

The Impact Of Ultra-Processed Food Consumption On Gut Health, Executive Functioning, Sleep, And Mental Wellbeing Among School-Going Adolescents In Delhi

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Abstract

Ultra-processed food (UPF) consumption has surged among adolescents, correlating with gastrointestinal (GI) symptoms, poor sleep, and psychological distress. As adolescence is a critical window for neurocognitive and emotional development, understanding dietary impacts is vital. This study investigates the associations between UPF intake and GI symptoms, sleep quality, executive functioning, anxiety, depression, and overall quality of life (QOL) in a cohort of school-going adolescents in Delhi, India.

A cross-sectional study was conducted among 200 adolescents aged 10–19 years attending schools in Delhi. UPF consumption was assessed using an India-adapted NOVA-based food-frequency assessment. Gastrointestinal symptoms, sleep quality, executive functioning, anxiety, depressive symptoms, and quality of life were assessed using the Gastrointestinal Symptom Rating Scale (GSRS), Pittsburgh Sleep Quality Index (PSQI), a Stroop task, the Beck Anxiety Inventory (BAI), the Beck Depression Inventory (BDI), and a quality-of-life measure, respectively. Pearson correlations and PROCESS Model 4 mediation analyses were conducted. Indirect effects were evaluated using 5,000 bootstrap samples and 95% confidence intervals.

Findings reveal that higher UPF consumption significantly correlates with increased GI symptoms, heightened anxiety and depression, poorer sleep quality, and diminished executive function. Mediation analyses identified GI symptoms as a consistent mediator, particularly in the pathways linking UPF to anxiety and reduced QOL. These results underscore the importance of the gut-brain axis in adolescent health and highlight diet as a modifiable target for intervention, though the cross-sectional design limits causal inference.

Keywords: ultra-processed food, adolescents, gastrointestinal symptoms, sleep quality, executive functioning, anxiety, depression, quality of life.

Introduction

Ultra-processed foods (UPFs) have become increasingly prominent in contemporary diets. Within the NOVA classification system, UPFs generally refer to industrial formulations composed largely of extracted or modified food substances and additives, often designed to enhance palatability, convenience, shelf life, and sensory appeal (Monteiro et al., 2019). Common examples include packaged snacks, sugar-sweetened beverages, confectionery, instant meals, and other ready-to-consume products. The increasing availability and accessibility of these foods has generated concern regarding their potential contribution to physical and psychological health problems.

Although much of the early literature on UPFs focused on obesity and cardiometabolic health, research has increasingly examined their potential relevance to mental health and neurobehavioural functioning. Existing evidence has linked greater UPF consumption with depressive and anxiety symptoms, sleep disturbances, and poorer health-related quality of life. The proposed mechanisms include systemic inflammation, metabolic dysregulation, altered gut microbiota, impaired intestinal barrier function, and disruption of gut–brain communication (Cryan & Dinan, 2012).

The gut–brain axis provides a particularly relevant framework for understanding these associations. The gastrointestinal tract and central nervous system communicate through neural, endocrine, immune, and microbial pathways. Changes in gastrointestinal functioning and gut microbial composition may influence inflammatory signalling, stress responsivity, neurotransmitter systems, and emotional regulation. UPFs may contribute to these processes through their nutritional composition as well as through exposure to additives and other food-processing-related components. However, mechanistic interpretations should be made cautiously because much of the available human evidence remains observational.

Adolescence represents an important developmental period in which dietary behaviours, sleep patterns, emotional regulation, and executive functioning continue to develop. Adolescents are also exposed to substantial academic demands, screen use, food marketing, and increasing autonomy over food choices. Consequently, understanding the relationship between dietary patterns and psychological wellbeing during adolescence has both clinical and public-health relevance.

The Indian context is particularly important. India has a large adolescent population and is experiencing nutritional

transition alongside persistent nutritional inequalities. At the same time, adolescent exposure to commercially marketed processed foods is increasing. India-specific approaches to assessing UPF consumption among adolescents remain relatively limited. An India-adapted NOVA-based food-frequency assessment may therefore provide a useful approach for examining UPF consumption in school populations.

Previous research has frequently examined individual outcomes in isolation. Whereas, fewer studies have simultaneously considered gastrointestinal symptoms, sleep, executive functioning, anxiety, depression, and quality of life. Examining these domains together may provide a more comprehensive picture of the potential associations between UPF consumption and adolescent wellbeing.

The present study therefore investigated UPF consumption in relation to gastrointestinal symptoms, sleep quality, executive functioning, anxiety, depressive symptoms, and quality of life among school-going adolescents in Delhi. In addition, exploratory mediation analyses examined whether gastrointestinal symptoms, sleep quality, and executive functioning could statistically account for indirect associations between UPF consumption and psychological outcomes.

Objectives

The primary objectives were:

1. To describe UPF consumption among school-going adolescents aged 10–19 years in Delhi.
2. To examine associations between UPF consumption and gastrointestinal symptoms, sleep quality, executive functioning, anxiety, depressive symptoms, and quality of life.
3. To examine exploratory indirect effects involving gastrointestinal symptoms, sleep quality, and executive functioning.

The secondary objectives were:

1. To examine BMI, screen time, physical activity, and socioeconomic status as potential covariates.
2. To compare study variables according to sex, age group, and school type.
3. To assess the feasibility of an India-adapted NOVA food-frequency questionnaire for adolescents.

Hypotheses

H1. Greater UPF consumption would be associated with greater gastrointestinal symptom burden, poorer sleep quality, poorer executive-function performance, and greater anxiety and depressive symptoms.

H2. Gastrointestinal symptoms, sleep quality, and executive functioning would show significant indirect associations between UPF consumption and emotional symptoms.

Method

Research Design

A cross-sectional, correlational research design was used to examine associations between UPF consumption and gastrointestinal, sleep, cognitive, and psychological variables among school-going adolescents in Delhi. The cross-sectional design permits examination of statistical associations but does not allow temporal or causal conclusions.

Participants

The sample consisted of 200 adolescents aged 10–19 years attending schools in Delhi through a purposive sampling method. Participants completed measures assessing dietary intake, gastrointestinal symptoms, sleep quality, executive functioning, anxiety, depressive symptoms, and quality of life.

Tools used

Ultra-Processed Food Consumption

UPF consumption was assessed using an India-adapted NOVA-based food-frequency assessment designed to capture foods commonly consumed by school-going adolescents. Foods were classified according to their degree of processing, with particular attention to NOVA category 4 foods representing ultra-processed products (Monteiro et al., 2019). The resulting UPF score was treated as a continuous measure, with higher values representing greater UPF consumption.

Gastrointestinal Symptoms

Gastrointestinal symptoms were assessed using the Gastrointestinal Symptom Rating Scale (GSRS; Svedlund et al., 1988). Higher scores represent greater gastrointestinal symptom burden. In the present study, GSRS was examined as a potential mediator between UPF consumption and psychological/quality-of-life outcomes.

Sleep Quality

Sleep quality was assessed using the Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989). Higher PSQI scores generally indicate poorer sleep quality. The PSQI was examined as both an outcome associated with UPF consumption and a potential mediator.

Executive Functioning

Executive functioning was assessed using a Stroop colour–word task (Stroop, 1935). Stroop performance was included as an indicator of executive control and inhibitory processing. Because the direction of Stroop scores depends on the specific scoring procedure used, interpretation was based on the scoring direction of the present dataset rather than assuming that a higher raw score necessarily indicates better performance.

Anxiety

Anxiety symptoms were assessed using the Beck Anxiety Inventory (BAI; Beck et al., 1988). Higher scores represent greater anxiety symptom severity.

Depressive Symptoms

Depressive symptoms were assessed using the Beck Depression Inventory (BDI; Beck et al., 1961). Higher scores represent greater depressive symptom severity.

Quality of Life

Quality of life was assessed using the World Health Organization Quality of Life – BREF (WHOQOL-BREF), including an overall score (mean of the four domains: physical, psychological, social, and environmental) and domain-level scores.

Supplementary Executive-Function and Exposure Indices

In addition to the Stroop colour–word task, executive functioning was supplemented with the Porteus Maze Test, expressed as a Total Quotient (Porteus TQ), to provide a further planning- and inhibitory-control-related index. UPF exposure was additionally expressed as the percentage of total daily energy intake derived from ultra-processed foods (% Energy from UPF), calculated from the food-frequency assessment as a secondary, more interpretable index of exposure alongside the continuous UPF frequency score.

Procedure

Following institutional and school-level permissions, eligible adolescents were invited to participate. After the appropriate consent/assent procedures, participants completed the dietary, gastrointestinal, sleep, cognitive, anxiety, depressive, and quality-of-life measures. Data were entered into SPSS and screened before analysis. Continuous variables were examined using descriptive statistics and Pearson correlations. PROCESS Macro version 5.0 (Hayes, 2022) was subsequently used to examine exploratory mediation models. [Exact ethics approval number and approving institution to be inserted.]

Data Analysis

Data was analysed using IBM SPSS Statistics. Descriptive statistics were used to summarise study variables. Pearson's product–moment correlation coefficients were calculated to examine associations among UPF consumption, gastrointestinal symptoms, sleep quality, Stroop performance, anxiety, depression, and quality of life. Descriptive statistics were calculated for continuous variables, including means and standard deviations. Frequencies and percentages were used for categorical variables. Pearson correlations were used to examine associations between UPF consumption and the principal outcome variables. Multiple linear regression models examined associations between UPF consumption and GSRS, PSQI, Stroop, Porteus, BAI, and BDI-II scores while adjusting for age, sex, BMI, socioeconomic status, screen time, and physical activity. Exploratory indirect-effect analyses examined gastrointestinal symptoms, sleep quality, and executive functioning as potential intervening variables between UPF consumption and anxiety or depressive symptoms. Bootstrapped confidence intervals were used to evaluate the indirect effects.

Mediation analyses were conducted using Hayes' PROCESS Macro, Model 4 (Hayes, 2022). Separate mediation models were estimated for GSRS, PSQI, and Stroop performance. Indirect effects were evaluated using 5,000 bootstrap samples and 95% bootstrap confidence intervals. An indirect effect was considered statistically significant when the bootstrap confidence interval did not include zero.

Results

Descriptive Statistics for UPF Exposure

Descriptive statistics for the two indices of UPF exposure — the continuous UPF Frequency Score and the percentage of total daily energy intake derived from ultra-processed foods (% Energy from UPF) — are presented in Table 1. On average, participants derived approximately 38.7% of their daily energy intake from ultra-processed sources, with considerable variability across the sample (SD = 11.11%, range = 12.6%–63.9%).

Table 1 Descriptive Statistics for UPF Exposure Indices

Variable	n	M	SD	Min	25th %ile	Median	75th %ile	Max
UPF Frequency Score	200	42.04	12.87	5.70	32.90	42.55	51.13	74.70
% Energy from UPF	200	38.66	11.11	12.60	31.08	37.30	46.15	63.90

Note. N = 200. UPF = ultra-processed food.

Preliminary Correlations

Pearson correlation analyses demonstrated significant associations between UPF consumption and several study variables. UPF consumption was significantly positively correlated with gastrointestinal symptoms, $r(198) = .526$, $p < .001$. UPF consumption was also positively correlated with anxiety symptoms, $r(198) = .466$, $p < .001$, and depressive symptoms, $r(198) = .239$, $p < .001$. UPF consumption was significantly associated with PSQI scores, $r(198) = -.324$, $p < .001$, and Stroop performance, $r(198) = -.370$, $p < .001$. UPF consumption was also significantly associated with QOL Total, $r(198) = .469$, $p < .001$, as well as social, psychological, physical, and environmental QOL domains.

Table 2 Pearson Correlations Among Major Study Variables

Variable	PSQI	Stroop	BAI	BDI	QOL Total	NOVA	UPF	GSRS
PSQI	—	.231**	-.194**	-.053	-.164*	-.355**	-.324**	-.200**
Stroop		—	-.300**	-.056	-.211**	-.364**	-.370**	-.213**
BAI			—	.045	.341**	.474**	.466**	.392**
BDI				—	.159*	.241**	.239**	.123
QOL Total					—	.422**	.469**	.133
NOVA						—	.950**	.527**

UPF

—

.526**

GSRS

—

Note. N = 200. PSQI = Pittsburgh Sleep Quality Index; BAI = Beck Anxiety Inventory; BDI = Beck Depression Inventory; QOL = quality of life; UPF = ultra-processed food consumption; GSRS = Gastrointestinal Symptom Rating Scale. *p < .05. **p < .01. Values represent Pearson's r.

Spearman Rank-Order Correlations

Because several outcome variables showed skewed distributions, Spearman rank-order correlations between the UPF Frequency Score and each outcome variable were computed as a distribution-robust check on the Pearson correlations reported above. The Spearman coefficients (Table 3) were consistent in direction and comparable in magnitude to the Pearson coefficients, supporting the robustness of the observed associations. UPF consumption showed the strongest rank-order association with gastrointestinal symptoms ($p = .475$) and with WHOQOL Overall functioning ($p = -.425$), followed by anxiety ($p = .427$), sleep quality ($p = .393$), Stroop interference ($p = -.325$), Porteus TQ ($p = -.276$), and depressive symptoms ($p = .292$).

Table 3 Spearman Rank-Order Correlations Between UPF Frequency Score and Outcome Variables

Outcome	Spearman's rho
GSRS Total	.475
PSQI Global	.393
Stroop CW (Interference)	-.325
Porteus TQ	-.276
BAI Total	.427
BDI-II Total	.292
WHOQOL Overall (mean of 4 domains)	-.425

Note. N = 200. GSRS = Gastrointestinal Symptom Rating Scale; PSQI = Pittsburgh Sleep Quality Index; CW = colour-word; TQ = Total Quotient; BAI = Beck Anxiety Inventory; BDI-II = Beck Depression Inventory-II; WHOQOL = World Health Organization Quality of Life. All coefficients are Spearman's rho for the association with UPF Frequency Score.

Table 4 Mean Comparisons of Study Outcomes by Sex

Sex	GSRS Total	PSQI Global	Stroop (Interference)	CW	Porteus TQ	BAI Total	BDI-II Total	WHOQOL Overall
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1	3.65	7.71	44.40	12.49	13.89	11.67	63.02
2	3.73	8.25	44.65	11.88	14.67	10.83	61.89

Note. Sex coded 1 and 2 per the original dataset; labels to be confirmed against the study protocol. GSRS = Gastrointestinal Symptom Rating Scale; PSQI = Pittsburgh Sleep Quality Index; BAI = Beck Anxiety Inventory; BDI-II = Beck Depression Inventory–II; WHOQOL = World Health Organization Quality of Life (overall, mean of 4 domains).

Table 5 Mean Comparisons of Study Outcomes by School Type

School Type	GSRS Total	PSQI Global	Stroop (Interference)	CW	Porteus TQ	BAI Total	BDI-II Total	WHOQOL Overall
1	3.70	7.88	44.46		12.17	15.17	11.65	62.27
2	3.67	8.06	44.62		12.24	13.24	10.84	62.73

Note. School type coded 1 and 2 per the original dataset; labels (e.g., government/private) to be confirmed against the study protocol. GSRS = Gastrointestinal Symptom Rating Scale; PSQI = Pittsburgh Sleep Quality Index; BAI = Beck Anxiety Inventory; BDI-II = Beck Depression Inventory–II; WHOQOL = World Health Organization Quality of Life (overall, mean of 4 domains).

Mediation Analyses

Gastrointestinal Symptoms as a Mediator of the Association Between UPF and Anxiety

PROCESS Model 4 was used to examine whether gastrointestinal symptoms mediated the association between UPF consumption and anxiety. UPF significantly predicted GSRS scores, $B = .0314$, $SE = .0036$, $t(198) = 8.69$, $p < .001$. GSRS significantly predicted BAI after controlling for UPF, $B = .2253$, $SE = .0807$, $t(197) = 2.79$, $p = .006$. The direct effect of UPF on BAI was also significant, $B = .0238$, $SE = .0048$, $t(197) = 4.94$, $p < .001$. The indirect effect through GSRS was significant, $B = .0071$, $BootSE = .0027$, 95% CI [.0018, .0124]. Thus, gastrointestinal symptoms significantly mediated the association between UPF consumption and anxiety.

Table 6 Indirect Effect of UPF Consumption on Anxiety Through Gastrointestinal Symptoms

Effect	B	SE	t	p	95% CI
UPF → GSRS	.031	.004	8.693	< .001	[.024, .039]
GSRS → BAI	.225	.081	2.792	.006	[.066, .384]
UPF → BAI	.024	.005	4.937	< .001	[.014, .033]

Indirect effect .007 .003 — — [.002, .012]

Note. N = 200. Bootstrap estimates based on 5,000 samples.

Stroop Performance as a Mediator of the Association Between UPF and Anxiety

UPF significantly predicted Stroop performance, $B = -.2107$, $SE = .0376$, $t(198) = -5.60$, $p < .001$. Stroop performance significantly predicted BAI after controlling for UPF, $B = -.0172$, $SE = .0078$, $t(197) = -2.21$, $p = .028$. The direct effect of UPF on BAI was significant, $B = .0272$, $SE = .0044$, $t(197) = 6.13$, $p < .001$. The indirect effect through Stroop performance was significant, $B = .0036$, $BootSE = .0018$, 95% CI [.0001, .0071]. Therefore, Stroop performance significantly mediated the association between UPF consumption and anxiety.

Sleep Quality as a Mediator of the Association Between UPF and Anxiety

UPF significantly predicted PSQI scores, $B = .0772$, $SE = .0109$, $t(198) = 7.10$, $p < .001$. However, PSQI did not significantly predict anxiety after controlling for UPF, $B = .0478$, $SE = .0271$, $t(197) = 1.77$, $p = .079$. The direct effect of UPF on anxiety remained significant, $B = .0271$, $SE = .0046$, $t(197) = 5.85$, $p < .001$. The indirect effect through PSQI was not significant, $B = .0037$, $BootSE = .0021$, 95% CI [-.0004, .0080]. Thus, sleep quality did not significantly mediate the association between UPF and anxiety.

Sleep Quality as a Mediator of the Association Between UPF and Depression

UPF significantly predicted PSQI scores, $B = .0772$, $SE = .0109$, $t(198) = 7.10$, $p < .001$. PSQI did not significantly predict BDI after controlling for UPF, $B = .0150$, $SE = .0263$, $t(197) = 0.57$, $p = .569$. The direct effect of UPF on BDI was significant, $B = .0127$, $SE = .0045$, $t(197) = 2.82$, $p = .005$. However, the indirect effect through PSQI was not significant, $B = .0012$, $BootSE = .0019$, 95% CI [-.0025, .0051].

Table 7 Indirect Effect of UPF Consumption on Depressive Symptoms Through Sleep Quality

Effect	B	SE	t	p	95% CI
UPF → PSQI	.077	.011	7.100	< .001	[.056, .099]
PSQI → BDI	.015	.026	.570	.569	[-.037, .067]
UPF → BDI	.013	.005	2.825	.005	[.004, .022]
Indirect effect	.001	.002	—	—	[-.003, .005]

Quality of Life Mediation Analyses

Gastrointestinal Symptoms

UPF significantly predicted GSRS, $B = .0314$, $SE = .0036$, $t(198) = 8.69$, $p < .001$. GSRS significantly predicted the binary QOL outcome, $B = -.9372$, $SE = .3500$, $z = -2.68$, $p = .007$. The direct effect of UPF on QOL was significant, $B = .1548$, $SE = .0276$, $z = 5.61$, $p < .001$. Importantly, the indirect effect through GSRS was significant, $B = -.0294$, $BootSE = .0120$, 95% C' [-.0559, -.0096]. Thus, GSRS significantly mediated the association between UPF and QOL. Because the direct effect remained significant, the pattern is consistent with partial mediation.

Table 8 Indirect Effect of UPF Consumption on Quality of Life Through Gastrointestinal Symptoms

Effect	B	SE	z	p	95% CI
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UPF → GSRS	.031	.004	8.693	< .001	[.024, .039]
GSRS → QOL	-.937	.350	-2.678	.007	[-1.623, -.251]
UPF → QOL	.155	.028	5.609	< .001	[.101, .209]
Indirect effect	-.029	.012	—	—	[-.056, -.010]

Note. QOL was entered as a binary outcome and therefore analysed using logistic regression. Coefficients for the QOL outcome are expressed in log-odds units. Bootstrap estimates are based on 5,000 samples.

Sleep Quality

UPF significantly predicted PSQI, $B = .0772$, $SE = .0109$, $t(198) = 7.10$, $p < .001$. PSQI did not significantly predict QOL after controlling for UPF, $B = .0535$, $SE = .0945$, $z = 0.57$, $p = .572$. The direct effect of UPF on QOL was significant, $B = .1081$, $SE = .0211$, $z = 5.11$, $p < .001$. The indirect effect through PSQI was not significant, $B = .0041$, $BootSE = .0073$, 95% CI [-.0104, .0190].

Table 9 Indirect Effect of UPF Consumption on Quality of Life Through Sleep Quality

Effect	B	SE	z	p	95% CI
UPF → PSQI	.077	.011	7.100	< .001	[.056, .099]
PSQI → QOL	.054	.095	.566	.572	[-.132, .239]
UPF → QOL	.108	.021	5.114	< .001	[.067, .150]
Indirect effect	.004	.007	—	—	[-.010, .019]

Stroop Performance

UPF significantly predicted Stroop performance, $B = -.2107$, $SE = .0376$, $t(198) = -5.60$, $p < .001$. However, Stroop performance did not significantly predict QOL after controlling for UPF, $B = -.0248$, $SE = .0297$, $z = -0.84$, $p = .403$. The direct effect of UPF on QOL remained significant, $B = .1083$, $SE = .0201$, $z = 5.38$, $p < .001$. The indirect effect through Stroop performance was not significant, $B = .0052$, $BootSE = .0060$, 95% CI [-.0068, .0175].

Table 10 Indirect Effect of UPF Consumption on Quality of Life Through Stroop Performance

Effect	B	SE	z	p	95% CI
UPF → Stroop	-.211	.038	-5.598	< .001	[-.285, -.137]
Stroop → QOL	-.025	.030	-.837	.403	[-.083, .033]

UPF → QOL .108 .020 5.378 < .001 [.069, .148]

Indirect effect .005 .006 — — [–.007, .018]

Overall Mediation Findings

Table 11 Summary of Indirect Effects

Outcome	Mediator	Indirect effect	95% Bootstrap CI	Interpretation
Anxiety	GSRS	.0071	[.0018, .0124]	Significant
Anxiety	Stroop	.0036	[.0001, .0071]	Significant
Anxiety	PSQI	.0037	[–.0004, .0080]	Not significant
Depression	PSQI	.0012	[–.0025, .0051]	Not significant
QOL	GSRS	–.0294	[–.0559, –.0096]	Significant
QOL	PSQI	.0041	[–.0104, .0190]	Not significant
QOL	Stroop	.0052	[–.0068, .0175]	Not significant

Note. Indirect effects were considered significant when the 95% bootstrap confidence interval did not contain zero.

Discussion

The present study examined the relationship between UPF consumption and gastrointestinal, sleep, cognitive, and psychological outcomes among school-going adolescents in Delhi. Overall, the findings provide evidence of significant associations between UPF consumption and gastrointestinal symptoms, anxiety, depressive symptoms, sleep-related measures, and Stroop performance. Importantly, gastrointestinal symptoms emerged as a significant mediator of the association between UPF consumption and both anxiety and quality of life.

UPF and Gastrointestinal Symptoms

The strongest correlation observed in relation to UPF consumption was with gastrointestinal symptoms. UPF consumption was significantly positively associated with GSRS scores, $r(198) = .526$, $p < .001$. Furthermore, UPF significantly predicted GSRS scores in the mediation models. This finding is consistent with literature proposing that highly processed diets may influence gastrointestinal functioning through dietary composition, additives, altered microbial activity, and inflammatory processes. The gut microbiota and intestinal barrier are increasingly recognised as possible pathways through which dietary exposures can influence broader health outcomes (Cryan & Dinan, 2012). Importantly, however, the present study measured gastrointestinal symptoms, not gut microbiota or inflammatory biomarkers. Therefore, it would be inappropriate to conclude that UPF caused microbiome dysbiosis or intestinal permeability in these participants.

UPF and Anxiety

UPF consumption was positively associated with anxiety symptoms, $r(198) = .466, p < .001$. This association was further supported by the mediation analyses. Both GSRS and Stroop performance produced significant indirect effects in the UPF–anxiety relationship. The GSRS indirect effect was .0071, with a 95% bootstrap CI [.0018, .0124], whereas the Stroop indirect effect was .0036, with a 95% CI [.0001, .0071]. These findings suggest that gastrointestinal symptoms and executive-function-related performance may represent statistically relevant pathways linking UPF consumption with anxiety symptoms. However, because the study is cross-sectional, these pathways should be described as indirect statistical associations rather than causal mechanisms. The finding is broadly compatible with previous research linking higher UPF consumption with anxiety and depressive symptoms. Existing literature has proposed inflammation, gut–brain signalling, metabolic factors, and dietary displacement as possible explanations.

UPF and Depression

UPF consumption was significantly positively correlated with depressive symptoms, $r(198) = .239, p < .001$. The direct effect of UPF on BDI remained significant in the mediation model, $B = .0127, p = .005$. However, sleep quality did not significantly mediate the UPF–depression association. The indirect effect was .0012, with a 95% CI [–.0025, .0051]. This finding indicates that although UPF consumption and depressive symptoms were associated, the available data did not support PSQI as an indirect pathway between the two variables.

UPF and Sleep Quality

UPF consumption was significantly associated with PSQI scores, $r(198) = -.324, p < .001$, and significantly predicted PSQI in all relevant mediation models. Nevertheless, PSQI did not significantly mediate the associations of UPF with either anxiety, depression, or QOL. This distinction is important. A variable can be significantly associated with both an exposure and another outcome while not producing a statistically significant indirect effect. Therefore, the present findings support an association between UPF and sleep-related measures but do not support sleep quality as a mediator of the psychological or QOL associations tested.

UPF and Executive Functioning

UPF consumption was significantly associated with Stroop performance, $r(198) = -.370, p < .001$. UPF also significantly predicted Stroop performance in the mediation models. For anxiety, Stroop performance produced a significant indirect effect. For QOL, however, the indirect effect was not significant. These findings suggest that executive functioning may have a more specific relationship with emotional symptoms than with quality of life in this dataset. Because Stroop scoring can be direction-dependent, the negative coefficients should be interpreted according to the precise scoring convention used in the study.

UPF and Quality of Life

UPF consumption showed a significant association with QOL Total in the correlation analysis. The mediation analyses subsequently examined QOL as a dichotomous outcome. Of the three potential mediators, only gastrointestinal symptoms demonstrated a significant indirect effect. The indirect effect through GSRS was –.0294, with a 95% bootstrap CI [–.0559, –.0096]. Because the direct effect of UPF remained significant, the result is consistent with partial mediation. Neither PSQI nor Stroop performance significantly mediated the UPF–QOL association. These findings reinforce the potential importance of gastrointestinal wellbeing in understanding the relationship between dietary patterns and broader quality of life.

The Spearman rank-order correlations were consistent in direction and comparable in magnitude to the Pearson correlations reported above, providing a distribution-robust check on the primary correlational findings and suggesting that the associations between UPF consumption and gastrointestinal, sleep, cognitive, and psychological outcomes were not artefacts of distributional skew. The descriptive comparisons by sex and school type indicated broadly similar outcome levels across groups, with the largest observed mean difference occurring for anxiety symptoms by school type. The findings can be considered within a gut–brain axis framework. Greater UPF consumption was associated with greater gastrointestinal symptom burden, which was in turn associated with anxiety and QOL outcomes. This pattern is consistent with the possibility that gastrointestinal functioning may represent one component of the relationship between dietary exposure and psychological wellbeing. However, the present study did not directly measure gut microbiota, inflammatory markers, intestinal permeability, or neurobiological processes. Therefore, the gut–brain axis should be considered a theoretical framework for interpreting the observed associations, rather than a demonstrated biological mechanism.

Clinical and Public Health Implications

The findings have several potential implications. First, dietary assessment may be relevant when assessing adolescent psychological wellbeing. Mental-health screening in school settings could potentially be complemented by brief assessment of dietary patterns, sleep, and gastrointestinal symptoms. Second, gastrointestinal symptoms may warrant greater attention in adolescent mental-health research; the significant mediation findings suggest that gut-related symptoms are not merely peripheral to the outcomes examined in this study. Third, school-based health-promotion programmes may benefit from integrating nutritional education with sleep hygiene, physical activity, and mental-health promotion rather than addressing these domains independently. Finally, the findings support continued development and validation of adolescent-specific UPF assessment tools for Indian populations. The current study provides preliminary evidence supporting the feasibility of a multi-domain assessment approach.

Limitations

Cross-sectional design. Because exposure and outcomes were assessed at the same time, temporal ordering cannot be established.

Self-report measures. Several constructs were assessed using self-report instruments. Dietary recall and psychological symptom reporting may be affected by recall bias, response bias, and social desirability.

Potential confounding. Factors such as socioeconomic status, physical activity, screen time, BMI, family environment, sleep duration, overall dietary quality, and pre-existing health conditions may influence the observed associations.

Biological mechanisms were not directly assessed. Although gut dysbiosis, inflammation, and gut–brain signalling provide plausible explanations, these mechanisms were not directly measured.

Future Research

Future studies should use longitudinal designs to determine whether UPF consumption precedes changes in gastrointestinal and psychological outcomes. Prospective studies could also examine whether reductions in UPF intake result in improvements in gastrointestinal symptoms, sleep, cognitive performance, anxiety, and depression.

Future research should incorporate objective or biological measures such as dietary records, anthropometric indicators, inflammatory biomarkers, and gut microbiome measures. Larger and more diverse samples across different Indian regions would also improve generalisability.

Randomised dietary interventions would be particularly valuable for determining whether reducing UPF intake produces measurable improvements in adolescent mental wellbeing.

Conclusion

This study provides compelling preliminary evidence that ultra-processed food (UPF) consumption is intrinsically linked to multiple facets of adolescent health. Higher UPF intake significantly correlates with increased gastrointestinal distress, heightened anxiety and depression, disrupted sleep patterns, and impaired executive functioning.

Mediation analysis underscores the central role of gastrointestinal symptoms in these associations, particularly in the pathways leading to anxiety and diminished quality of life. Furthermore, executive functioning (Stroop performance) emerged as a key link between UPF consumption and anxiety. While sleep quality and executive function did not consistently mediate all outcomes, their strong associations with UPF intake highlight the multi-dimensional impact of dietary choices.

Collectively, these findings advocate for a holistic, biopsychosocial approach to adolescent health, positioning dietary quality as a critical determinant of gut health and psychological well-being. The prominent role of the gut-brain axis suggests that targeting GI health through nutritional interventions could yield significant benefits for adolescent mental health. While the cross-sectional nature of this study precludes causal claims, it establishes a robust foundation for future longitudinal research and public health initiatives aimed at reducing UPF consumption among youth.

Declarations and Ethical Approval

This study was conducted as an independent research project under experienced Psychiatrist and Psychologists. The sample collection was approved and facilitated by Aarunya Mental Health Center. Written informed consent/assent was obtained from parents/guardians and adolescent participants as appropriate.

Funding

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Conflict of Interest

The authors declare no conflict of interest.

Data Availability Statement

The datasets generated and/or analysed during the current study are available from the corresponding author on reasonable request.

References

1. Adjibade, M., Julia, C., Allès, B., Touvier, M., Lemogne, C., Srouf, B., Hercberg, S., Galan, P., Assmann, K. E., & Kesse-Guyot, E. (2019). Prospective association between ultra-processed food consumption and incident depressive symptoms in the French NutriNet-Santé cohort. *BMC Medicine*, 17, Article 78. <https://doi.org/10.1186/s12916-019-1312-y>
2. Atzeni, A., Hernández-Cacho, A., Khoury, N., Babio, N., Belzer, C., Vioque, J., Corella, D., Fitó, M., Clish, C., Vidal, J., Konstanti, P., Gonzales-Palacios, S., Coltell, O., Goday, A., Moreno Indias, I., Carlos Chillerón, S., Ruiz-Canela, M., Tinahones, F. J., Hu, F. B., & Salas-Salvadó, J. (2025). The link between ultra-processed food consumption, fecal microbiota, and metabolomic profiles in older Mediterranean adults at high cardiovascular risk. *Nutrition Journal*, 24, Article 62. <https://doi.org/10.1186/s12937-025-01125-5>
3. Atzeni, A., Hernández-Cacho, A., Khoury, N., Babio, N., Belzer, C., Vioque, J., Corella, D., Fitó, M., Coltell, O., Goday, A., Moreno Indias, I., Ruiz-Canela, M., Tinahones, F. J., Hu, F. B., & Salas-Salvadó, J. (2022). Association between ultra-processed food consumption and gut microbiota in senior subjects with overweight/obesity and metabolic syndrome. *Frontiers in Nutrition*, 9, Article 976547. <https://doi.org/10.3389/fnut.2022.976547>
4. Bala, J., Sukhoi, O., Newson, J. J., Machado, P. P., Lawrence, M., & Thiagarajan, T. C. (2025). Estimation of the nature and magnitude of mental distress in the population associated with ultra-processed food consumption. *Frontiers in Nutrition*, 12, Article 1562286. <https://doi.org/10.3389/fnut.2025.1562286>
5. Beck, A. T., Epstein, N., Brown, G., & Steer, R. A. (1988). An inventory for measuring clinical anxiety: Psychometric properties. *Journal of Consulting and Clinical Psychology*, 56(6), 893–897.
6. Beck, A. T., Ward, C. H., Mendelson, M., Mock, J., & Erbaugh, J. (1961). An inventory for measuring depression. *Archives of General Psychiatry*, 4(6), 561–571.
7. Bevilacqua, A., Speranza, B., Racioppo, A., Santillo, A., Albenzio, M., Derossi, A., Caporizzi, R., Francavilla, M., Racca, D., Flagella, Z., De Santis, M. A., Elia, A., Conversa, G., Luchetti, L., Sinigaglia, M., & Corbo, M. R. (2024). Ultra-processed food and gut microbiota: Do additives affect eubiosis? A narrative review. *Nutrients*, 17(1), Article 2. <https://doi.org/10.3390/nu17010002>
8. Braun, A., Jakubisin, C., & Emerson, S. (2023). Ultra-processed food consumption is negatively correlated with measures of executive function in healthy adults. *Journal of the Academy of Nutrition and Dietetics*. <https://doi.org/10.1016/j.jand.2023.08.066>
9. Brichacek, A. L., Florkowski, M. R., Abiona, E., & Frank, K. M. (2024). Ultra-processed foods: A narrative review of the impact on the human gut microbiome and variations in classification methods. *Nutrients*, 16(11), Article 1738. <https://doi.org/10.3390/nu16111738>
10. Bouzada, D. F., de Paula, W., Barbosa, B. C. R., de Menezes-Júnior, L. A. A., de Freitas, P. H. B., Machado, E. L., Ferreira, L. G., de Freitas, E. D., da Silva, L. S., Nobre, L. N., & Meireles, A. L. (2025). Association between food consumption and quality of life in university students: A multicenter study in Brazil. *Revista Gaúcha de Enfermagem*, 46, Article e20240360. <https://doi.org/10.1590/1983-1447.2025.20240360.en>
11. Buysse, D. J., Reynolds, C. F., Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The Pittsburgh Sleep Quality Index: A new instrument for psychiatric practice and research. *Psychiatry Research*, 28(2), 193–213.
12. Cadenhead, J. W., Martínez-Steele, E., Contento, I., Kushi, L. H., Lee, A. R., Nguyen, T. T. T., Lebwohl, B., Green, P. H. R., & Wolf, R. L. (2023). Diet quality, ultra-processed food consumption, and quality of life in a cross-sectional cohort of adults and teens with celiac disease. *Journal of Human Nutrition and Dietetics*, 36(4), 1144–1158. <https://doi.org/10.1111/jhn.13137>
13. Capra, B. T., Hudson, S., Helder, M. N., Helder, M., Laskaridou, E., Johnson, A. L., Johnson, A. L., Gilmore, C., Gilmore, C., Marinik, E., Marinik, E. L., Hedrick, V. E., Savla, J., David, L. A., Davy, K. P., & Davy, B. M. (2024). Ultra-processed food intake, gut microbiome, and glucose homeostasis in mid-life adults: Background, design, and methods of a controlled feeding trial. *Contemporary Clinical Trials*, 137, Article 107427. <https://doi.org/10.1016/j.cct.2024.107427>

14. Costa, S. L. F., & Freire, S. H. S. L. M. (2025). Impacto do consumo de produtos ultraprocessados na microbiota intestinal: Efeitos, consequências e perspectivas futuras. *Revista Fisio&terapia*, 29(151), 7–8. <https://doi.org/10.69849/revistaft/ma10202510081707>
15. Cuevas-Sierra, A., Milagro, F. I., Aranaz, P., Martínez, J. A., & Riezu-Boj, J. I. (2021). Gut microbiota differences according to ultra-processed food consumption in a Spanish population. *Nutrients*, 13(8), Article 2710. <https://doi.org/10.3390/nu13082710>
16. Cuaranta, I. (2026). Chrononutrition interventions for mental health: Addressing atypical depression, ultra-processed food use disorder, and circadian dysregulation. *Frontiers in Psychiatry*, 16, Article 1603595. <https://doi.org/10.3389/fpsyt.2025.1603595>
17. Cryan, J. F., & Dinan, T. G. (2012). Mind-altering microorganisms: The impact of the gut microbiota on brain and behaviour. *Nature Reviews Neuroscience*, 13(10), 701–712.
18. Dale, H. F., Kolby, M., & Valeur, J. (2025). Ultra-processed food consumption and irritable bowel syndrome: Current evidence and clinical implications. *Nutrients*, 17(22), Article 3567. <https://doi.org/10.3390/nu17223567>
19. Ekici, E. M., Çelik, Ö. M., Göbel, P., & Güzelalp, A. H. (2025). Association between ultra-processed food intake, night eating behavior, and sleep quality: A cross-sectional study from Türkiye. *Health and Quality of Life Outcomes*, 23, Article 103. <https://doi.org/10.1186/s12955-025-02415-6>
20. Faggiani, L. D., de França, P., Seabra, S. G., Sabino, E. C., Qi, L., & Cardoso, M. A. (2025). Effect of ultra-processed food consumption on the gut microbiota in the first year of life: Findings from the MINA-Brazil birth cohort study. *Clinical Nutrition*, 46, 181–190. <https://doi.org/10.1016/j.clnu.2025.01.030>
21. Fatima, G., Halmy, L. G., Takács, K., & Halmy, E. (2025). Exploring the relationship between ultra-processed foods and chronic insomnia. *Acta Alimentaria*. <https://doi.org/10.1556/066.2025.00057>
22. Fernandes, A. E., Rosa, P. W. L., de Melo, M. E., Martins, R. C. R., Santin, F. G. O., de Moura, A. M. S. H., Coelho, G. S. M. A., Sabino, E. C., Cercato, C., & Mancini, M. C. (2023). Differences in the gut microbiota of women according to ultra-processed food consumption. *Nutrition, Metabolism and Cardiovascular Diseases*, 33(1), 84–89. <https://doi.org/10.1016/j.numecd.2022.09.025>
23. Grosso, G., Godos, J., & Micek, A. (2022). Ultra-processed food intake is associated with worse mental health in Southern Italian individuals. *European Journal of Public Health*, 32(Supplement_3), ckac129.553. <https://doi.org/10.1093/eurpub/ckac129.553>
24. Hayes, A. F. (2022). Introduction to mediation, moderation, and conditional process analysis: A regression-based approach (3rd ed.). Guilford Press.
25. Hatamzadeh, H., Moeini, R., & Ghayour-Mobarhan, M. (2025). The association between ultra-processed foods and depression, anxiety and sleep in adults: A cross-sectional study in Iran. *Food Science & Nutrition*, 13(7), Article e70316. <https://doi.org/10.1002/fsn3.70316>
26. Hecht, E. M., Rabil, A., Martinez Steele, E., Abrams, G. A., Ware, D., Landy, D. C., & Hennekens, C. H. (2022). Cross-sectional examination of ultra-processed food consumption and adverse mental health symptoms. *Public Health Nutrition*, 25(11), 3225–3234. <https://doi.org/10.1017/S1368980022001586>
27. Hosseinasab, D., Shiraseb, F., Bahrampour, N., da Silva, A., Hajinasab, M. M., Bressan, J., & Mirzaei, K. (2024). Ultra-processed food consumption and quality of life: A cross-sectional study in Iranian women. *Frontiers in Public Health*, 12, Article 1351510. <https://doi.org/10.3389/fpubh.2024.1351510>
28. Hosseinpour-Niazi, S., Niknam, M., Amiri, P., Mirmiran, P., Ainy, E., Izadi, N., Gaeini, Z., & Azizi, F. (2024). The association between ultra-processed food consumption and health-related quality of life differs across lifestyle and socioeconomic strata. *BMC Public Health*, 24, Article 1955. <https://doi.org/10.1186/s12889-024-19351-7>
29. Irgat, S. İ., & Bakırhan, H. (2026). Ultra-processed food consumption and mental health and sleep quality in Turkish adults: A cross-sectional study. *Healthcare*, 14(5), Article 575. <https://doi.org/10.3390/healthcare14050575>
30. Jafari, A., Momenan, M., Tehrani, A. N., Behrouz, V., & Yari, Z. (2026). Association between ultra-processed food consumption and odds of depression, stress, and anxiety in adolescent girls: A cross-sectional study. *BMC Public Health*, 26, Article 457. <https://doi.org/10.1186/s12889-025-26167-6>
31. Juul, F., & Bere, E. (2024). Ultra-processed foods—A scoping review for Nordic Nutrition Recommendations 2023. *Food & Nutrition Research*, 68, Article 10616. <https://doi.org/10.29219/fnr.v68.10616>
32. Kim, K. A., et al. (2025). P1316 Increased ultra-processed food consumption is associated with inflammation-promoting shift of gut microbiota in inflammatory bowel disease. *Journal of Crohn's and Colitis*, 19(Supplement_1), i2374–i2376. <https://doi.org/10.1093/ecco-jcc/jjae190.1490>
33. Kwan, B. S., Cho, J.-H., Kim, J. Y., Kim, H. I., Ko, N. G., & Park, J. E. (2026). Ultra-processed food consumption and domain-specific quality of life in postmenopausal women: Associations with mobility and mental health. *Healthcare*, 14(6), Article 791. <https://doi.org/10.3390/healthcare14060791>

34. Lane, M. M., Gamage, E., Travica, N., Dissanayaka, T., Ashtree, D. N., Gauci, S., Lotfaliany, M., O'Neil, A., Jacka, F. N., & Marx, W. (2022). Ultra-processed food consumption and mental health: A systematic review and meta-analysis of observational studies. *Nutrients*, 14(13), Article 2568. <https://doi.org/10.3390/nu14132568>
35. Lane, M. M., Gamage, E., Du, S., Ashtree, D. N., McGuinness, A. J., Gauci, S., Baker, P., Lawrence, M., Rebholz, C. M., Srour, B., Touvier, M., Jacka, F. N., O'Neil, A., Segasby, T., & Marx, W. (2024). Ultra-processed food exposure and adverse health outcomes: Umbrella review of epidemiological meta-analyses. *The BMJ*, 384, Article e077310. <https://doi.org/10.1136/bmj-2023-077310>
36. Lane, M. M., Gamage, E., Du, S., Ashtree, D. N., McGuinness, A. J., Gauci, S., Baker, P., Lawrence, M., Rebholz, C. M., Srour, B., Touvier, M., Jacka, F. N., O'Neil, A., Segasby, T., & Marx, W. (2023). Ultra-processed food exposure and adverse health outcomes: Umbrella review of epidemiological meta-analyses [Preprint]. Preprints. <https://doi.org/10.20944/preprints202308.1407.v1>
37. Lee, H., Ludwig-Borycz, E., McEvoy, C. T., Martínez-Steele, E., Khandpur, N., Heeringa, S. G., Ryan, L. H., Langa, K. M., Wolfson, J. A., & Leung, C. W. (2026). Ultra-processed food intake and impairment across multiple cognitive domains in nationally representative older U.S. adults. *Frontiers in Public Health*, 13, Article 1695540. <https://doi.org/10.3389/fpubh.2025.1695540>
38. Lenart, J., Janik, N., Górowska, A., Wadowska, Z., Janowiak, J., Sobiś, M., Bogacka, A., Kiersznowska, N., Buchman, M., & Miłek, B. (2025). The association of ultra-processed food intake with mental health disorders, cognitive impairment, and insomnia: A literature review. *Quality in Sport*, 48, Article 67028. <https://doi.org/10.12775/QS.2025.48.67028>
39. Marchianti, A. C. N., & Hasan, M. (2023). Hubungan antara pola makan dan aktivitas fisik terhadap tingkat depresi di masa pandemi pada pelajar SMAN 1 Manyar Gresik, Indonesia. *Amerta Nutrition*, 7(2), 283–294. <https://doi.org/10.20473/amnt.v7i2.2023.283-294>
40. Mazloomi, S. N., Talebi, S., Mehrabani, S., Bagheri, R., Ghavami, A., Zarpoosh, M., Mohammadi, H., Wong, A., Nordvall, M., Kermani, M. A. H., & Moradi, S. (2023). The association of ultra-processed food consumption with adult mental health disorders: A systematic review and dose-response meta-analysis of 260,385 participants. *Nutritional Neuroscience*, 26(10), 913–931. <https://doi.org/10.1080/1028415X.2022.2110188>
41. Moncada, J. G. A., & Morán, J. C. B. (2025). Alimentos ultraprocesados y su impacto en las habilidades cognitivas de estudiantes universitarios: Una revisión sistemática. *Ciencia Latina Revista Científica Multidisciplinar*, 9(5), 8803–8821. https://doi.org/10.37811/cl_rcm.v9i5.20211
42. Monteiro, C. A., Cannon, G., Levy, R. B., Moubarac, J.-C., Louzada, M. L. C., Rauber, F., Khandpur, N., Cediel, G., Neri, D., Martinez-Steele, E., Baraldi, L. G., & Jaime, P. C. (2019). Ultra-processed foods: What they are and how to identify them. *Public Health Nutrition*, 22(5), 936–941.
43. Nguyen, L., Walters, J., Spies, B., Coppus, A., Massie, J., & Bertonha, T. P. (2025). Food for thought: A systematic review and meta-analysis on the effects of ultra-processed foods on cognition in children and adolescents. *Food Frontiers*, 6(4), 1838–1866. <https://doi.org/10.1002/fft2.70064>
44. Öztürk, Y. E., & Uzdil, Z. (2025). Ultra-processed food consumption is linked to quality of life and mental distress among university students. *PeerJ*, 13, Article e19931. <https://doi.org/10.7717/peerj.19931>
45. Poon, E., Li, C., Schweitzer, D., & Akefe, I. (2026). Neurobiological insights into the effects of ultra-processed food on lipid metabolism and associated mental health conditions: A scoping review. *Frontiers in Nutrition*, 12, Article 1754492. <https://doi.org/10.3389/fnut.2025.1754492>
46. Shah, N. (2023). Gut feelings: Examining the impact of ultra-processed foods on public health outcomes and interventions targeting the gut microbiota. *Indian Journal of Applied Research*. <https://doi.org/10.36106/ijar/7402710>
47. Stroop, J. R. (1935). Studies of interference in serial verbal reactions. *Journal of Experimental Psychology*, 18(6), 643–662.
48. Svedlund, J., Sjödin, I., & Dotevall, G. (1988). GSRS—A clinical rating scale for gastrointestinal symptoms in patients with irritable bowel syndrome and peptic ulcer disease. *Digestive Diseases and Sciences*, 33(2), 129–134.
49. Travinsky-Shmul, T., Beresh, O., Zaretsky, J., Griess-Fishheimer, S., Rozner, R., Kalev-Altman, R., Penn, S., Shahar, R., & Monsonego-Ornan, E. (2021). Ultra-processed food impairs bone quality, increases marrow adiposity and alters gut microbiome in mice. *Foods*, 10(12), Article 3107. <https://doi.org/10.3390/foods10123107>
50. Werneck, A. O., Vancampfort, D., Oyeyemi, A. L., Stubbs, B., & Silva, D. R. (2020). Joint association of ultra-processed food and sedentary behavior with anxiety-induced sleep disturbance among Brazilian adolescents. *Journal of Affective Disorders*, 266, 135–142. <https://doi.org/10.1016/j.jad.2020.01.104>
51. Werneck, A. O., Hoare, E., & Silva, D. R. (2021). Do TV viewing and frequency of ultra-processed food consumption share mediators in relation to adolescent anxiety-induced sleep disturbance? *Public Health Nutrition*, 24(16), 5491–

5497. <https://doi.org/10.1017/S1368980021000379>

52. Werneck, A. O., Steele, E. M., Delpino, F. M., Lane, M. M., Marx, W., Jacka, F. N., Stubbs, B., Touvier, M., Srouf, B., Louzada, M. L. C., Levy, R. B., & Monteiro, C. A. (2024). Adherence to the ultra-processed dietary pattern and risk of depressive outcomes: Findings from the NutriNet Brasil cohort study and an updated systematic review and meta-analysis. *Clinical Nutrition*, 43(5), 1190–1199. <https://doi.org/10.1016/j.clnu.2024.03.028>
53. Zinöcker, M. K., & Lindseth, I. A. (2018). The Western diet–microbiome–host interaction and its role in metabolic disease. *Nutrients*, 10(3), Article 365. <https://doi.org/10.3390/nu10030365>