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Supplementary information

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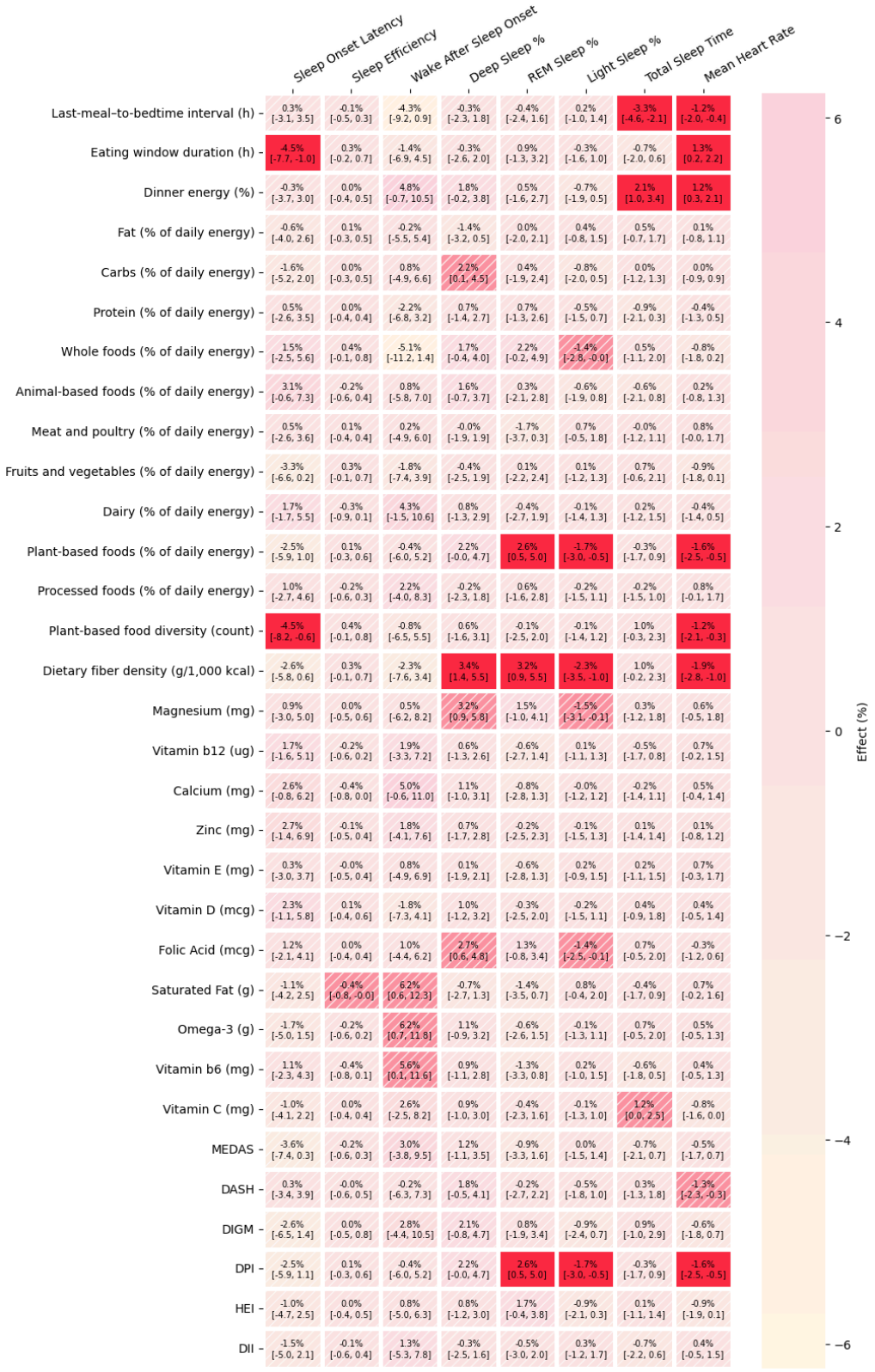
Impact of daily diet on sleep quality

In the format provided by the authors and unedited

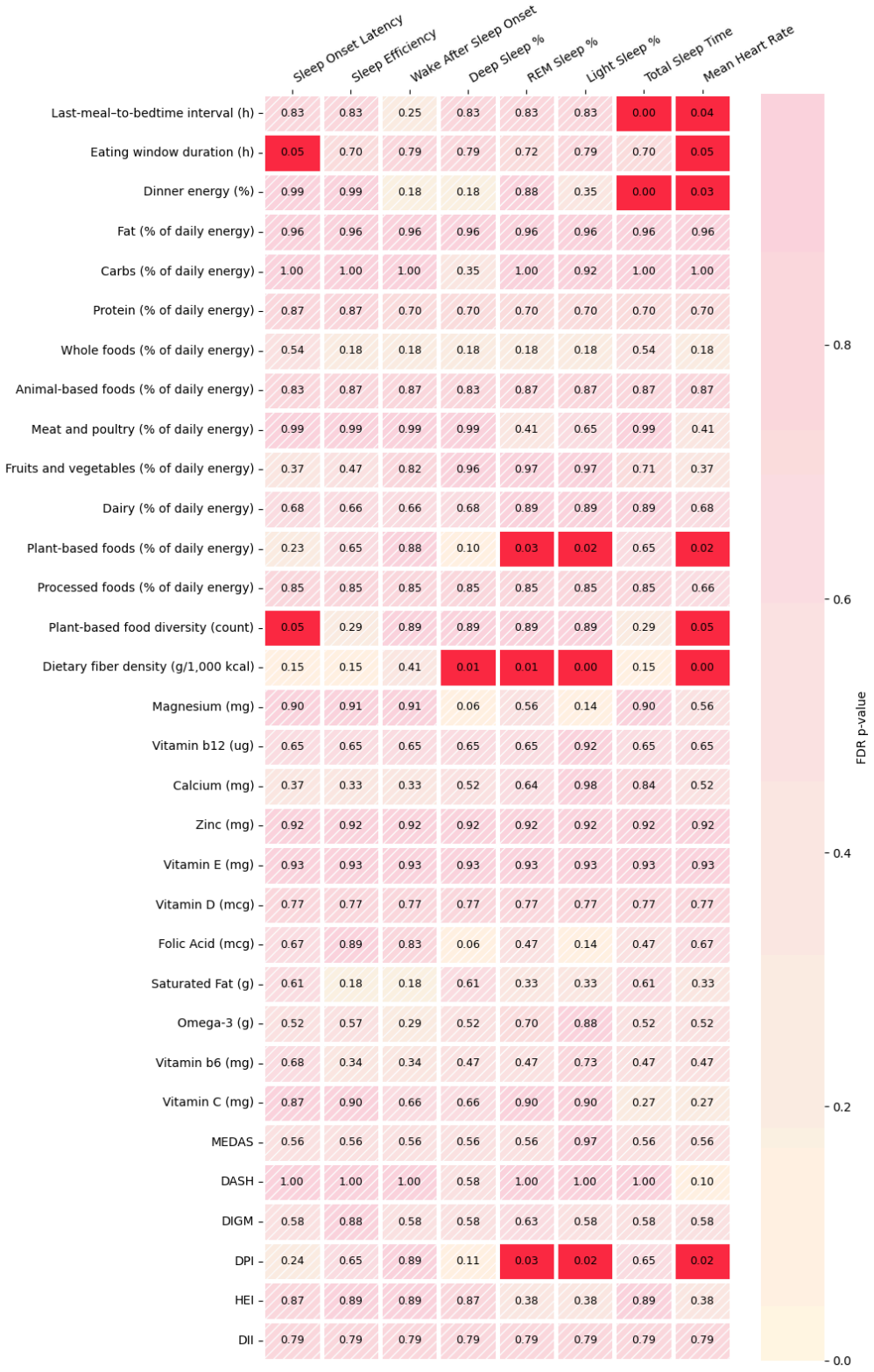
Null Findings and Their Interpretation

To complement the significant findings presented in the main text, we provide additional analyses of nutritional exposures that did not yield statistically significant associations with same night objective sleep physiology after bootstrap-based uncertainty estimation and inverse-probability weighting. These results help illustrate the specificity of our causal effects framework and highlight dietary domains where day-to-day nutritional variation did not produce measurable short-term changes in sleep architecture.

Of the 32 prespecified dietary exposures, 25 did not survive Benjamini–Hochberg correction at FDR < 0.05. We wish to be explicit about what these null results do and do not imply. Exposures for which we found no significant association, produced effect estimates near zero with confidence intervals that largely excluded clinically meaningful effect sizes. Under our study's statistical power (N=4,793 person-nights), the absence of significance provides reasonable evidence against large dietary effects on the tested sleep outcomes. We do not interpret null results as evidence that no relationship exists at all thresholds of intake or in all populations. Micronutrients such as magnesium have shown effects on sleep primarily in deficient populations. Across outcomes, several exposures exhibited 95% confidence intervals that did not cross the null, indicating statistically robust associations at the confidence-interval level. Importantly, the direction of effects varied systematically by food source. Higher intake of plant-based and whole-food patterns tended to be associated with favorable sleep characteristics, including higher proportions of deep and REM sleep and reduced wake after sleep onset. In contrast, higher intake of processed foods and saturated fat showed associations in the opposite direction, including longer wake after sleep onset and less favorable sleep-stage distributions.



Supplementary Figure S1. Heatmap indicating the relative effect (ATE) and its 95% confidence interval of each feature for all 32 dietary exposures and 8 sleep outcomes. Pink cells show statistically significant effects (q < 0.05); pink hatched cells show effects with CIs that do not cross zero; hatched heatmap shows non-significant effects. Colour intensity reflects effect magnitude.



Supplementary Figure S2. Heatmap of FDR-corrected p-values for all 32 dietary exposures and 8 sleep outcomes. Red cells show statistically significant effects ($q < 0.05$) and hatched show non-significant. Colour intensity reflects p-value magnitude.

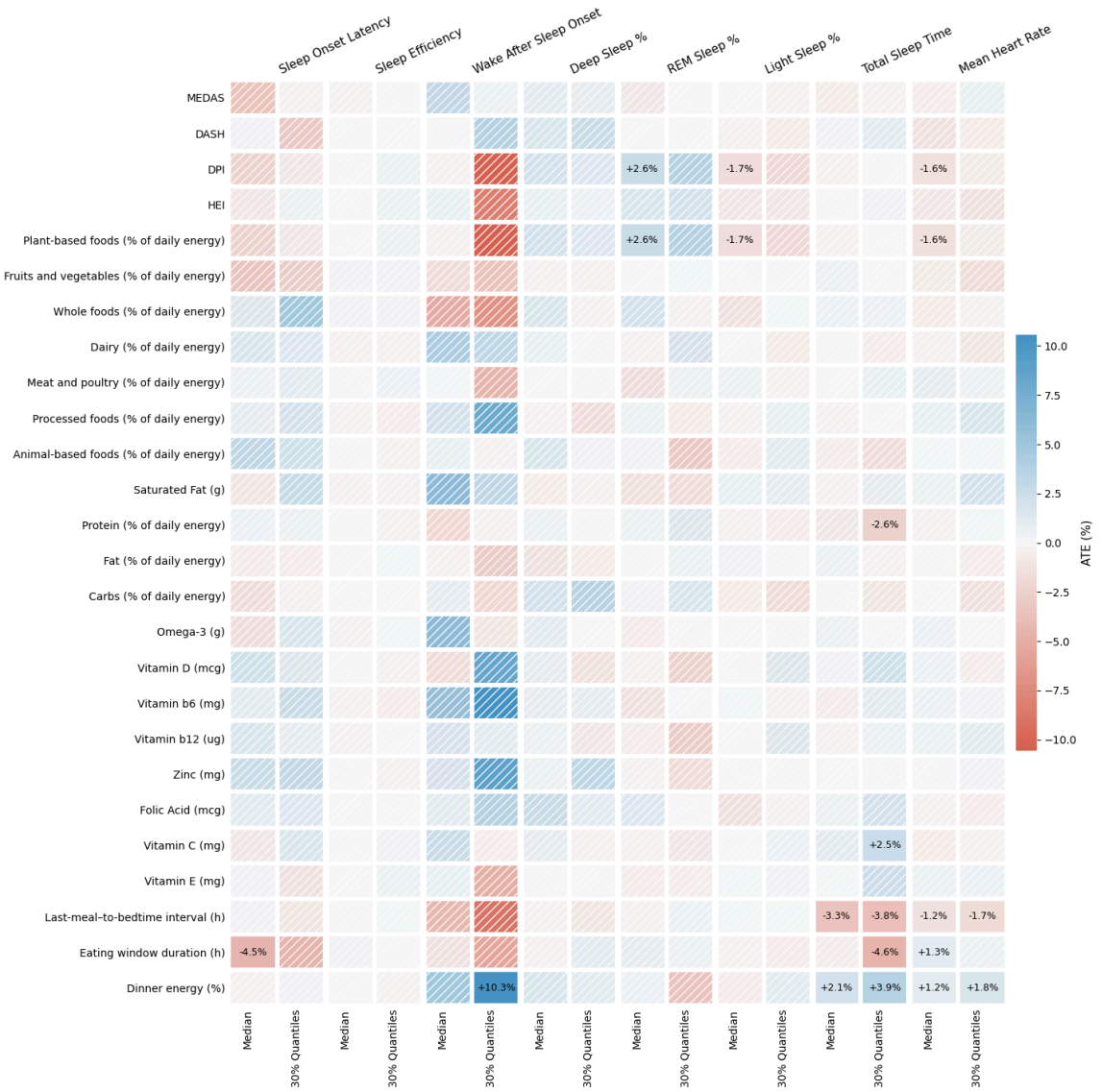
Extreme intake contrasts

Prior work examining lifestyle-sleep relationships has shown that increasing the magnitude of behavioral contrast can uncover stronger physiological effects that are attenuated under population-average comparisons. For example, a recent study demonstrated dose-dependent disruptions in sleep and nocturnal autonomic function by contrasting progressively higher levels of evening exercise strain rather than relying on binary exposure definitions [78]. Motivated by this framework, we performed complementary contrast-amplification analyses for all dietary and behavioral exposures.

Specifically, for each exposure, we contrasted individuals in the top and bottom 30% of the exposure distribution. We estimated average treatment effects (ATE) using inverse probability weighting (IPW), with weights derived from a logistic propensity model conditioned on baseline dietary intake, demographic variables, and anthropometric measures. False discovery rate correction was applied across outcomes.

Supplementary Results Figure 3 summarizes ATEs (%) across all exposures and sleep outcomes under both median-contrast and 30%-quantile-contrast specifications. Effect directions were highly consistent across specifications, supporting the robustness of the primary results. Under extreme contrasts, several effects were amplified. The largest estimated effect was observed for dinner energy proportion on wake after sleep onset (ATE: +10.3%), indicating that consuming a substantially greater share of daily energy at dinner markedly increases nighttime awakenings. Eating window duration showed significant effects on sleep onset latency (−4.5%) and total sleep time (−4.6%), and last-meal-to-bedtime interval was associated with reductions in total sleep time (−3.3%) and mean nocturnal heart rate (−3.8% to −1.7%). Dietary quality—indexed by DPI and plant-based food proportion—was associated with increased deep sleep (+2.6%) and reductions in light sleep (−1.7%) and total sleep time (−1.6%) under both contrast specifications. Protein intake and vitamin C showed isolated significant associations with total sleep time (−2.6% and +2.5%, respectively).

Compared with the primary analysis, extreme-quantile contrasts yielded larger point estimates for meal timing exposures in particular, suggesting dose-dependent relationships consistent with prior findings on evening behavioral exposures and sleep disruption. We present these results as supportive analyses corroborating the primary findings; the full set of ATEs across all exposures and sleep outcomes is provided in Supplementary Results Figure 3.



Supplementary Figure S3. Extreme-quantile IPW analysis reveals amplified diet-sleep associations across a broad panel of exposures.

This figure presents average treatment effects (ATEs, %) estimated via inverse probability weighting, contrasting individuals in the top and bottom 30% of each dietary and behavioral exposure distribution. Rows correspond to dietary and meal-timing exposures spanning dietary quality indices, macronutrient composition, micronutrients, and meal timing variables; columns correspond to eight sleep outcomes (sleep onset latency, sleep efficiency, wake after sleep onset, deep sleep %, REM sleep %, light sleep %, total sleep time, and mean nocturnal heart rate), each shown under both median-contrast and 30%-quantile-contrast specifications. Cell color encodes the direction and magnitude of the ATE (blue: increasing; red: decreasing), with values capped at ±10% for display. Hatched cells indicate non-significant effects after FDR correction; labeled values denote statistically significant ATEs.