).

O4 — Table 1 Baseline Characteristics incorrect IQR

> "Total cholesterol (mg/dL) 355.1 ± 89.9; median 338 (IQR 301–**337**)" — Table 1

Issue
The reported median (338 mg/dL) lies **outside** the stated IQR (301–337 mg/dL), which is mathematically impossible.

Requested action
Correct the IQR and verify all baseline statistics (units, rounding, transcription).

References

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