

From Plate to Reproductive Fate: How Ultra-Processed Foods Affect the Fertility of Men and Women

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Abstract: Over the past few decades, the human diet has undergone a transformation with few precedents in history: whole foods have been steadily replaced by industrially formulated products designed for shelf life, palatability and profit rather than nutritional quality. This shift toward increased consumption of ultra-processed foods (UPFs) over staples has coincided with a well-documented and concerning decline in fertility indicators across many parts of the world from falling sperm counts to increasing rates of unexplained infertility. This review brings together evidence from cross-sectional surveys, prospective cohort studies, a randomized controlled feeding trial and rodent models to address a simple question: does what we eat, after it has passed through industrial processing, influence our chances of becoming parents. The evidence while still incomplete, increasingly indicates that it does through pathways involving endocrine-disrupting packaging chemicals altered gonadal signalling, obesity-related hormonal imbalance, gut microbiome disruption and oxidative damage to gametes. The study also examined the less studied question of how parental consumption of ultra-processed foods before conception may influence the health of the next generation and we consider the dietary and lifestyle factors that may help to mitigate these effects. By integrating evidence from human and experimental studies, this review highlights current knowledge, identifies existing research gaps and discusses future directions for understanding the relationship between ultra-processed food consumption and reproductive health.

Keywords: Ultra-Processed Foods, Fertility, Reproductive Health, Endocrine Disruption, Oxidative Stress, Gut Microbiota, Preconception Nutrition.

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I. INTRODUCTION

Infertility is not a rare misfortune. It affects roughly one in six couples of reproductive age around the world and in the United States alone, it affects a comparable proportion of women trying to conceive (Thoma *et al.*, 2013; Carson & Kallen, 2021). For men, the burden is just as real: global estimates put male-factor infertility at somewhere between 9 and 15% of men worldwide and its prevalence has been climbing rather than falling since 1990 (Huang *et al.*, 2023). At the same time, another concerning trend has emerged: a decades-long decline in semen quality, with meta-analyses estimating that sperm counts among men in North America, Europe, Australia and New Zealand have fallen by more than half since the 1970s and the decline shows no sign of levelling off (Levine *et al.*, 2017, 2023). It would be too simple to attribute either trend to a single dietary factor. Infertility arises from multiple causes, including genetic and anatomical

abnormalities, environmental toxicant exposure and the growing prevalence of obesity. However, one notable dietary change has occurred during the same period in which fertility indicators have declined: the increasing consumption of ultra-processed foods (UPFs). In several high-income countries, these products now supply more than half of all calories consumed in a day (Juul *et al.*, 2022) and their consumption is increasing rapidly in middle and lower-income countries. Because UPFs combine several factors that may adversely affect reproductive health, including poor nutrient quality, packaging-derived chemical contamination and a food matrix that promotes overeating and weight gain, they have become an important area of investigation in reproductive research. This review synthesises current evidence from human and animal studies on the relationship between UPFs intake and fertility in both sexes. In addition to summarising current evidence, this review considers the biological mechanisms that may explain these associations, examines the potential

role of parental preconception diet in shaping offspring health and identifies important gaps and inconsistencies in the existing literature.

➤ *Defining the Modern diet: What Makes a Food "Ultra-Processed"*

The NOVA classification system, developed by Carlos Monteiro and colleagues at the University of São Paulo, is the most widely accepted framework for categorising foods according to the degree and purpose of industrial processing rather than their nutrient composition alone (Monteiro *et al.*, 2013, 2019). Under this system, foods are divided into four distinct categories. Group 1 includes unprocessed or minimally processed foods, such as fresh fruits and vegetables, grains, milk, meat and eggs. Group 2 consists of processed culinary ingredients, including oils, butter, sugar and salt, which are typically used in food preparation rather than consumed independently. Group 3 comprises processed foods produced by combining minimally processed foods with culinary ingredients through relatively simple preservation techniques such as canning, fermentation, or baking. Group 4, referred to as ultra-processed foods (UPFs), includes industrial formulations manufactured predominantly from refined food substances together with multiple additives intended to improve flavour, texture, appearance, shelf life and convenience. Colourings, flavour enhancers, emulsifiers, artificial sweeteners and stabilisers are common constituents of these products. Typical examples include soft drinks, packaged snack foods, reconstituted meat products, instant noodles and many commercially produced breakfast cereals.

Ultra-processed foods have become a dominant component of contemporary dietary patterns worldwide. National dietary surveys indicate that they contribute approximately half or more of total daily energy intake among adults in countries such as the United States, the United Kingdom and Canada, while substantial, although comparatively lower, consumption has also been reported in Mediterranean populations, including those in Italy and Spain (Juul *et al.*, 2022). Reflecting this global dietary transition, a three-part *Lancet* series published in 2025 recognised the increasing dominance of UPFs as a major public health concern, emphasising their displacement of traditional diets centred on fresh and minimally processed foods and calling for policy measures that extend beyond individual behaviour change (Monteiro *et al.*, 2025; Baker *et al.*, 2025). Although the initial focus of this discussion was largely directed towards metabolic and chronic diseases, increasing attention has recently been given to the possible implications of UPF consumption for reproductive health. As a result, understanding how these foods may influence fertility has become an emerging area of research.

II. UPFS AND MALE FERTILITY: FROM CORRELATION TO CAUSATION

➤ *What Observational Studies Show*

Earlier studies on diet and male fertility mainly focused on the effects of individual nutrients, such as antioxidants, saturated fats and trans fats. In recent years, researchers have increasingly examined overall dietary patterns because they

appear to have a greater influence on semen quality than individual nutrients alone. Within this context, the possible role of ultra-processed foods (UPFs) in male reproductive health has received growing attention. Several observational studies have reported an association between higher UPF intake and poorer semen quality. In Denmark, Nassan and colleagues observed that young men who consumed larger amounts of sugar-sweetened soft drinks, a common type of UPF, had lower sperm concentration and poorer testicular function (Nassan *et al.*, 2021). Similar findings were reported in a hospital-based case-control study from China, where men with the highest UPF consumption had a significantly greater risk of asthenozoospermia, a condition characterised by reduced sperm motility (Lv *et al.*, 2022). Likewise, the Spanish Led-Fertyl study found that greater intake of UPFs was associated with lower sperm concentration and reduced total sperm count among men attending a fertility clinic (Valle-Hita *et al.*, 2024).

Results from an Italian study also support this observation (Ceretti *et al.*, 2024). Ceretti and colleagues followed healthy, normal-weight young men participating in the FAST randomized trial and evaluated their UPF intake together with semen characteristics over an eight-month period. Men who consumed more UPFs showed lower sperm concentration and reduced sperm motility, even though the study population was free from obesity, chronic illness and other major confounding factors by design (Ceretti *et al.*, 2024). These findings suggest that the association between UPF intake and poorer semen quality cannot be explained entirely by obesity or an unhealthy lifestyle which may be related to the effects of ultra-processed foods themselves.

➤ *The First Randomized Evidence*

Although observational studies provide valuable information, they cannot confirm a cause-and-effect relationship between ultra-processed food consumption and male reproductive health outcomes because dietary habits are often influenced by other lifestyle and behavioural factors. A major step forward came from the randomized crossover feeding trial published in *Cell Metabolism*. In this study, Preston and colleagues assigned healthy men of reproductive age to diets based predominantly on either ultra-processed foods or minimally processed foods under both calorie-balanced and calorie-excess conditions. This design enabled the researchers to separate the effects of food processing from those of excess calorie intake (Preston *et al.*, 2025).

The study showed that several metabolic and reproductive changes occurred even when calorie intake was similar between the two diets. Men consuming the UPF-rich diet gained more body fat, showed changes in cholesterol and thyroid hormone levels and had lower follicle-stimulating hormone concentrations during the excess-calorie phase. A reduction in testosterone together with lower total sperm motility was also observed. In addition, blood and semen samples contained higher concentrations of a phthalate metabolite after consumption of the UPF-rich diet. Phthalates are well known endocrine disrupting chemicals that can interfere with reproductive hormone signalling (Diamanti-Kandarakis *et al.*, 2009; Buckley *et al.*, 2019). As a controlled

randomized feeding trial, this study provides the strongest evidence that the degree of food processing, rather than calorie intake alone may have a negative effect on male reproductive health.

➤ *Plausible Mechanisms*

Several biological mechanisms may explain the relationship between UPF consumption and impaired male reproductive function and these pathways are likely to act together rather than independently. One possible mechanism might be due to exposure to chemicals released from food packaging and processing materials. Phthalates and related plasticisers can migrate into foods during processing and storage due to higher consumption of UPFs has consistently been associated with increased urinary concentrations of these compounds in population-based studies (Buckley *et al.*, 2019). Since phthalates and similar chemicals can act as endocrine disruptors, chronic low-level dietary exposure represents a mechanistically plausible route to disturbed steroidogenesis and impaired sperm production. Another pathway involves metabolism and body composition. UPFs are energy-dense, low in fibre and formulated to encourage overconsumption. The clearest causal evidence comes from an NIH inpatient randomized crossover trial in which adults consuming an ultra-processed diet matched for presented calories, sugar, fat and fibre nevertheless consumed roughly 500 additional calories per day and gained more weight than those receiving an unprocessed diet, establishing UPFs as a direct driver of excess energy intake rather than merely a marker of an unhealthy lifestyle (Hall *et al.*, 2019). Excess adiposity, in turn, disrupts the hypothalamic-pituitary-gonadal axis, promotes insulin resistance and drives chronic low-grade inflammation, all of which impair testicular function, lower testosterone levels and reduce sperm motility and concentration (Ferramosca & Zara, 2022). Insulin resistance also appears to impair glucose uptake in the testis, interfering with the energy-generating pathways of glycolysis and oxidative phosphorylation that spermatozoa rely on for motility (Ferramosca & Zara, 2022).

Leptin, released in excess from expanded adipose tissue when adds a further layer of harm by promoting oxidative stress within the testicular microenvironment. A related pathway is oxidative stress arising independently of body weight. Refined sugars, industrial trans fats and additives commonly found in UPFs are inherently pro-oxidant and pro-inflammatory and spermatozoa are particularly vulnerable to oxidative damage because their plasma membranes are rich in polyunsaturated fatty acids while their cytoplasm contains limited antioxidant defences. Diets rich in polyunsaturated fats and antioxidant micronutrients, by contrast, appear to support healthier mitochondrial function in sperm (Ferramosca & Zara, 2022), a protective environment that ultra-processed diets, which are generally low in these nutrients, fail to provide.

➤ *Vegetarian Diets: An Instructive Comparison*

Comparing vegetarian dietary patterns with other healthy diets provides additional insight into whether the adverse effects on fertility are related to food processing rather than simply the exclusion of meat. Vegetarian diets

share several features with the Mediterranean diet, particularly their high intake of fruits, vegetables and other plant-based foods. However, the evidence regarding their effects on male reproductive health remains less consistent.

Vegetarian diets are generally rich in antioxidant-containing fruits and vegetables, which are known to support sperm quality by reducing oxidative stress. Despite these potential benefits, several studies have reported lower sperm concentration and reduced sperm motility among vegetarian men compared with those consuming mixed diets. This difference has been attributed not to the absence of meat itself, but to a greater intake of plant-derived compounds such as soy isoflavones and other phytoestrogens which can exert weak oestrogen-like activity in the body (West *et al.*, 2005). Further evidence comes from a Spanish cohort study that compared Mediterranean and Western dietary patterns among healthy young men. The study reported that greater adherence to the Mediterranean diet was associated with better indicators of testicular function, suggesting that overall dietary quality is more important than simply avoiding animal-based foods (Cutillas-Tolín *et al.*, 2015). Taken together, these findings indicate that the relationship between diet and male fertility is more complex than classifying foods as simply "good" or "bad." Instead, reproductive health appears to be influenced by the overall quality of the diet, the degree of food processing and the balance between protective nutrients and factors that promote hormonal imbalance, inflammation and oxidative stress.

III. UPFS AND FEMALE FERTILITY: AN EMERGING AREA OF RESEARCH

➤ *Evidence From Human Studies*

Compared with male fertility, the relationship between UPF consumption and female reproductive health has received much less attention and the available evidence remains limited and somewhat inconsistent. One of the largest investigation conducted using data from the US National Health and Nutrition Examination Survey (NHANES). After applying propensity score matching to minimise the influence of potential confounding factors, Su and colleagues analysed data from more than 3,600 women and found that participants in the highest quartile of UPF consumption had significantly greater odds of self-reported infertility than those in the lowest quartile of intake. The association became more pronounced once UPF intake exceeded approximately 40 per cent of total daily food intake (Su *et al.*, 2025). A separate NHANES based analysis by Baric and colleagues similarly reported that women who experienced difficulty in conceiving had higher UPF intake and lower adherence to a Mediterranean diet than fertile women. Although the protective association with the Mediterranean dietary pattern was attenuated after adjustment for obesity (Baric *et al.*, 2026).

Not all studies have reached the same conclusion. A separate cross-sectional analysis of more than 3,000 American women, also using NHANES data found no significant association between UPF consumption and self-reported infertility after appropriate adjustment for potential

confounders (Evans *et al.*, 2025). The discrepancy between these findings and those reported by Su *et al.* (2025) and Baric *et al.* (2026) despite the use of overlapping data sources, highlights the limitations of cross-sectional studies based on self-reported infertility and short-term dietary recall. Such studies are susceptible to misclassification, reverse causation and differences in analytical approaches, including the categorisation of UPF intake and the selection of confounding variables. Female fertility is also inherently more difficult to evaluate in a single cross-sectional assessment than male fertility as ovulatory and tubal causes of infertility are not always reflected by readily measurable biomarkers comparable to those obtained from semen analysis.

➤ *Mechanistic Clues from the Ovary*

In the absence of a female equivalent to the male feeding trial described above, most mechanistic insight into the relationship between UPFs and female fertility comes from studies of obesity and high-fat diets which substantially overlap with the metabolic consequences of a UPF-rich diet. Excess body weight in women is strongly associated with ovulatory dysfunction, menstrual irregularities and reduced rates of spontaneous pregnancy, operating partly through hyperandrogenism and disruption of the hypothalamic-pituitary-ovarian axis (Hohos & Skaznik-Wikiel, 2017; Wang *et al.*, 2014). Animal studies further support this relationship. Feeding female Sprague–Dawley rats with a high-fat diet containing 20% lard for 18 weeks accelerated ovarian follicle development and follicle loss, suggesting that dietary composition may directly affect ovarian reserve (Wang *et al.*, 2014). One possible mechanism linking diet, metabolic status and ovarian function is the kisspeptin system which plays a central role in regulating the reproductive axis by controlling the pulsatile secretion of gonadotropin-releasing hormone. Since kisspeptin signalling is closely influenced by metabolic status, obesity can impair its normal function. Furthermore, kisspeptin receptors are expressed in both the ovary and testis, where they contribute to follicular development, oocyte maturation and steroid hormone production (Dudek *et al.*, 2018; Grzęda *et al.*, 2025). As ultra-processed diets are known to promote weight gain and metabolic disturbances, they may interfere with kisspeptin signalling, potentially affecting ovarian function even before obvious menstrual abnormalities become evident.

➤ *A Special Case: Polycystic Ovary Syndrome*

No single condition illustrates the relationship between diet and female fertility more clearly than polycystic ovary syndrome (PCOS), the most common endocrine disorder affecting women of reproductive age and the leading cause of anovulatory infertility (Azziz *et al.*, 2016). PCOS is fundamentally characterised by insulin resistance and hyperandrogenism, making it biologically predisposed to interact with a UPF-rich diet in both directions. Insulin resistance may increase the preference for highly palatable ultra-processed foods which while the high sugar and refined carbohydrate content of these foods further exacerbates insulin resistance, creating a self-reinforcing cycle. A frequency-matched case-control study from Iran found that women with newly diagnosed PCOS had markedly higher odds of the condition if they consumed greater amounts of red

and processed meat, a major UPF category, than women with the lowest intake (Darand *et al.*, 2025). Although this represents only one aspect of the relationship between diet quality and PCOS, it provides a clear example of how an ultra-processed dietary pattern may contribute to one of the most common causes of female infertility.

IV. BEFORE CONCEPTION: PARENTAL DIET AND THE NEXT GENERATION

➤ *Periconceptional Exposure and Early Embryonic Development*

Fertility research has traditionally considered maternal and paternal diet separately from the health outcomes of the resulting pregnancy, but these factors are increasingly recognised as interconnected. A recent analysis from the Dutch Generation R Next cohort examined UPF intake in both members of a couple during the preconception period and early pregnancy and assessed its relationship with fecundability, subfertility and first-trimester embryonic growth (Lin *et al.*, 2026). This study is notable for including both parents rather than focusing only on mothers and for capturing dietary exposure during the narrow period surrounding conception, when the embryo and its supporting structures may be particularly sensitive to environmental influences.

The broader evidence base remains limited and somewhat inconsistent. A large NHANES-based study of American women found no measurable association between UPF intake and subfertility (Evans *et al.*, 2025). However, maternal UPF consumption during pregnancy has been associated with adverse maternal cardiometabolic outcomes in a prospective periconceptional cohort (Yisahak *et al.*, 2022). In addition, systematic reviews and meta-analyses of cohort studies have reported consistent associations between maternal UPF-rich diets and increased risks of gestational diabetes and preeclampsia, although direct effects on fertility remain difficult to establish (Paula *et al.*, 2022; Talebi *et al.*, 2024). Thus, taken together, the available findings indicate biological plausibility, but the relationship between UPF intake and fertility outcomes requires further investigation.

➤ *The Paternal Contribution: An Emerging Frontier*

An increasingly recognised but less explored aspect of this field is the role of paternal diet before conception. The Paternal Origins of Health and Disease (POHaD) hypothesis proposes that a father's lifestyle during the period preceding conception may influence offspring development through epigenetic modifications carried in sperm, particularly changes in DNA methylation, independent of direct maternal effects (Jahan-Mihan *et al.*, 2024). Evidence supporting this concept has been reported in both human and animal studies. The Newborn Epigenetics Study showed that paternal obesity was associated with altered DNA methylation of the IGF2 gene in umbilical cord blood. Since IGF2 is expressed only from the paternal allele and plays an important role in fetal growth, these findings suggest that paternal metabolic health before conception may influence early developmental programming (Soubry *et al.*, 2013).

Further evidence from the TIEGER project, which examined the relationship between paternal dietary habits and sperm epigenetic profiles indicated that men with higher consumption of foods such as chips and pizza, common examples of ultra-processed foods (UPFs), showed distinct methylation patterns at imprinted regions of sperm DNA compared with men consuming healthier diets (Soubry *et al.*, 2021). More recently, a Brazilian study evaluated paternal UPF intake during the antenatal period as an indicator of preconception dietary exposure and investigated its association with newborn body size and adiposity at birth. The study found that greater paternal consumption of UPFs was associated with differences in infant anthropometric measurements, even after accounting for maternal dietary intake (Carvalho *et al.*, 2026). These observations suggest that paternal diet before conception may influence offspring development through mechanisms that extend beyond the direct effects of maternal nutrition.

➤ *Lessons from Animal Models*

Although human studies provide valuable evidence, ethical constraints and the need for prolonged follow-up make it difficult to investigate the long-term effects of parental diet before conception and during pregnancy. Animal models therefore offer an important opportunity to explore the biological mechanisms involved. In one rat study, animals were fed a "cafeteria diet" composed of highly palatable, energy-dense processed foods that closely resemble a typical western dietary pattern. Exposure to this diet before conception and throughout pregnancy and lactation produced marked reproductive changes in the offspring, many of which persisted beyond weaning (Grzęda *et al.*, 2025).

Male offspring born to cafeteria-fed mothers exhibited reduced seminiferous tubule size, fewer sertoli cells, fewer seminiferous tubules containing mature spermatozoa and altered expression of androgen receptor and kisspeptin genes in the testis. Female offspring also showed significant reproductive changes, including altered expression of oestrogen androgen and luteinizing hormone receptors in the ovary together with a reduced number of early-stage ovarian follicles. These findings demonstrate that maternal consumption of a highly processed diet can affect reproductive development in both sexes, although the biological responses differ between male and female offspring. They also highlight potential mechanisms involving kisspeptin signalling and steroid hormone regulation, providing biological support for the emerging evidence from human studies on the influence of parental diet on offspring reproductive health.

➤ *Dietary Patterns and Fertility: Potential Protective Approaches:*

• *Can Healthier Dietary Patterns Offset the Effects of UPFs?*

As evidence linking UPF consumption with poor reproductive outcomes continues to grow, increasing attention has also been given to dietary patterns that may support reproductive health. Among these, the Mediterranean diet has been studied extensively because it emphasises

minimally processed foods, including fruits, vegetables, legumes, whole grains, fish and olive oil. These foods provide a wide range of antioxidants, polyphenols and other bioactive compounds that are thought to protect reproductive function (Trichopoulou *et al.*, 2003). Findings from multiple investigations indicate favourable reproductive outcomes among women who closely follow Mediterranean-style dietary patterns. The "fertility diet" proposed by Chavarro and colleagues' shares many features with the Mediterranean diet, such as a higher intake of plant proteins, monounsaturated fats and low-glycaemic carbohydrates. Women who adhered more closely to this dietary pattern had a lower risk of ovulatory infertility (Chavarro *et al.*, 2007). Similar observations have been made in women attempting natural conception as well as those undergoing fertility treatment, where greater adherence to the Mediterranean diet has been associated with improved reproductive outcomes (Toledo *et al.*, 2011; Karayiannis *et al.*, 2018).

Evidence from studies in men is clearly suggests a possible benefit of Mediterranean-style diets due to their high content of monounsaturated fatty acids, dietary fibre, antioxidants and polyphenols may help preserve sperm quality by reducing oxidative stress and supporting normal cellular energy metabolism (Ferramosca & Zara, 2022). Nevertheless, these findings should be interpreted with caution because dietary habits are often closely linked with other healthy lifestyle behaviours, including maintaining a healthy body weight and regular physical activity. Recent analyses indicated that these factors may partly explain the reported associations (Baric *et al.*, 2026). In addition, studies involving couples undergoing assisted reproductive technologies have produced inconsistent results and systematic reviews continue to emphasise the need for larger, well-designed clinical studies before firm conclusions can be drawn (Kellow *et al.*, 2022). Taken together, the available evidence indicates that dietary patterns based on minimally processed, nutrient-rich foods may contribute to better reproductive health. However, these diets should be regarded as one component of a broader lifestyle approach rather than as a standalone intervention for infertility. The clear contrast between Mediterranean-style diets and diets rich in UPFs further highlights the importance of overall dietary quality in maintaining reproductive function. The studies discussed throughout this review have evaluated the association between UPF consumption and reproductive health across different populations, study designs and fertility outcomes. Although the evidence is stronger for men than for women, the overall findings consistently indicate that greater intake of UPFs is associated with poorer reproductive health. A summary of the principal human studies included in this review is presented in Table 1.

• *An Integrated Framework Linking UPFs, Biological Pathways and Fertility Outcomes*

Current evidence indicates that the effects of ultra-processed food (UPF) consumption on fertility cannot be explained by a single biological mechanism. Instead, several interconnected processes appear to contribute to reproductive dysfunction in both men and women (Figure 1). These include endocrine disruption, metabolic alterations, oxidative

stress, chronic inflammation, exposure to food-related chemicals and changes in the gut microbiome. Emerging evidence also suggests that the influence of dietary habits may extend beyond the individuals consuming these foods with parental nutrition before conception potentially affecting offspring development. Exposure to endocrine disrupting chemicals is considered one of the major mechanisms linking UPFs with impaired reproductive health. Industrial processing, packaging materials and food contact surfaces can introduce compounds such as phthalates into these foods. Once absorbed, these chemicals can interfere with hormone signalling, alter reproductive hormone profiles, impair semen quality and influence normal reproductive development. Other food contaminants may also contribute to this process. For example, mycoestrogens produced during fungal contamination of food products have been reported to interact with oestrogen-related pathways, further affecting hormonal regulation (Buckley *et al.*, 2019; Kinkade *et al.*, 2024; Baker *et al.*, 2024).

Metabolic dysfunction provides another important explanation for the association between UPFs and fertility. These foods are generally energy-dense, low in dietary fibre and have limited satiety value, making excessive energy intake and weight gain more likely. Obesity and related metabolic disturbances can disrupt hypothalamic-pituitary-gonadal axis function, reduce testosterone production and sperm quality in men and contribute to insulin resistance, hyperandrogenism and ovulatory disorders in women (Ferramosca & Zara, 2022; Hohos & Skaznik-Wikiel, 2017). However, findings from controlled feeding studies indicated that the effects of UPFs cannot be attributed to body weight alone. The randomized trial conducted by Preston and colleagues demonstrated changes in metabolic and hormonal markers even when calorie intake was carefully controlled, suggesting that food processing itself may exert direct biological effects (Preston *et al.*, 2025). Oxidative stress and chronic low-grade inflammation are also likely to play an important role. Diets rich in refined carbohydrates and industrial fats but low in antioxidant containing foods can increase oxidative damage, reducing sperm DNA integrity, impairing mitochondrial function and affecting overall gamete quality. Similar inflammatory responses may also interfere with ovarian function and normal reproductive hormone signalling.

Another pathway receiving increasing attention is gut microbiome. Experimental studies have shown that food additives commonly present in UPFs, including emulsifiers such as carboxymethylcellulose and polysorbate-80, can alter gut microbial composition, weaken intestinal barrier function and promote inflammatory responses (Chassaing *et al.*, 2015). Changes in the gut microbiota may subsequently influence reproductive physiology through the gut–reproductive axis by modifying oestrogen metabolism via the estrobolome and affecting metabolic and inflammatory pathways involved in hormone regulation (Ashonibare *et al.*, 2024). Recent studies further suggest that the effects of parental diet may continue into the next generation through epigenetic mechanisms. Altered sperm DNA methylation patterns and changes in reproductive signalling pathways

observed in both human and animal studies indicated that dietary exposures before conception may influence offspring development and future reproductive health (Soubry *et al.*, 2013, 2021; Grzęda *et al.*, 2025). Although this area of research is still developing, it provides a biologically plausible explanation for how preconception nutrition may also have lasting effects beyond the directly exposed individual. Overall, the available evidence supported that the multifactorial model in which several biological pathways act together rather than independently. Endocrine disruption, metabolic dysfunction, oxidative stress, inflammation, gut microbiome alterations and epigenetic modifications are likely to interact in shaping reproductive outcomes. This integrated framework also emphasises that improving overall dietary quality rather than focusing on a single nutrient or food component may offer greater benefits for reproductive health.

V. LIMITATIONS OF THE CURRENT EVIDENCE

Although the available evidence has provided significant insight into our understanding of the relationship between ultra-processed food (UPF) consumption and reproductive health, several important limitations should be considered. Most studies conducted to date are observational and cross-sectional, making it difficult to establish a causal relationship. In addition, many investigations rely on self reported dietary intake and infertility outcomes both of which are prone to measurement errors and reporting bias. Reverse causation is another concern, as individuals experiencing fertility problems may change their dietary habits which can influence the observed associations. The evidence relating to female fertility is particularly inconsistent. Analysis based on data from the National Health and Nutrition Examination Survey (NHANES) have produced conflicting results. While some studies reported a positive association between higher UPF intake and infertility (Su *et al.*, 2025; Baric *et al.*, 2026), another analysis using an overlapping dataset found no significant relationship (Evans *et al.*, 2025). These differences suggest that study design, statistical methods and population characteristics may all influence the reported findings.

The evidence for male reproductive health is generally more consistent, but it is still limited. To date, only one randomized feeding trial has directly examined the effects of UPFs on male reproductive function and the study involved a relatively small number of participants over a short intervention period (Preston *et al.*, 2025). Consequently, it remains uncertain whether the observed changes in reproductive hormones and semen quality persist, progress, or recover following long-term exposure to UPF-rich diets. Research on parental diet before conception and its influence on offspring health is also at an early stage. Current evidence is largely based on a small number of cohort studies and investigations of epigenetic changes, which provide valuable biological insights but require confirmation in larger and more diverse populations (Lin *et al.*, 2026; Carvalho *et al.*, 2026; Soubry *et al.*, 2013, 2021). Likewise, many of the proposed mechanisms underlying female reproductive

effects, including those involving kisspeptin signalling and the gut microbiome have been demonstrated mainly in experimental animal models. Additional human studies are therefore needed to determine their clinical relevance.

➤ Future Research Directions

Future research should prioritise larger prospective cohorts that follow couples from the preconception period through pregnancy and early offspring development, together with additional controlled feeding studies in both men and women. A female equivalent of the male crossover feeding trial conducted by Preston and colleagues would be particularly valuable, as much of the current understanding of female reproductive effects is based on obesity-related studies and experimental models rather than direct dietary intervention trials. Further studies should also examine the effects of UPF exposure across different age groups and follow couples long enough to assess outcomes such as live birth rather than fecundability alone. In the meantime, the broader public health evidence regarding the adverse effects of high UPF consumption provides a reasonable basis for encouraging dietary patterns that emphasise minimally processed foods while fertility-specific evidence continues to develop (Monteiro *et al.*, 2025; Baker *et al.*, 2025).

➤ Clinical Implications

Although current evidence does not establish a direct causal relationship between ultra-processed food consumption and infertility, the consistency of epidemiological findings and emerging mechanistic evidence support considering dietary quality as part of preconception counselling. Reducing reliance on ultra-processed foods and encouraging dietary patterns rich in minimally processed foods may provide broader metabolic and reproductive health benefits. For clinicians involved in reproductive care, dietary assessment may extend beyond nutrient intake to include the degree of food processing. However, dietary modification should be presented as a supportive lifestyle approach rather than a standalone treatment for infertility, as stronger evidence from long-term perspective studies and intervention trials is still required.

VI. CONCLUSION

The increasing consumption of UPFs and the decline in fertility indicators across many industrialised populations have occurred over a period of time and current evidence suggests that dietary patterns may represent one contributing factor among many. In men, observational studies supported by a randomized feeding trial indicated that ultra-processed diets may adversely influence sperm quality and reproductive hormones through pathways involving chemical exposure, obesity-related endocrine disruption and oxidative stress. In women, the available evidence is less consistent, largely because of methodological challenges in evaluating female reproductive outcomes and the limited number of intervention studies. Nevertheless, findings from epidemiological studies, experimental animal models and research involving conditions such as polycystic ovary syndrome (PCOS) support a biologically plausible link between dietary quality and reproductive health. In addition,

recent studies indicated that parental nutrition before conception may influence offspring health through epigenetic and developmental mechanisms. From a clinical perspective, the current evidence does not support the view of avoiding ultra-processed foods alone will ensure successful conception. Fertility is a complex trait influenced by genetic, biological, environmental and lifestyle factors. However, limiting the intake of highly processed foods while adopting a diet rich in minimally processed, nutrient-dense protective foods represent a practical and low-risk strategy that is consistent with current recommendations for improving both metabolic and reproductive health. Adopting practical dietary guidance such as My Plate and the NIN's New Food Guide Pyramid can further support individuals in making healthier food choices and developing balanced, nutrient-dense dietary patterns which may contribute to better overall metabolic and reproductive health.

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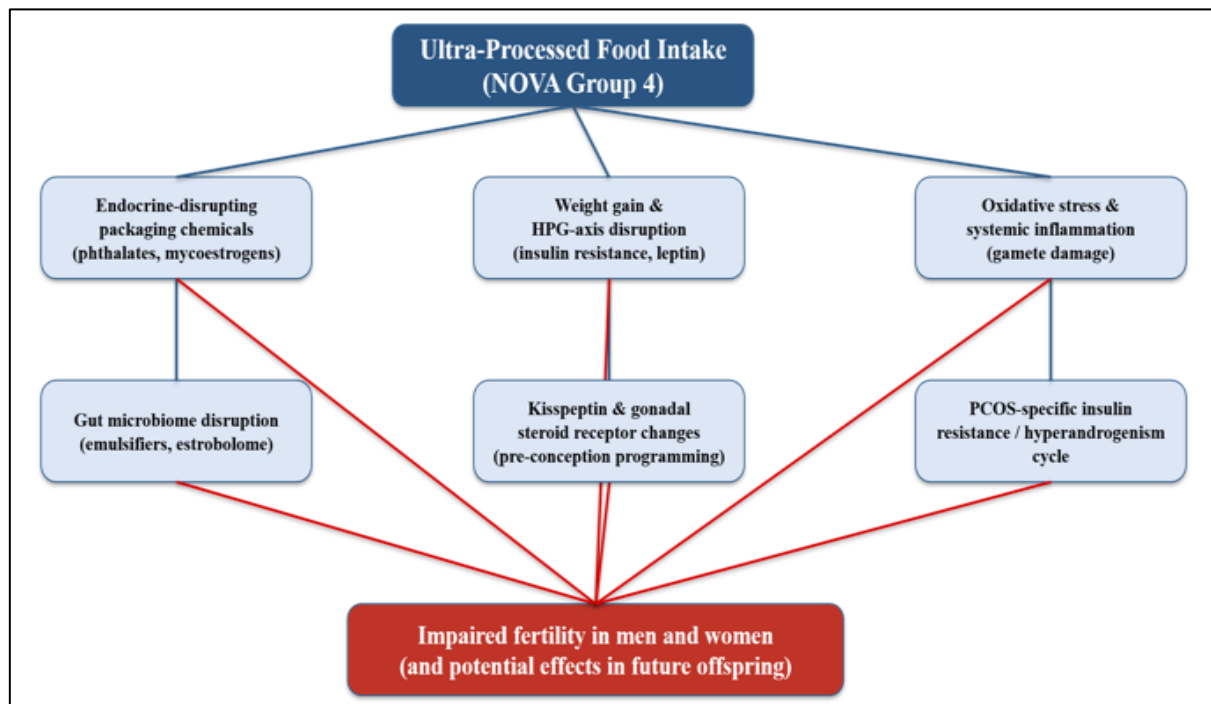


Fig 1 Proposed Overlapping Mechanisms Linking Ultra-Processed Food Intake (NOVA Group 4) to Impaired Fertility in Men and Women, Including Potential Effects on Future Offspring.

Table 1 Summary of Human Studies Evaluating Associations Between Ultra-Processed Food Intake, Dietary Patterns and Reproductive Health Outcomes

Study	Population / Design	Exposure / Comparison	Key Finding	Direction
Nassan <i>et al.</i> , 2021	Danish men, cohort	Soft-drink (UPF) intake vs. testicular function	Lower sperm concentration with higher intake	Adverse
Lv <i>et al.</i> , 2022	Chinese men, case-control	UPF intake vs. asthenozoospermia	Higher odds of asthenozoospermia	Adverse
Valle-Hita <i>et al.</i> , 2024	Spanish men, fertility clinic	UPF intake vs. semen parameters	Lower sperm concentration and count	Adverse
Ceretti <i>et al.</i> , 2024	Italian young men, RCT cohort	UPF intake vs. semen quality	Lower concentration and motility	Adverse
Preston <i>et al.</i> , 2025	Men, randomized crossover trial	UPF vs. unprocessed diet, fixed calories	↓ FSH, ↓ testosterone trend, ↓ motility, ↑ phthalate metabolite	Adverse (causal)
Su <i>et al.</i> , 2025	US women, NHANES cross-sectional	UPF intake vs. self-reported infertility	Higher odds of infertility at high intake	Adverse
Baric <i>et al.</i> , 2026	US women, NHANES cross-sectional	UPF and Mediterranean diet vs. fertility status	Higher UPF / lower MD adherence in infertile women	Adverse / protective
Evans <i>et al.</i> , 2025	US women, NHANES cross-sectional	UPF intake vs. self-reported infertility	No significant association	Null
Lin <i>et al.</i> , 2026	Dutch couples, prospective cohort	Periconceptional UPF (both partners) vs. fecundability, embryonic growth	Associations under investigation; contrasts with null US finding	Mixed
Darand <i>et al.</i> , 2025	Iranian women, case-control	Red/processed meat intake vs. PCOS	Higher odds of PCOS at high intake	Adverse
Kellow <i>et al.</i> , 2022	Meta-analysis, couples undergoing ART	Dietary patterns vs. clinical pregnancy/live birth	Mediterranean pattern linked to live birth; overall evidence inconsistent	Mixed / protective