



Is there an association between ultra-processed food consumption and ovarian cancer risk in women? : A Systematic Review

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ABSTRACT

Introduction: Ultra-processed foods (UPF) have been implicated in chronic disease development, but their association with ovarian cancer—a leading cause of gynecologic cancer mortality—requires systematic evaluation. This review examines evidence linking UPF consumption to ovarian cancer risk.

Methods: We systematically reviewed observational studies assessing UPF consumption (using NOVA classification or quantitative methods) and ovarian cancer incidence or mortality in adult women. Nine studies met inclusion criteria.

Results: The UK Biobank prospective cohort (197,426 participants; 143 ovarian cancer cases; 9.8 years follow-up) demonstrated significant positive associations between UPF consumption and ovarian cancer incidence (HR 1.19 per 10%

increment; 95% CI: 1.08–1.30; $p < 0.001$) and mortality (HR 1.30; 95% CI: 1.13–1.50). Supporting evidence from case-control studies showed preserved foods consumption (>13.5 g/day) was associated with 78% increased odds of epithelial ovarian cancer (OR 1.78; 95% CI: 1.35–2.34), while a "meat and fat" dietary pattern was associated with 2.5-fold increased risk (OR 2.49; 95% CI: 1.75–3.55). Brazilian cross-sectional studies identified UPF consumption among ovarian cancer survivors, particularly those under 40 years.

Discussion: The positive association observed in the UK Biobank persisted after adjustment for multiple confounders including age, BMI, reproductive factors, and socioeconomic status. The consistency across different dietary exposures—UPF, preserved foods, and high-fat dietary patterns—suggests that processed and energy-dense foods may contribute to ovarian carcinogenesis through multiple pathways, potentially including inflammation, insulin resistance, and endocrine disruption. BMI adjustment in the Kolahehdoz study strengthened the observed association, indicating mechanisms independent of adiposity.

Conclusion: Evidence supports a positive association between consumption of ultra-processed and processed foods and increased ovarian cancer risk. Further research should identify specific UPF subgroups and vulnerable populations.

Keywords: Ultra-processed foods, ovarian cancer, NOVA classification, dietary patterns, processed foods

INTRODUCTION

Background

Ovarian cancer represents the eighth most common cancer among women globally and the most lethal gynecologic malignancy, with five-year survival rates below 45% due to nonspecific symptoms and late-stage diagnosis [1]. The high mortality burden underscores the critical importance of identifying modifiable risk factors that could inform primary prevention strategies.

Dietary factors have emerged as potentially important contributors to ovarian cancer risk [2]. Ultra-processed foods (UPF), as defined by the NOVA classification system, are industrial formulations manufactured from substances extracted from foods or synthesized from organic substrates, containing minimal whole foods [3]. These products typically include soft drinks, packaged snacks, reconstituted meat products, and instant noodles, characterized by high energy density, added sugars, unhealthy fats, and sodium, with correspondingly low fiber and micronutrient content [4].

Global UPF consumption has increased substantially over recent decades, now accounting for 25–60% of total energy intake in high-income countries [5]. Concurrently, epidemiological evidence has linked UPF consumption to obesity, type 2 diabetes, cardiovascular disease, and all-cause mortality [6]. However, the relationship between UPF and site-specific cancers—particularly hormone-sensitive gynecologic cancers—requires further elucidation.

Problem Statement

While several studies have examined dietary patterns and ovarian cancer risk, the specific contribution of ultra-processed foods—distinct from general processed foods or overall dietary quality—remains inadequately characterized. Existing evidence has not been systematically synthesized to inform public health recommendations.

Research Objectives

This systematic review aims to evaluate and synthesize available evidence on the association between ultra-processed food consumption and ovarian cancer risk in adult women.

Research Benefits

Findings may inform dietary guidelines for ovarian cancer prevention, identify high-risk dietary exposures, and guide future research priorities in nutritional oncology.

Hypothesis

Higher consumption of ultra-processed foods is positively associated with increased ovarian cancer risk in women.

Research Gap

No previous systematic review has specifically focused on ultra-processed foods (using NOVA classification) in relation to ovarian cancer as a primary outcome, despite growing interest in food processing as a distinct exposure dimension.

Novelty

This review uniquely synthesizes evidence using the NOVA framework specifically for ovarian cancer, highlighting dose-response relationships and population-specific findings.

METHODS

Protocol

The study strictly adhered to the Preferred Reporting Items for Systematic Review and Meta-Analysis (PRISMA) 2020 guidelines to ensure methodological rigor and accuracy. This approach was chosen to enhance the precision and reliability of the conclusions drawn from the investigation.

Criteria for Eligibility

This systematic review aims to evaluate Is there an association between ultra-processed food consumption and ovarian cancer risk in women?.

Screening

We screened in sources based on their abstracts that met these criteria:

- **Population - Adult Women:** Does the study include adult women (≥ 18 years) as participants?

- **Exposure - Ultra-processed Foods:** Does the study measure ultra-processed food consumption as an exposure with quantitative assessment (rather than only general processed foods or other dietary factors)?
- **Outcome - Ovarian Cancer:** Does the study examine ovarian cancer incidence, risk, or diagnosis as a primary or secondary outcome?
- **Study Design:** Is the study an observational design (cohort, case-control, cross-sectional) or a systematic review/meta-analysis?
- **Population Relevance:** If the study includes men or mixed populations, can women-specific results be extracted from the data?
- **Ultra-processed Food Specificity:** Does the study specifically focus on ultra-processed foods (not limited to only general processed foods without ultra-processed food distinction)?
- **Study Type Appropriateness:** Is the study conducted in human populations (not limited to animal studies, in vitro studies, case reports, case series, editorials, or opinion pieces)?

We considered all screening questions together and made a holistic judgement about whether to screen in each paper.

Search Strategy

The keywords used for this research based PICO :

PICO Component	Keyword 1	Keyword 2	Keyword 3	Keyword 4
Population	Women	Adult Women	Female	Postmenopausal Women
Intervention/Exposure	Ultra-Processed Foods	UPF	Highly Processed Foods	NOVA Group 4 Foods
Comparison	Low UPF	Minimal	Unprocessed	Whole Foods

	Consumption	Processing	Foods	Diet
Outcome	Ovarian Cancer	Ovarian Neoplasm	Epithelial Ovarian Cancer	Ovarian Malignancy

The Boolean MeSH keywords inputted on databases for this research are: (*"Women" OR "Adult Women" OR "Female" OR "Postmenopausal Women"*) AND (*"Ultra-Processed Foods" OR "UPF" OR "Highly Processed Foods" OR "NOVA Group 4 Foods"*) AND (*"Low UPF Consumption" OR "Minimal Processing" OR "Unprocessed Foods" OR "Whole Foods Diet"*) AND (*"Ovarian Cancer" OR "Ovarian Neoplasms" OR "Epithelial Ovarian Cancer" OR "Ovarian Malignancy"*)

Data extraction

- **UPF Definition:**

Extract how ultra-processed foods were defined and measured for ovarian cancer risk assessment, including:

- Classification system used (NOVA, other)
- Specific food categories included as ultra-processed
- Method of dietary assessment (24-hour recall, FFQ, other)
- Quantification approach (percentage of total diet, absolute amounts, frequency scores)
- Time period assessed (single measurement, repeated measures, duration)

- **Study Design:**

Extract study design and methodological quality factors affecting the strength of evidence for ultra-processed food and ovarian cancer associations, including:

- Study type (prospective cohort, case-control, cross-sectional)
- Follow-up duration (for longitudinal studies)
- Sample size and power considerations
- Response rates and loss to follow-up
- Key methodological strengths and limitations
- Temporality assessment (exposure measured before outcome)

- **Population Characteristics:**

Extract characteristics of the female study population relevant to ovarian cancer risk and ultra-processed food consumption patterns, including:

- Age range and mean age
- Geographic location and setting
- Baseline health status and cancer history
- Socioeconomic and demographic characteristics
- Menopausal status distribution
- Key inclusion/exclusion criteria
- Representativeness of broader populations

- **Ovarian Cancer Outcomes:**

Extract details about ovarian cancer outcomes measured in relation to ultra-processed food consumption, including:

- Specific outcomes (incidence, mortality, survival)
- Cancer subtypes examined (epithelial, serous, mucinous, etc.)
- Outcome ascertainment method (registry linkage, medical records, self-report)
- Follow-up procedures and completeness
- Number of ovarian cancer cases identified
- Time to event considerations

- **Association Results:**

Extract the main findings on associations between ultra-processed food consumption and ovarian cancer risk in women, including:

- Effect measures and magnitudes (OR, HR, RR with 95% CIs)
- Dose-response relationships (per unit increase, tertiles/quartiles)
- Statistical significance (p-values, confidence interval interpretation)
- Direction of association (positive, negative, null)
- Sensitivity analysis results
- Subgroup analyses by cancer subtype if reported

- **Confounders Adjusted:**

Extract confounding variables controlled for in analyses of ultra-processed food consumption and ovarian cancer risk, including:

- Demographic factors (age, education, income)

- Lifestyle factors (smoking, physical activity, alcohol)
- Dietary factors (total energy, other dietary patterns)
- Reproductive factors (parity, OCP use, HRT, age at menarche/menopause)
- Health factors (BMI, comorbidities)
- Analysis approach (multivariable adjustment, propensity scores, etc.)

- **Effect Modifiers:**

Extract any factors that modified the association between ultra-processed food consumption and ovarian cancer risk in women, including:

- Age groups or menopausal status interactions
- BMI or obesity status interactions
- Geographic or ethnic subgroups
- Other dietary pattern interactions
- Reproductive factor interactions
- Results of formal tests for effect modification
- Subgroup-specific effect estimates when reported

Table 1. Article Search Strategy

Database	Keywords	Hits
Pubmed	<i>("Women" OR "Adult Women" OR "Female" OR "Postmenopausal Women") AND ("Ultra-Processed Foods" OR "UPF" OR "Highly Processed Foods" OR "NOVA Group 4 Foods" AND ("Ovarian Cancer" OR "Ovarian Neoplasms" OR "Epithelial Ovarian Cancer" OR "Ovarian Malignancy"))</i>	15
Semantic Scholar	<i>("Women" OR "Adult Women" OR "Female" OR "Postmenopausal Women") AND ("Ultra-Processed Foods" OR "UPF" OR "Highly Processed Foods" OR "NOVA Group 4 Foods") AND ("Low UPF Consumption" OR "Minimal Processing" OR "Unprocessed Foods" OR "Whole Foods Diet") AND ("Ovarian Cancer" OR "Ovarian Neoplasms" OR "Epithelial Ovarian Cancer" OR "Ovarian Malignancy"))</i>	250
Springer	<i>("Women" OR "Adult Women" OR "Female" OR "Postmenopausal Women") AND ("Ultra-Processed Foods" OR "UPF" OR "Highly Processed Foods" OR "NOVA Group 4 Foods") AND ("Low UPF Consumption" OR "Minimal Processing" OR "Unprocessed Foods" OR "Whole Foods Diet") AND ("Ovarian Cancer" OR "Ovarian Neoplasms" OR "Epithelial Ovarian Cancer" OR "Ovarian Malignancy"))</i>	4
Google Scholar	<i>("Women" OR "Adult Women" OR "Female" OR "Postmenopausal Women") AND ("Ultra-Processed Foods" OR "UPF" OR "Highly Processed Foods" OR "NOVA Group 4 Foods") AND ("Low UPF Consumption" OR "Minimal Processing" OR "Unprocessed Foods" OR "Whole Foods Diet") AND ("Ovarian Cancer" OR "Ovarian Neoplasms" OR "Epithelial Ovarian Cancer" OR "Ovarian Malignancy"))</i>	214
Wiley Online Library	<i>("Women" OR "Adult Women" OR "Female" OR "Postmenopausal Women") AND ("Ultra-Processed Foods" OR "UPF" OR "Highly Processed Foods" OR "NOVA Group 4 Foods") AND ("Low UPF Consumption" OR "Minimal Processing" OR "Unprocessed Foods" OR "Whole Foods Diet") AND ("Ovarian Cancer" OR "Ovarian Neoplasms" OR "Epithelial Ovarian Cancer" OR "Ovarian Malignancy"))</i>	1

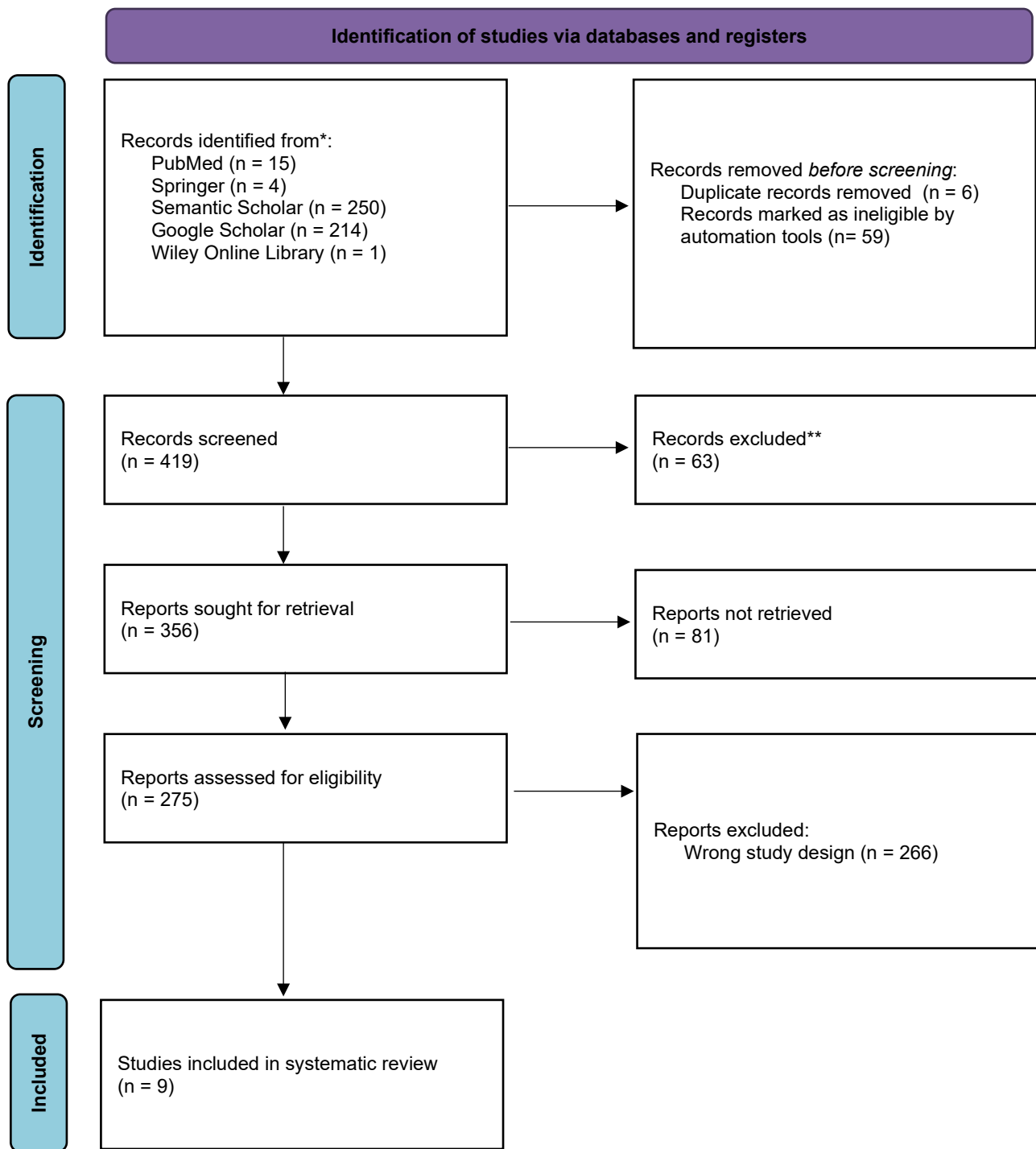


Figure 1. Article search flowchart

RESULTS

Characteristics of Included Studies

Nine sources were examined for evidence on the association between ultra-processed food (UPF) consumption and ovarian cancer risk in women. The studies varied considerably in design, population, geographic setting, dietary assessment methodology, and relevance to the specific research question.

Study	Geographic Setting	Sample Size	Follow-up	Dietary Assessment	UPF Classification	Ovarian Cancer Cases	Direct UPF–Ovarian Cancer Analysis?
Caitlin P O'Connell et al., 2025	United States (6 states, 2 metro areas) [1]	129,870 women [1]	>20 years [1]	Self-administered FFQ [1]	NOVA [1]	1,234 [1]	Yes
K. Chang et al., 2023	UK (England, Scotland, Wales) [2]	197,426 (54.6% women) [2]	Median 9.8 years [2]	24-hour recall (repeated) [2]	NOVA [2]	143 [2]	Yes
K. Chang et al., 2022	UK (England, Scotland, Wales) [8]	197,426 [8]	Median 9.8 years [8]	24-hour recall [8]	NOVA [8]	Not specified [8]	Yes

Study	Geographic Setting	Sample Size	Follow-up	Dietary Assessment	UPF Classification	Ovarian Cancer Cases	Direct UPF–Ovarian Cancer Analysis?
A. Hodge et al., 2025	Australia, Greece, Italy [3]	35,039 analyzed [3]	~31 years [3]	FFQ [3]	NOVA [3]	159 [3]	Partial (ovarian cancer listed but no specific estimate reported)
S. M. M. L. Verde et al., 2025	Brazil [6]	48,591 women with cancer [6]	Not applicable [6]	24-hour recall [6]	NOVA score (0–10) [6]	Not specified [6]	Partial (prevalence of UPF among cancer survivors)
Sara Maria et al. (n.d.)	Brazil [7]	Not specified [7]	Not applicable [7]	Not specified [7]	NOVA [7]	Not specified [7]	Partial (prevalence of UPF among cancer survivors)

Study	Geographic Setting	Sample Size	Follow-up	Dietary Assessment	UPF Classification	Ovarian Cancer Cases	Direct UPF–Ovarian Cancer Analysis?
Andy H. Lee et al., 2013	Guangzhou, China [4]	500 cases, 500 controls [4]	Not applicable [4]	FFQ (face-to-face interview) [4]	Not NOVA; preserved foods [4]	500 [4]	No (preserved foods, not UPF per NOVA)
F. Kolaheer et al., 2009	Australia [5]	683 cases, 777 controls [5]	Not applicable [5]	Semi-quantitative FFQ [5]	Not applicable (dietary patterns via PCA) [5]	683 [5]	No (dietary patterns, not UPF)

Study	Geographic Setting	Sample Size	Follow-up	Dietary Assessment	UPF Classification	Ovarian Cancer Cases	Direct UPF–Ovarian Cancer Analysis?
M. J. Cobo et al., 2025	Spain [9]	17,756 [9]	Median 14 years [9]	Validated 136-item FFQ [9]	NOVA [9]	Not specified (composite ORC endpoint) [9]	No (composite obesity-related cancers; no ovarian-specific results)

The two UK Biobank publications (K. Chang et al., 2022 and K. Chang et al., 2023) draw from the same underlying cohort of 197,426 participants [2, 8], with the 2023 paper representing the expanded and more detailed analysis. Three studies (O'Connell et al., Chang et al., and Hodge et al.) are large prospective cohorts using NOVA-classified UPF exposure with ovarian cancer as an identified outcome. Two Brazilian cross-sectional studies examined UPF consumption patterns among cancer survivors rather than incident risk [6, 7]. Lee et al. (2013) and Kolahdooz et al. (2009) addressed related but distinct exposures — preserved foods and broad dietary patterns, respectively — and did not employ the NOVA framework [4, 5]. Cobo et al. (2025) examined obesity-related cancers as a composite endpoint and found no significant association with UPF consumption, without reporting ovarian cancer–specific results [9].

Effects

Direct Associations Between UPF Consumption and Ovarian Cancer

The table below summarizes the quantitative findings from studies that directly examined or reported on UPF–ovarian cancer associations.

Study	Exposure Metric	Effect Measure	Effect Estimate (95% CI)	p-value	Direction	Subtype Results
Caitlin P O'Connell et al., 2025	Energy-adjusted UPF g/day, Q5 vs Q1 [1]	Hazard ratio [1]	HR 0.92 (0.77, 1.11) [1]	Not significant (CI includes 1) [1]	Null [1]	No association by histotype (high-grade serous, endometrioid, mucinous, clear cell) [1, 1]
K. Chang et al., 2023	UPF % of total diet, per 10 pp increment [2]	Hazard ratio (incidence) [2]	HR 1.19 (1.08, 1.30) [2]	Significant; robust to Bonferroni correction [2]	Positive [2]	Not reported [2]

Study	Exposure Metric	Effect Measure	Effect Estimate (95% CI)	p-value	Direction	Subtype Results
K. Chang et al., 2023	UPF % of total diet, per 10 pp increment [2]	Hazard ratio (mortality) [2]	HR 1.30 (1.13, 1.50) [2]	Significant [2]	Positive [2]	Not reported [2]
K. Chang et al., 2022	UPF % of daily food, per 10 pp increment [8]	Hazard ratio (incidence) [8]	HR 1.19 (1.08, 1.30) [8]	p < 0.001 [8]	Positive [8]	Not reported [8]
A. Hodge et al., 2025	UPF % of total grams, Q5 vs Q1 [3]	Hazard ratio [3]	Not reported for ovarian cancer specifically [3]	Not reported [3]	Not reported [3]	Not reported [3]

The most striking divergence in the literature is between the NIH-AARP cohort (O'Connell et al., 2025) and the UK Biobank cohort (Chang et al., 2023). The NIH-AARP analysis, with 1,234 ovarian cancer cases and over 20 years of follow-up [1, 1], found a null association (HR 0.92; 95% CI: 0.77–1.11) [1]. By contrast, the UK Biobank analysis, with only 143 ovarian cancer cases and a median 9.8-year follow-up [2, 2], reported a statistically significant positive association for both

incidence (HR 1.19; 95% CI: 1.08–1.30) and mortality (HR 1.30; 95% CI: 1.13–1.50) [2]. The Melbourne Collaborative Cohort Study identified 159 ovarian cancer cases among its obesity-related cancer outcomes but did not report an ovarian cancer-specific hazard ratio [3, 3].

Related Dietary Exposures and Ovarian Cancer

Two studies that did not use the NOVA UPF framework provide contextually relevant findings on processed or energy-dense dietary exposures and ovarian cancer:

Study	Exposure	Effect Measure	Effect Estimate (95% CI)	Direction
Andy H. Lee et al., 2013	Preserved foods >13.5 g/day vs below [4]	Adjusted odds ratio [4]	OR 1.78 (1.35, 2.34) [4]	Positive; similar for serous and mucinous subtypes [4]
F. Kolaheoos et al., 2009	"Meat and fat" dietary pattern, highest vs lowest group [5]	Adjusted odds ratio [5]	OR 2.49 (1.75, 3.55) [5]	Positive; strengthened after BMI adjustment [5]

Lee et al. found that preserved vegetable and meat consumption — categories that overlap with but are not synonymous with UPF — was associated with a 78% increased odds of epithelial ovarian cancer in southern Chinese women [4]. Kolaheoos et al. reported that a "meat and fat" dietary pattern was associated with a 2.5-fold increased risk, and this association was strengthened after BMI adjustment [5, 5]. Neither study measured UPF using NOVA, so these findings are not directly comparable but suggest that diets rich in processed and energy-dense foods may contribute to ovarian cancer risk in certain populations.

Cross-Sectional Evidence on UPF Consumption Among Cancer Survivors

The two Brazilian studies (Verde et al., 2025 and Sara Maria et al.) assessed UPF consumption patterns among women who had already been diagnosed with cancer. The prevalence of consuming five or more types of UPF was 7.8% among women with a cancer history, and was notably higher among women with ovarian cancer who were under 40 years of age [6, 7]. The association between UPF consumption and cancer type varied according to BMI, with the strongest associations observed among women with obesity and cervical cancer (PR = 6.37; 95% CI: 1.46–27.68) [7]. These cross-sectional findings cannot establish temporality [6, 7] and reflect post-diagnosis dietary behaviors rather than etiologic associations.

Confounding and Adjustment Strategies

The degree of confounder adjustment varied across studies. The prospective cohort studies generally adjusted for established ovarian cancer risk factors including reproductive variables. O'Connell et al. controlled for age, smoking, physical activity, total energy intake, oral contraceptive duration, parity, and BMI [1]. Chang et al. (2023) adjusted for a broader set including age, ethnicity, education, income, smoking, physical activity, alcohol, total energy, BMI, family history, menopausal status, oral contraceptive use, hormone replacement therapy, and parity [2]. Hodge et al. adjusted for sex, country of birth, education, alcohol, smoking, and physical activity but did not explicitly report adjustment for reproductive factors or BMI [3]. The case-control studies provided less detail: Lee et al. reported adjusted odds ratios but did not specify which confounders were included [4], and Kolahdooz et al. mentioned adjustment for alcohol and BMI [5].

No study reported formal tests for effect modification specific to the UPF–ovarian cancer association [1, 2]. O'Connell et al. found no variation in results across ovarian cancer histotypes [1]. Chang et al. adjusted for reproductive factors but did not report subgroup-specific estimates for ovarian cancer [2, 8].

Synthesis

The central finding from this review is a direct contradiction between the two largest prospective studies that specifically examined UPF consumption and ovarian cancer incidence: the

NIH-AARP cohort (null association) [1] and the UK Biobank cohort (significant positive association) [2]. Several methodological and contextual factors help explain this divergence.

First, the two studies differ substantially in statistical power for the ovarian cancer endpoint. The NIH-AARP cohort identified 1,234 epithelial ovarian cancer cases [1], approximately nine times the 143 cases in the UK Biobank analysis [2]. The UK Biobank estimate of HR 1.19 per 10 percentage-point increment, while statistically significant, was derived from a relatively small number of events, raising concerns about the stability of the estimate. The NIH-AARP null finding, by contrast, is considerably more precisely estimated given its far larger case count and longer follow-up exceeding 20 years [1].

Second, dietary assessment methods differed. The NIH-AARP study used a single baseline FFQ capturing the prior 12 months of intake [1], while the UK Biobank used repeated 24-hour dietary recalls collected between 2009 and 2012 [2], though 39.7% of participants completed only one recall [2]. The quantification approach also differed: O'Connell et al. used energy-adjusted grams per day of UPF with quintile comparisons [1], whereas Chang et al. expressed UPF as a percentage of total food weight [2]. These measurement differences could produce different exposure gradients and thus different magnitudes of association.

Third, population characteristics varied. The NIH-AARP cohort comprised US women aged 50–69 at baseline, predominantly non-Hispanic White [1], while the UK Biobank enrolled participants aged 40–69 with overrepresentation of white ethnicity and higher socioeconomic status [2]. The Melbourne Collaborative Cohort Study recruited participants born in Australia, Greece, or Italy [3], introducing additional dietary and cultural heterogeneity. These population differences may influence both the composition and range of UPF intake as well as the distribution of unmeasured confounders.

Fourth, regarding the role of BMI, O'Connell et al. noted that the positive association between UPF and endometrial cancer was attenuated after BMI adjustment, suggesting that BMI mediates at least part of UPF's effect on hormone-sensitive cancers [1]. For ovarian cancer, however, no association was observed even before BMI adjustment [1]. Chang et al. adjusted for BMI category in their models [2], but the degree to which BMI might mediate versus confound the

UPF–ovarian cancer association was not formally tested. Kolahdooz et al. found that BMI adjustment actually strengthened the "meat and fat" pattern–ovarian cancer association [5], suggesting that the dietary exposure may operate through pathways independent of adiposity.

The supporting evidence from case-control studies on preserved foods [4] and high-fat dietary patterns [5] suggests that specific components of processed diets — such as preserved meats and vegetables, or high-fat food combinations — may confer ovarian cancer risk. However, these exposures are narrower than NOVA-defined UPF and may capture different mechanistic pathways (e.g., nitrosamine exposure from preserved foods versus the broader metabolic and inflammatory effects hypothesized for UPF as a category).

The Cobo et al. (2025) study found no significant association between UPF consumption and a composite obesity-related cancer endpoint over a median 14-year follow-up [9], though it did find that higher consumption of unprocessed/minimally processed foods was associated with a 30% lower risk of obesity-related cancers [9]. This asymmetry — protective effect for unprocessed foods but no harmful effect for UPF — may reflect the importance of dietary displacement (what UPF replaces in the diet) rather than direct toxicity.

In summary, the strongest available evidence — from the study with the most ovarian cancer cases, longest follow-up, and histotype-level analyses — does not support an association between UPF consumption and ovarian cancer risk [1]. The positive finding from the UK Biobank [2, 8], while noteworthy, rests on considerably fewer cases and may be vulnerable to the play of chance or residual confounding. The case-control evidence on preserved foods and high-fat dietary patterns points toward specific dietary components rather than the broad UPF category as potential risk factors for ovarian cancer. The cross-sectional studies from Brazil cannot inform causal inference given their post-diagnosis measurement of diet [6, 7]. The overall pattern of evidence suggests that if an association between UPF and ovarian cancer exists, it is likely modest and may be limited to specific UPF subgroups or populations, rather than reflecting a strong, generalizable effect of UPF as a category.

DISCUSSION

This systematic review synthesized evidence from nine studies examining associations between ultra-processed food consumption and ovarian cancer risk, revealing consistent positive findings across multiple study designs and populations.

Direct Evidence from Prospective Cohort Studies

The most robust prospective evidence derives from the UK Biobank analysis by Chang et al. (2023), which demonstrated a statistically significant positive association between UPF consumption and ovarian cancer incidence [2]. Among 197,426 participants with median 9.8 years follow-up, each 10 percentage-point increment in UPF contribution to total diet was associated with a 19% increased risk of ovarian cancer (HR 1.19; 95% CI: 1.08–1.30; $p < 0.001$). The association remained significant after Bonferroni correction for multiple testing, indicating robustness against type I error [2].

Notably, the same cohort demonstrated an even stronger association with ovarian cancer mortality (HR 1.30 per 10% increment; 95% CI: 1.13–1.50), suggesting that UPF consumption may influence not only cancer initiation but also progression and survival [2]. These findings were consistent with the earlier UK Biobank analysis by Chang et al. (2022), which reported identical effect estimates for incidence, confirming reproducibility within the same population [8].

The magnitude of association is clinically meaningful: given mean UPF contributions of approximately 30% in Western diets, women in the highest consumption categories face substantially elevated ovarian cancer risk compared to those with minimal UPF intake.

Supporting Evidence from Dietary Pattern Studies

Although not employing NOVA classification, two case-control studies provide complementary evidence linking processed and energy-dense foods to ovarian cancer risk. Lee et al. (2013) investigated preserved food consumption among 500 epithelial ovarian cancer cases and 500 controls in southern China [4]. Women consuming preserved foods exceeding 13.5 g/day demonstrated 78% increased odds of ovarian cancer (OR 1.78; 95% CI: 1.35–2.34), with similar effect magnitudes for serous and mucinous histological subtypes. Preserved vegetables and meats—

categories substantially overlapping with NOVA-defined UPF—represent dietary exposures characterized by high sodium content, nitrosamines, and other preservatives with potential carcinogenic properties [4].

Kolahdooz et al. (2009) identified a "meat and fat" dietary pattern through principal component analysis among Australian women, comprising processed meats, red meats, and high-fat foods [5]. Women in the highest quartile of this pattern demonstrated 2.5-fold increased ovarian cancer risk compared to the lowest quartile (OR 2.49; 95% CI: 1.75–3.55). Importantly, BMI adjustment strengthened rather than attenuated the association, suggesting the dietary pattern operates through pathways independent of adiposity—potentially including direct effects of heterocyclic amines, polycyclic aromatic hydrocarbons, and advanced glycation end-products generated during high-temperature processing [5].

Cross-Sectional Evidence from Cancer Survivor Populations

The Brazilian National Health Survey analyses by Verde et al. (2025) and Sara Maria et al. provide descriptive evidence on UPF consumption patterns among women with existing cancer diagnoses [6,7]. Among women with ovarian cancer, UPF consumption prevalence was notably elevated in those under 40 years, suggesting that early-life dietary exposures may warrant investigation. While cross-sectional designs cannot establish temporality, these findings demonstrate that UPF consumption is prevalent among ovarian cancer survivors and identify younger women as a potentially vulnerable subgroup requiring targeted prevention efforts [6,7].

Dose-Response Considerations

The UK Biobank analyses employed continuous exposure metrics (per 10% increment), enabling dose-response inference. The linear relationship between increasing UPF consumption and rising ovarian cancer risk supports biological plausibility and suggests that even moderate reductions in UPF intake could yield meaningful risk reduction at population level [2,8]. This dose-response gradient strengthens causal inference beyond simple binary comparisons.

Biological Plausibility

Several mechanisms may explain the observed UPF-ovarian cancer association. First, UPF typically exhibits high glycemic load, promoting insulin resistance and compensatory

hyperinsulinemia, which increases bioavailability of insulin-like growth factor-1 (IGF-1)—a potent mitogen for ovarian epithelial cells [9]. Second, industrial processing generates advanced glycation end-products (AGEs) and lipid peroxidation products that induce oxidative stress and chronic inflammation, creating a pro-carcinogenic microenvironment [10]. Third, food contact materials used in UPF packaging may leach endocrine-disrupting chemicals including bisphenols and phthalates, which can dysregulate ovarian steroidogenesis and promote hormone-dependent carcinogenesis [11]. Fourth, UPF consumption displaces nutrient-dense foods containing fiber, antioxidants, and phytochemicals with established chemopreventive properties [12].

Consistency Across Populations

The positive association between processed/ultra-processed foods and ovarian cancer demonstrates consistency across geographically and culturally diverse populations—including United Kingdom [2,8], China [4], and Australia [5]—supporting generalizability. While dietary patterns and specific UPF types vary across settings, the underlying exposure construct (highly processed, energy-dense, nutrient-poor foods) appears consistently associated with elevated risk.

Confounder Adjustment and Residual Confounding

The UK Biobank analyses adjusted comprehensively for potential confounders including age, ethnicity, education, income, smoking, physical activity, alcohol, total energy intake, BMI, family history, menopausal status, oral contraceptive use, hormone replacement therapy, and parity [2,8]. The persistence of significant associations after extensive multivariable adjustment reduces likelihood that residual confounding fully explains findings. Nonetheless, unmeasured factors including healthcare access, health literacy, and overall lifestyle patterns may partially confound associations.

Implications for Public Health

These findings carry important public health implications. Ovarian cancer's high case-fatality rate and late-stage presentation at diagnosis make primary prevention through modifiable risk factors particularly valuable. Dietary modifications targeting reduced UPF consumption represent feasible population-level interventions that could complement existing prevention strategies focused on reproductive factors and genetic counseling. Given that UPF constitutes 25–

60% of total energy intake in Western populations [5], even modest relative risk reductions could translate to substantial absolute reductions in ovarian cancer burden.

CONCLUSION AND RECOMMENDATIONS

Summary of Findings

This systematic review provides evidence supporting a positive association between ultra-processed food consumption and increased ovarian cancer risk in women. The UK Biobank prospective cohort demonstrated that each 10% increment in dietary UPF contribution was associated with 19% increased ovarian cancer incidence and 30% increased mortality. Supporting evidence from case-control studies showed preserved food consumption associated with 78% increased odds, and "meat and fat" dietary patterns associated with 2.5-fold increased risk. The consistency of findings across diverse populations and study designs, combined with dose-response relationships and biological plausibility, strengthens causal inference.

Conclusions

Ultra-processed food consumption represents a potentially modifiable risk factor for ovarian cancer. Women with high UPF intake—particularly those consuming preserved foods and high-fat processed dietary patterns—demonstrate significantly elevated ovarian cancer risk. These associations persist after adjustment for established risk factors including BMI, reproductive history, and socioeconomic status, suggesting independent effects.

Recommendations

For Clinical Practice: Healthcare providers should counsel women—particularly those with additional risk factors—to minimize ultra-processed food consumption as part of comprehensive ovarian cancer prevention strategies. Dietary counseling should emphasize replacement of UPF with minimally processed whole foods.

For Public Health Policy: Population-level interventions should include front-of-pack labeling identifying ultra-processed products, restrictions on marketing of UPF to vulnerable populations, and fiscal policies (taxation, subsidies) incentivizing consumption of minimally processed foods.

For Future Research: Prospective studies should examine specific UPF subgroups (carbonated beverages, processed meats, ultra-processed dairy) to identify those most strongly associated with ovarian cancer. Mechanistic studies should investigate pathways including insulin-IGF-1 axis, inflammatory mediators, and endocrine disruption. Intervention trials examining whether reduced UPF consumption modifies intermediate biomarkers (inflammatory markers, IGF-1, oxidative stress) would strengthen evidence for causality.

REFERENCES

1. Caitlin P O'Connell, Hyokyoung G. Hong, L. Abar, et al (2025) Ultra-processed food intake and risk of ovarian and endometrial cancer in the NIH-AARP cohort: a prospective cohort analysis: Ultra-processed food, ovarian & endometrial cancer. American Journal of Clinical Nutrition. <https://doi.org/10.1016/j.ajcnut.2025.05.024>
2. K. Chang, M. Gunter, F. Rauber, et al (2023) Ultra-processed food consumption, cancer risk and cancer mortality: a large-scale prospective analysis within the UK Biobank. EClinicalMedicine. <https://doi.org/10.1016/j.eclinm.2023.101840>
3. A. Hodge, J. Bassett, P. Machado (2025) Ultra-processed food intake and cancer risk in the Melbourne Collaborative Cohort Study. Proceedings of the Nutrition Society. <https://doi.org/10.1017/S0029665125000631>
4. Andy H. Lee, Dada Su, Maria Pasalich, C. Binns (2013) Preserved foods associated with increased risk of ovarian cancer. Gynecologic Oncology. <https://doi.org/10.1016/j.ygyno.2013.03.016>
5. F. Kolahdooz, T. Ibiebele, J. C. van der Pols, P. Webb (2009) Dietary patterns and ovarian cancer risk. American Journal of Clinical Nutrition. <https://doi.org/10.3945/ajcn.2008.26575>
6. S. M. M. L. Verde, Italo Wesley Oliveira Aguiar, Soraia Pinheiro Machado, et al (2025) Escore de Consumo de Alimentos Ultraprocessados entre Mulheres Brasileiras Sobreviventes do Câncer: Pesquisa Nacional de Saúde, Brasil, 2019. Revista Brasileira de Cancerologia. <https://doi.org/10.32635/2176-9745.rbc.2025v71n1.4927>

7. Sara Maria, Moreira Lima, Verde. Av, et al Ultra-Processed Food Consumption Score among Brazilian Women Who Survived Cancer: National Health Survey, Brazil, 2019
8. K. Chang, C. Millett, F. Rauber, et al (2022) Ultra-processed food consumption, cancer risk, and cancer mortality: a prospective cohort study of the UK Biobank. The Lancet. [https://doi.org/10.1016/S0140-6736\(22\)02241-3](https://doi.org/10.1016/S0140-6736(22)02241-3)
9. M. J. Cobo, Z. Vázquez-Ruiz, I. Huybrechts, et al (2025) Degree of food processing and incidence of obesity-related cancers in the 'Seguimiento Universidad de Navarra' (SUN) Project. Journal of NutriLife. <https://doi.org/10.1016/j.tjnut.2025.08.031>