

Ultra-Processed Food Consumption, Sleep Patterns, and Cardiometabolic Risk Among Young Adults: A Harmonized Comparative Framework for the United Kingdom and Ghana

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Abstract

Background: The joint distribution of ultra-processed food (UPF) consumption, sleep behaviour and cardiometabolic risk is poorly characterised in comparable young-adult samples across the United Kingdom and Ghana.

Objective: To develop a harmonised cross-country epidemiological protocol and a technically explicit analytical strategy for estimating nonlinear exposure–outcome associations, between-setting heterogeneity and measurement uncertainty.

Methods: The proposed cross-sectional field design targets adults aged 18–35 years and integrates repeated NOVA-coded dietary recall, sleep timing and Pittsburgh Sleep Quality Index assessment, anthropometry, blood pressure and glycaemic measurements. An existing synthetic dataset of 2,400 records, divided evenly between country-labelled scenarios, is retained strictly to demonstrate the analysis workflow. The revised framework introduces latent usual-intake models, spline-based and hierarchical generalised models, flexible sleep interaction structures, multiple imputation, measurement invariance checks and quantitative bias analysis. Results: Six existing simulation figures illustrate predetermined scenario differences and correlations; none constitute observed UK or Ghana estimates. The expanded models are specified for future application but have not been empirically fitted to participant data.

Conclusions: A harmonised, pre-registered design can test whether UPF–cardiometabolic associations vary across sleep profiles and food environments. Representative recruitment, ethics approvals and independent model validation are prerequisites for population claims.

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Keywords: ultra-processed foods, NOVA, young adults, sleep, cardiometabolic risk, United Kingdom, Ghana, nonlinear epidemiology, measurement error, hierarchical regression

1. Introduction

1.1. Epidemiological Background

Cardiometabolic vulnerability begins to accumulate during early adulthood, when diet, employment schedules, sleep timing and socioeconomic circumstances interact. The NOVA system classifies foods by the nature and purpose of industrial processing, and category 4 identifies industrial formulations that cannot simply be inferred from nutrient content (Monteiro *et al*., 2019) [39]. Global food-system transitions have increased the relevance of this distinction (Baker *et al*., 2020) [5]. Evidence syntheses report associations between greater UPF exposure and several adverse health endpoints, while substantial variation persists across study designs, exposures and outcome definitions (Lane *et al*., 2024; Barbaresko *et al*., 2025; Idowu, *et al*., 2025; Odozor, *et al*., 2025) [9, 29, 34, 42].

Controlled feeding research provides a plausible behavioural mechanism: adults offered an ultra-processed diet consumed more energy than during an unprocessed diet under experimentally controlled conditions (Hall *et al*., 2019) [27]. That experiment does not establish that all UPF categories have equivalent metabolic consequences. Eating rate, dietary energy density, food matrices and nutrient substitutions warrant separate assessment (Fazzino *et al*., 2023; Vadiveloo & Gardner, 2023) [20, 53].

1.2. Rationale for Comparison

The United Kingdom and Ghana provide contrasting retail environments, food histories and patterns of packaged-food availability. However, country differences cannot be inferred from unweighted convenience samples. Ghana's national STEPS surveillance provides contextual risk-factor information, but not a harmonised individual-level file of NOVA-classified food intake, sleep scores and laboratory indicators for the proposed age group (World Health Organization [WHO], 2024) [55]. Accordingly, this work specifies comparable field instruments, transparent sampling and a country-by-exposure interaction rather than presuming that one setting has higher underlying risk.

1.3. Sleep as Co-Exposure and Modifier

Sleep is linked with appetite regulation, glucose metabolism and behavioural routines. The Pittsburgh Sleep Quality Index (PSQI) measures several dimensions of perceived sleep quality (Buysse *et al*., 1989) [10]. Reviews also report associations between UPF exposure and sleep-related outcomes, although directionality remains unresolved (Delpino *et al*., 2023) [14]. We therefore distinguish effect modification from mediation: cross-sectional covariation alone cannot establish a pathway from UPF intake through sleep to subsequent cardiometabolic disease.

1.4. Objectives and Research Contribution

The primary objective is to estimate the adjusted association between usual UPF energy share and a pre-specified cardiometabolic endpoint separately for each country, permitting nonlinear gradients. Secondary objectives are to examine sleep duration and PSQI jointly, test country and sleep interactions, quantify food-coding uncertainty, and evaluate how site clustering, missingness and selection could alter the estimates. This revision treats models as a coherent inferential framework rather than as a collection of equations divorced from the study design.

2. Literature Review and Conceptual Framework

2.1. Processing-Based Dietary Classification

NOVA 4 coding requires ingredient-level review and consistent decisions for composite dishes, branded products and changing formulations (Monteiro *et al*., 2018, 2019) [39, 40]. It is therefore essential to measure sodium, free sugars, saturated fat, fibre, total energy and dietary affordability in parallel. Reviews caution that nutritional adequacy and processing classification are not interchangeable (Gibney *et al*., 2017; Scrinis & Monteiro, 2022) [25, 50]. Idoko *et al*. 2024; Bamilede, *et al*., 2025 evaluated wheat varieties and processing methods in relation to diabetes risk. Their review provides contextual support for separating food-processing operations from the metabolic properties of foods; it did not estimate UPF–sleep associations in UK or Ghanaian young adults.

2.2. Metabolic and Behavioural Pathways

Prospective and pooled analyses associate UPF intake with type 2 diabetes, hypertension and adverse cardiometabolic endpoints, subject to confounding and varying operational definitions (Chen *et al*., 2023; Wang *et al*., 2022; Shu *et al*., 2023) [13, 51, 54]. Controlled feeding evidence demonstrates short-term energy-intake differences, while mechanistic work concerning emulsifiers and host–microbiota responses remains hypothesis-generating for population-level dietary pathways (Hall *et al*., 2019; Chassaing *et al*., 2022; Frank, 2026) [12, 22, 27]. A flexible epidemiological model should consequently allow non-monotonic exposure functions, interactions with sleep and uncertainty in the measured exposure.

2.3. Socioeconomic Context and Measurement Comparability

Food price, household resources, shift work and urbanicity may jointly determine diet and sleep. Godwins *et al*. (2024) [26] examined cleaner energy, food security and nutritional outcomes, a contextual contribution rather than direct evidence on the current UPF–sleep question. Their article was co-authored by Amina Catherine Ijiga, a distinct researcher from Onuh Matthew Ijiga. The present manuscript does not merge these author identities. For valid country comparisons, translation and cognitive testing of questionnaires should be followed by tests of differential item functioning and instrument reliability.

2.4. Causal Structure and Research Gap

A conceptual directed acyclic graph would connect socioeconomic circumstances, baseline health, work schedules and physical activity to both UPF exposure and cardiometabolic measurements, with sleep acting potentially as an antecedent, co-exposure or consequence. The inferential target is therefore an adjusted association and its conditional heterogeneity, not an identified causal effect (Ogundola, 2024, 2026) [45]. Otugene *et al*. (2024) [47] illustrate how model assumptions and sensitivity analyses can be made explicit in mathematical epidemiology; their infectious-disease model is not direct evidence about nutrition or sleep.

3. Materials and Methods

3.1. Design, Sampling and Estimands

The proposed observational study recruits adults aged 18–35 years from urban and peri-urban sites in the United Kingdom and Ghana. Sampling frames should be stratified by age, sex and socioeconomic category, and site-level response fractions recorded. Exclusion from the primary composite analysis of pregnancy is planned because physiologic interpretation differs; chronic disease and medication history are instead measured. The target association is the change in marginal cardiometabolic risk under a hypothetical ten-percentage-point difference in usual UPF energy share at comparable measured covariate profiles. It must not be labelled a causal effect without stronger identification assumptions.

Let h index sampling strata and k index recruitment clusters. To capture both finite population correction and cluster inflation, the approximate design-based recruitment requirement is expressed in Equation (1).

$$n_{\text{target}} = \frac{z^2_{1-\alpha/2}p(1-p)}{d^2}\,\frac{1+(m-1)\rho}{r}\,\frac{N-n_0}{N-1} \quad (1)$$

Here $z_{1-\alpha/2}$ is the normal critical value, p the anticipated prevalence, d the desired half-width, m the expected cluster size, ρ the intracluster correlation, r the response fraction, N the eligible sampling population and n_0 the uncorrected simple-random-sample target. Equation (1) is a planning approximation; power for spline and interaction tests must be evaluated by simulation and will generally require more participants.

Because site selection and nonresponse could distort the combined sample, inverse probability weights will be calibrated to known target-population margins using Equation (2).

$$w_i^* = \frac{1}{\pi_{h(i)}\,\pi_{k(i)|h(i)}\,\hat{r}_i}\exp(\boldsymbol{\lambda}^{\top}\mathbf{z}_i) \quad (2)$$

In Equation (2), π denotes stratum and conditional cluster selection probabilities, $\hat{r}_i$ the fitted response probability, z_i the calibration-variable vector and λ the vector chosen so weighted population margins match external totals. These parameters will be estimated only after actual recruitment.

3.2. Dietary Intake and Latent Usual-Exposure Estimation

Two or more non-consecutive 24-hour recalls, including a non-workday when feasible, will record item identity, ingredients, quantities and energy intake. Duplicate NOVA coding will resolve ambiguous products using a documented adjudication protocol. The observed share can be calculated by Equation (3), explicitly retaining total-energy denominators.

$$U_{it} = 100\,\frac{\sum_{j=1}^{J_{it}} E_{ijt}\,\mathbb{I}(C_{ijt}=4)}{\sum_{j=1}^{J_{it}} E_{ijt}} \quad (3)$$

Here U_{it} is observed UPF energy percentage for person i on day t, J_{it} the number of reported items, E_{ijt} the energy from item j, C_{ijt} the adjudicated NOVA category and I(·) the indicator function. Equation (3) yields a day-specific measure, not necessarily a person's usual consumption.

Rather than simply averaging recalls and ignoring attenuation, a mixed-effects latent measurement model can estimate usual exposure while allowing differential reporting by country. Equation (4) defines a logit-scale observation process for shares constrained to (0, 1).

$$\operatorname{logit}\left(\frac{U_{it}}{100}\right) = \eta_i + \mathbf{a}_{it}^{\top}\boldsymbol{\delta} + b_{s(i)} + \epsilon_{it}, \quad \eta_i \sim \mathcal{N}\left(\mu_{c(i)} + \mathbf{x}_i^{\top}\boldsymbol{\gamma}, \tau^2_{c(i)}\right) \quad (4)$$

In Equation (4), η_i is participant i's latent usual logit UPF share; a_{it} captures recall-day conditions; δ and γ are regression coefficients; $b_{s(i)}$ is the site random intercept; ε_{it} is day-level error; μ_c and τ²_c are country-specific mean and latent variance; x_i is the measured covariate vector. Zero and 100 percent observations require a pre-specified boundary model rather than arbitrary silent truncation.

An important extension is probabilistic NOVA adjudication. If item coding is uncertain, Equation (5) integrates the coder posterior probability of NOVA 4 status rather than treating all disputed products as certain.

$$\tilde{U}_{it} = 100\,\frac{\sum_j E_{ijt}\Pr(C_{ijt}=4|\mathbf{q}_{ijt})}{\sum_j E_{ijt}} \quad (5)$$

The symbol q_{ijt} denotes recorded ingredients, product attributes and adjudication evidence; Pr(·) is the item-level probability of NOVA 4 membership. The difference between U_{it} and $\tilde{U}_{it}$ defines one planned classification-sensitivity analysis. Neither posterior coding probabilities nor model parameters are yet estimated.

3.3. Sleep Measurement and Instrument Equivalence

Respondents will report workday and non-workday bed and wake times, shift schedules, and PSQI components. The weekly sleep mean is a weighted average of observed sleep periods, but irregular employment requires participant-specific day weights; this is made explicit in Equation (6).

$$S_i = \frac{\sum_{t=1}^{T_i}\omega_{it}\left(t_{it}^{\text{wake}} - t_{it}^{\text{bed}} - A_{it}\right)}{\sum_{t=1}^{T_i}\omega_{it}} \quad (6)$$

Here S_i is estimated hours slept per night; t^{Wake}_{it} and t^{bed}_{it} are waking and bedtime timestamps on a continuous time axis, A_{it} is wakefulness during the sleep interval and ω_{it} is the number of nights represented by the reported schedule. In implementation, raw dates and time zones must be used to avoid overnight-clock subtraction errors.

Because Ghanaian and UK respondents may interpret questionnaire items differently, confirmatory-factor measurement invariance will be evaluated through Equation (7), with nested tests of loadings and thresholds across groups.

$$\mathbf{y}_{ig} = \boldsymbol{\nu}_g + \boldsymbol{\Lambda}_g\boldsymbol{\xi}_{ig} + \boldsymbol{\varepsilon}_{ig}, \qquad \operatorname{Cov}(\boldsymbol{\varepsilon}_{ig}) = \boldsymbol{\Theta}_g \quad (7)$$

In Equation (7), y_{ig} is the vector of observed sleep items for individual i in group g; νg contains item intercepts; Λg is the loading matrix; ξ_{ig} is the latent sleep-quality construct; ε_{ig} is measurement error and Θg its covariance matrix. Invariant or partially invariant measurement is needed before interpreting country differences in latent sleep scores.

3.4. Cardiometabolic Outcomes and Nonlinear Regression

The primary clinical outcome will be a pre-registered composite based on measured blood pressure, adiposity and glycaemia, with all components also reported individually. Clinical thresholds and standardized repeat-measurement procedures must be stated before the field study begins. A component-weighted continuous score can be tested as sensitivity endpoint under Equation (8).

$$R_i = \sum_{k=1}^{K}\alpha_k\,\frac{X_{ik}-\mu_k^{\text{ref}}}{\sigma_k^{\text{ref}}}, \quad \alpha_k \geq 0, \quad \sum_{k=1}^{K}\alpha_k = 1 \quad (8)$$

In Equation (8), X_{ik} is cardiometabolic component k; μ_{ref} and σ_{ref} are pre-specified reference mean and standard deviation; α_k is a clinically justified nonnegative weight and K the total number of components. Signs must be harmonized so larger values consistently signify greater risk. Deriving weights from the study outcome would create optimism and is not proposed.

The primary nonlinear regression will use restricted cubic spline bases to avoid imposing a linear relationship between UPF share and outcome. Equation (9) includes country-specific curve differences and recruitment-site variation.

$$\text{logitPr}(Y_i = 1 \mid \cdot) = \beta_0 + \sum_{r=1}^{R} \beta_r B_r(\eta_i) + \theta C_i + \sum_{r=1}^{R} \kappa_r C_i B_r(\eta_i) + \mathbf{x}_i^{\top}\boldsymbol{\phi} + u_{s(i)} \quad (9)$$

Here Y_i is the binary risk indicator; $B_r(\eta_i)$ is the rth pre-selected restricted-cubic-spline basis evaluated at latent UPF exposure; R is the number of spline components; C_i indicates country; βr are exposure coefficients; θ is a country intercept difference; κr quantify country-specific curve modification; φ adjusts covariates; $u_{s(i)}$ is a site random intercept. Knots will be chosen by pre-specified pooled exposure quantiles, not by retrospective searching for statistically significant results.

To communicate effect magnitude beyond odds ratios, the model will yield standardised marginal risks and a ten-percentage-point risk difference using Equation (10).

$$\Delta_c(u;10) = \frac{\sum_{i:C_i=c} w_i^{*}[\hat{p}_i(u+10) - \hat{p}_i(u)]}{\sum_{i:C_i=c} w_i^{*}} \quad (10)$$

In Equation (10), Δc is the country-specific standardized risk difference; u is a specified reference UPF percentage; $\hat{p}_i(v)$ is the fitted risk at exposure v holding a person's other measured covariates fixed; and w^*_i are calibrated survey weights. Evaluation must be limited to exposure values with adequate observed overlap. Confidence intervals will incorporate both model fitting and measurement uncertainty.

3.5. Joint Dietary and Sleep Effects

A flexible interaction model allows sleep duration to have a nonlinear relationship with risk and avoids dichotomizing all sleep information at seven hours. The tensor-product interaction in Equation (11) captures departures from additive log-odds across the two continuous exposures.

$$\text{logit}(p_i) = \alpha_0 + \sum_{r=1}^{R} \alpha_r B_r(\eta_i) + \sum_{q=1}^{Q} \zeta_q D_q(S_i) + \sum_{r=1}^{R}\sum_{q=1}^{Q} \psi_{rq} B_r(\eta_i) D_q(S_i) + \mathbf{x}_i^{\top}\boldsymbol{\omega} \quad (11)$$

Here $Dq(S_i)$ is the qth spline basis for sleep duration; Q denotes the number of sleep bases; ζq describes the sleep curve; ψrq is the cross-basis coefficient; ω denotes covariate effects and p_i the modelled risk. A parsimonious penalised formulation will be preferred if sample size is insufficient for the full interaction surface. Separate models will evaluate PSQI without combining correlated sleep constructs indiscriminately.

The additive interaction contrast will compare the joint high-UPF/short-sleep profile with the sum of the separate contrasts, as formalised in Equation (12).

$$\text{RERI}_{RD} = p_{11} - p_{10} - p_{01} + p_{00} \quad (12)$$

The four p_ab values in Equation (12) are adjusted marginal risks for two pre-specified binary exposure contrasts (a = higher versus lower UPF; b = shorter versus reference sleep). The risk-difference interaction is a descriptive effect-modification measure. Cutpoints, standardisation population and uncertainty intervals will be pre-registered.

3.6. Missing Data, Bias, and Inference

Missingness by site, income, food exposure and outcome will be described before imputation. Multiple imputation will use models including the country interactions and nonlinear exposure terms; additional sensitivity analysis will evaluate departures from missing at random. For coefficient vector estimates, Rubin-style pooling in Equation (13) combines within- and between-imputation uncertainty.

$$\bar{\boldsymbol{\beta}} = \frac{1}{M}\sum_{m=1}^{M} \hat{\boldsymbol{\beta}}^{(m)}, \quad \mathbf{T} = \frac{1}{M}\sum_{m=1}^{M} \mathbf{U}^{(m)} + \left(1 + \frac{1}{M}\right)\frac{1}{M-1}\sum_{m=1}^{M}\left(\hat{\boldsymbol{\beta}}^{(m)} - \bar{\boldsymbol{\beta}}\right)\left(\hat{\boldsymbol{\beta}}^{(m)} - \bar{\boldsymbol{\beta}}\right)^{\top} \quad (13)$$

Here M is the imputation count; β(m) is the fitted coefficient vector in imputed dataset m; U(m) its within-imputation covariance; β̄ the pooled coefficient vector and T its total covariance. Rubin pooling of complex nonlinear marginal estimands will be performed at the estimand level rather than relying only on transformed pooled coefficients.

The agreement of duplicate NOVA coding will be assessed by chance-corrected agreement and probabilistic sensitivity analysis. Equation (14) expresses the conventional agreement estimator.

$$\kappa = \frac{p_o - p_e}{1 - p_e}, \qquad p_e = \sum_{a=1}^{A} p_{a+}\, p_{+a} \quad (14)$$

In Equation (14), po is observed agreement; pe is agreement expected from coder-specific marginal classification proportions; A is the number of NOVA categories; pa+ and p+a are the two coders' marginal proportions for category a. Kappa will be accompanied by class-specific disagreement rates because prevalence affects its interpretation.

To assess unmeasured confounding, Equation (15) defines a bias-factor adjustment for a hypothesised binary confounder. This is an analytical sensitivity device, not evidence that such a confounder exists.

$$\text{BF} = \frac{RR_{UY}\, RR_{EU}}{RR_{UY} + RR_{EU} - 1}, \qquad RR_{\text{adjusted}} \approx \frac{RR_{\text{observed}}}{\text{BF}} \quad (15)$$

Here RRUY is the maximum confounder–outcome risk ratio within exposure categories; RREU is the exposure–confounder risk ratio; BF is the conventional bounding bias factor; and RR denotes risk ratio. The approximation is intended for sensitivity discussion under its assumptions, not an automatic correction of the logistic estimates.

Model calibration and discrimination will be evaluated separately from coefficient inference. Equation (16) specifies the weighted Brier score for individual probability predictions, while cross-validation will be blocked by site where sample size permits.

$$\text{BS}_w = \frac{\sum_{i=1}^{n} w_i^{*}(Y_i - \hat{p}_i)^2}{\sum_{i=1}^{n} w_i^{*}} \quad (16)$$

In Equation (16), n is sample size, Y_i the observed binary endpoint, $\hat{p}_i$ the predicted probability and w_i^* the calibrated observation weight. Calibration-in-the-large, calibration slope and bootstrap optimism will also be reported if prediction is a stated secondary goal.

3.7. Synthetic Demonstration and Reproducibility

The supplied source manuscript contains a simulated dataset with a fixed seed (20261107), 1,200 records per country-labelled scenario and six figures. No recruited participant data or external validation set were supplied with the current document. The six original displays are preserved and explicitly labelled synthetic; however, the newly specified hierarchical, latent-variable and tensor-spline procedures have not been fitted to those underlying records, which were not supplied separately. No new effect estimates, confidence intervals or validation metrics are therefore asserted. All future empirical analyses should be pre-registered and follow STROBE reporting principles.

Table 1

Analysis component	Primary specification	Planned check / limitation
Dietary exposure	Repeated NOVA-coded 24-hour recalls; latent usual intake	Adjudication, day-to-day variability, probabilistic coding
Sleep	Duration, PSQI and shift schedule	Factor invariance; optional actigraphy subset
Primary endpoint	Predefined measured cardiometabolic composite	Component-wise analyses and calibration
Primary association	Weighted hierarchical logistic spline model	Positivity, overlap, site-level heterogeneity
Interaction	UPF × country; spline UPF × sleep	Joint tests and marginal risk contrasts
Uncertainty	Bootstrap and multiple imputation	Missing-not-at-random and unmeasured confounding

4. Results: Illustrations from Simulated Scenarios

All findings in this section are from the existing synthetic demonstration, not national surveillance data or recruited UK or Ghana participants. Numerical values from the supplied manuscript are reproduced without presenting them as estimated population differences. The expanded methods in Section 3 are prospective analytical specifications, not retrospective claims about computations performed on the synthetic data.

4.1. Scenario Characteristics and UPF Distributions

Table 2 reports the synthetic generator's scenario summaries as presented in the original manuscript. These quantities are neither observed estimates nor defensible measures of cross-country inequality. Figure 1 displays the imposed exposure-distribution shift; its dashed lines correspond to scenario means. Once participant data are collected, the measurement-error model in Equation (4) will test whether this apparent shift persists after adjustment for within-person diet variability.

Table 2

Synthetic scenario	n	Mean age, years	UPF energy share, %	Sleep, hours	Mean PSQI	Mean BMI	Composite-risk frequency
United Kingdom	1,200	26.3	52.8 ± 15.3	7.06 ± 0.85	6.01	25.00	27.4%
Ghana	1,200	26.5	39.1 ± 15.2	7.25 ± 0.81	5.15	23.58	13.2%

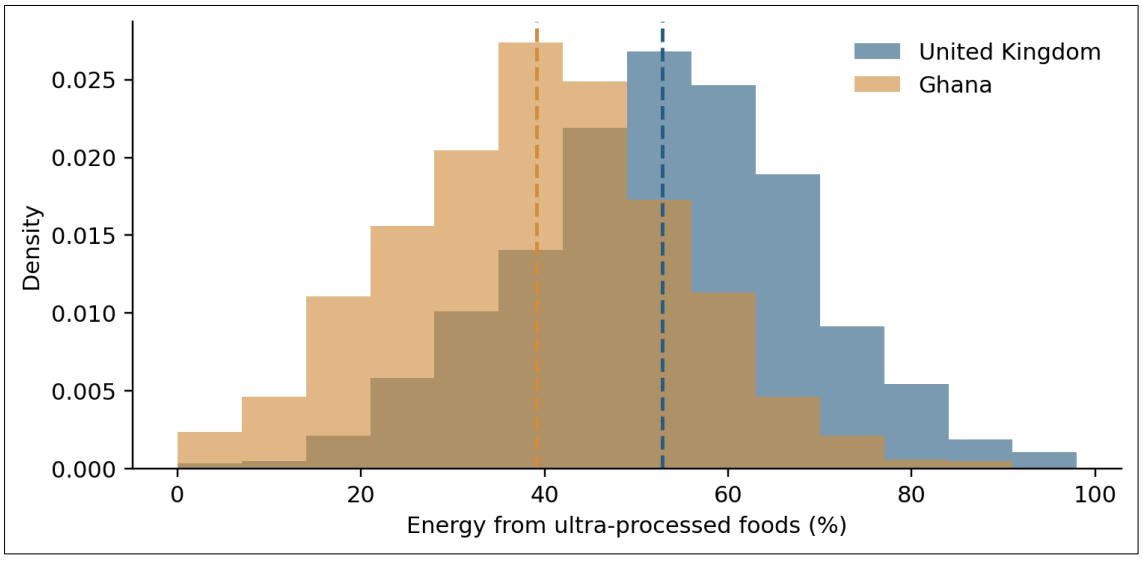


Fig 1: Simulated distributions of UPF energy share in the two country-labelled scenarios (not empirical prevalence estimates).

4.2. UPF and Sleep Patterns

Figure 2 illustrates decreasing simulated sleep duration across exposure bands, while Figure 3 illustrates higher simulated PSQI scores at greater UPF quartiles. The shapes arise from generator assumptions and random variation. The nonlinear joint model in Equation (11), rather than a visually inferred trend, will provide the pre-specified test of whether empirical dietary gradients differ across sleep-duration profiles.

4.3. Cardiometabolic Risk Patterns

Figure 4 shows a programmed increase in the fraction classified as at risk across within-country UPF quartiles. Figure 5 standardizes synthetic between-scenario differences across variables measured in different units. These graphics

are useful for checking output formats, but neither supplies a regression-adjusted country contrast. Equations (9) and (10) instead define the future inferential models and interpretable marginal risk contrast.

4.4. Stratification and Robustness

Figure 6 stratifies the simulated UPF–risk relationship by a seven-hour sleep threshold. A crossing or separation of plotted lines does not establish interaction. A future empirical interaction test will use the continuous sleep basis of Equation (11), pre-specified marginal contrasts under Equation (12), and robust confidence intervals. Sensitivity work will also compare the probabilistic NOVA coding in Equation (5), missing-data uncertainty in Equation (13), and sensitivity scenarios under Equation (15).

5. Discussion

5.1. Interpretation Against Existing Evidence

The proposed framework distinguishes three questions often collapsed in observational nutrition studies: whether higher UPF intake is associated with measured cardiometabolic risk, whether this association changes over the exposure range, and whether sleep modifies its magnitude. Published umbrella reviews support investigation of the first question but do not settle the latter two in harmonised UK–Ghana young-adult populations (Lane *et al*., 2024; Barbaresko *et al*., 2025) [9, 34]. The controlled feeding study by Hall *et al*. (2019) [27] strengthens plausibility for an energy-intake pathway while leaving the relative roles of processing, nutrient composition and eating behaviour unresolved.

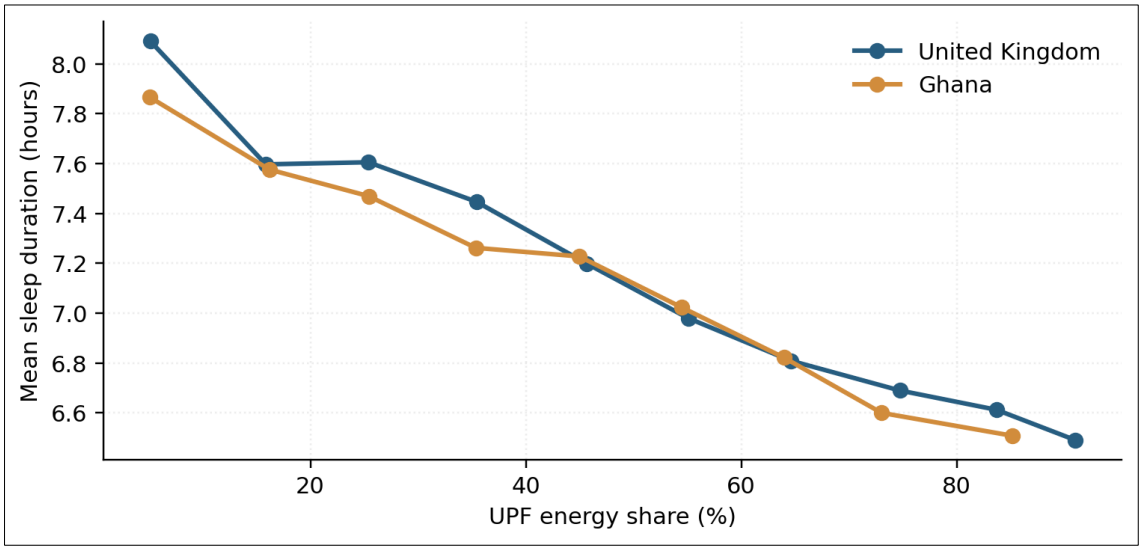


Fig 2: Scenario-based mean sleep duration across UPF exposure bands; slopes were programmed in the synthetic data.

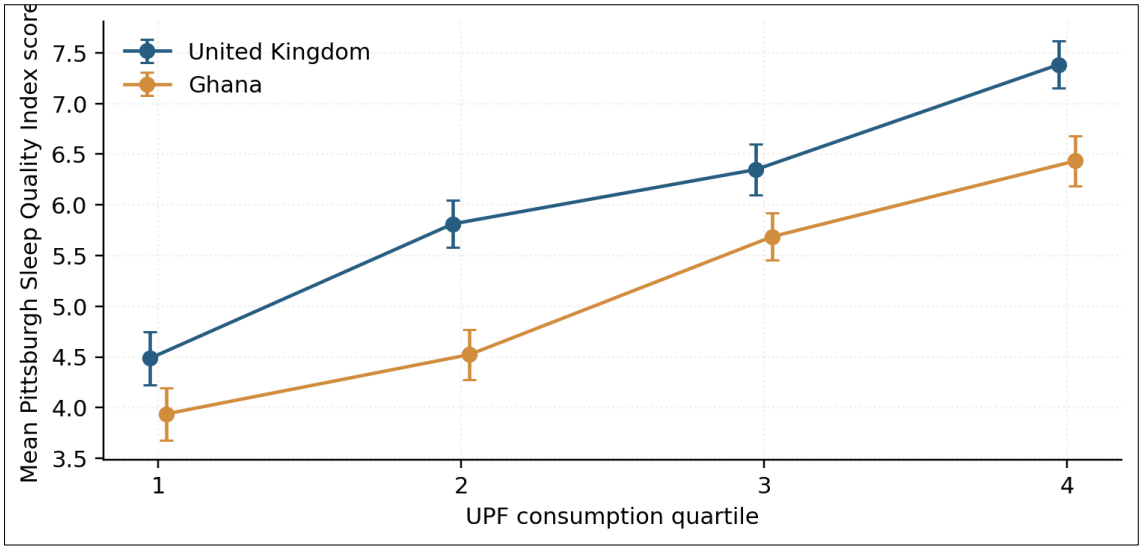


Fig 3: Scenario-based PSQI averages across UPF quartiles with uncertainty intervals computed from synthetic observations.

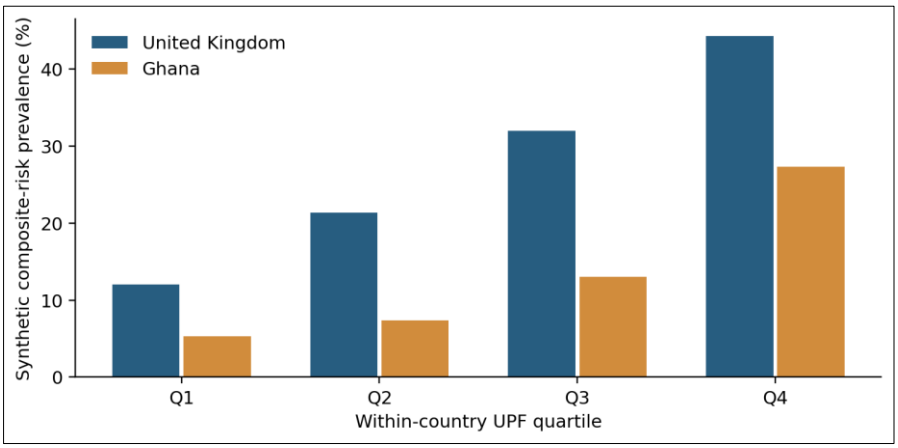


Fig 4: Synthetic cardiometabolic composite-risk frequency by UPF quartile; no measured diagnoses are represented.

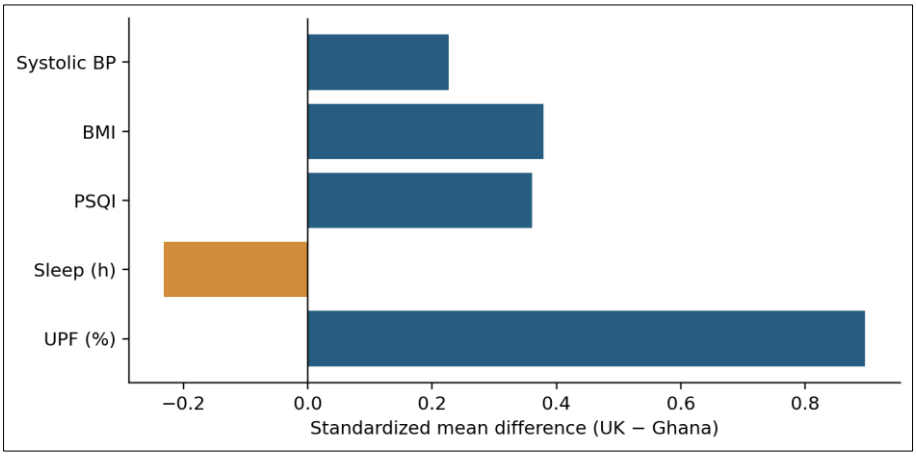


Fig 5: Standardized synthetic between-scenario differences for selected dietary, sleep and cardiometabolic variables.

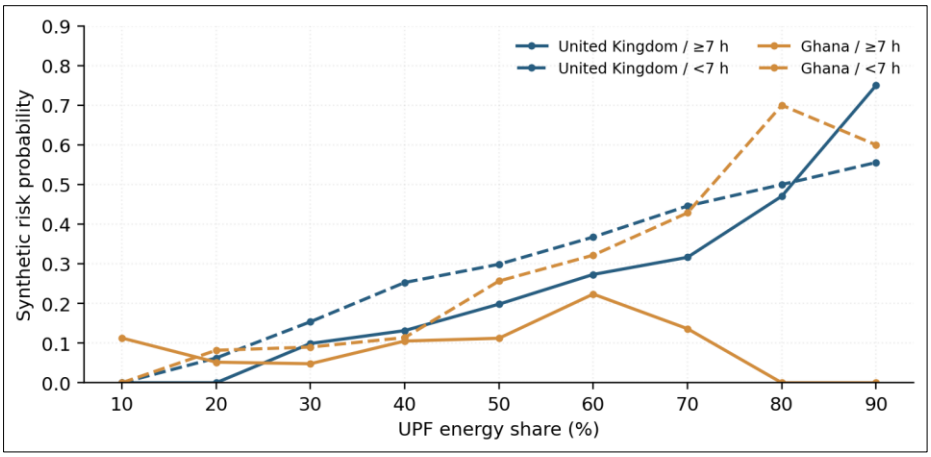


Fig 6: Synthetic UPF–risk relationships by country-labelled scenario and sleep category; model-generated illustration only.

The source manuscript's synthetic results are best interpreted as a test of reporting conventions. They do not support claims about national UPF exposure, clinical disease prevalence or comparative effect size. Equations (4), (9) and (11) clarify how genuine future observations would be analysed, with participant-level uncertainty, nonlinear gradients and country-specific effects. Model complexity is valuable only insofar as measured sample size, exposure overlap and instrument quality allow stable estimation.

5.2. UK–Ghana Comparability and Policy Meaning

The comparison must account for national food environments without reducing Ghana to an assumed early-transition setting or the UK to a homogeneous industrial-food environment. Explicit coding of local mixed dishes, verified branded ingredients, affordability and employment schedules will be essential. Measurement invariance testing in Equation (7) is especially important: an observed PSQI score difference would be difficult to interpret if respondents in the two settings understand questions differently. Policy interpretations should remain conditional on empirical effect estimates, existing nutrition-security constraints and prospective triangulation.

5.3. Position of the Requested Author-Group Literature

The author-group literature should contribute where its methods or findings genuinely intersect with this nutrition question. Idoko *et al*. (2024) [28] is relevant to food processing and diabetes risk, while Otugene *et al*. (2024) [47] offers a methodological example of modelling transparency in a different epidemiological problem. These papers cannot substitute for direct longitudinal nutrition and sleep studies. A large number of publications by the named authors concern renewable power, digital systems, cybersecurity or infectious-disease models, and citing approximately 30 of those as though they substantiate UPF epidemiology would misrepresent their scope. The reference list therefore prioritizes topical relevance, complete bibliographic information and explicit limitations.

5.4. Limitations and Next Steps

The main limitation remains the absence of participant observations, which prevents empirical estimation, causal inference and independent external validation. The models also demand more information than the supplied synthetic demonstration can establish: item-level food composition, repeat recalls, sampling probabilities, site identifiers, sleep item responses, clinical replicates and missingness indicators. A multi-site pilot should validate the instruments and data pipeline, and sample-size simulations should assess country-by-spline and sleep-interaction power before the full survey. Research outputs should clearly distinguish observed descriptive statistics from model-based marginal estimates and sensitivity scenarios.

6. Conclusions and Recommendations

This revised article presents a comparative research protocol and analytic architecture rather than an empirical demonstration of UK–Ghana differences. Its main methodological advance is the explicit accommodation of uncertain usual dietary exposure, nonlinear response functions, measurement equivalence, multilevel recruitment, flexible sleep interactions and sensitivity to missingness and unmeasured confounding. Subsequent recruitment, ethics approval, transparent pre-registration and independent clinical validation are required before substantive epidemiological or policy conclusions can be drawn.

Declarations

Ethics: This manuscript revision analyses no recruited human participants. The proposed field study requires country-specific ethics review and informed consent. Data availability: Six synthetic figures and summarized synthetic results were supplied in the source manuscript; the underlying simulated record-level dataset and executable analysis code were not provided with this revision. Funding, conflicts and author approval: To be completed and confirmed by Rosetta Quaicoe prior to submission. Reporting: The proposed observational field study should follow STROBE.

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