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REVIEW

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Ultra-processed food and non-communicable diseases in the United Kingdom: A narrative review and thematic synthesis of literature

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Summary

The social and economic constructs of the United Kingdom (UK) provide a fertile food environment for the dramatic expansion in the ultra-processed food (UPF) market, driving increased UPF consumption. This has coincided with the significant increase in the incidence and prevalence of non-communicable diseases (NCDs) such as obesity, type 2 diabetes, cardiovascular disease, and cancer, with an inherent impact on morbidity and mortality. Our review aims to assess the current epidemiological and public health trends in the United Kingdom, specifically examining consumption of UPFs and subsequent development of NCDs, summarizing existing meta-analytical and experimental approaches. First, we address important socioeconomic and psychosocial domains that may contribute to increased availability and consumption of UPF. Additionally, we explore the putative mechanistic basis for the association between UPFs and NCDs: partly attributable to their *energy density*, the *macro- and micronutrient composition* (including high refined carbohydrate, saturated, and trans fats composition, in addition to low fiber and protein content), and *artificially engineered additives and other compounds* that adversely affect health in inadequately researched pathophysiological pathways. This review highlights the importance of promoting minimally processed diets to both clinical and political decision makers.

KEYWORDS
diet quality, non-communicable disease, NOVA, ultra-processed, United Kingdom

1 | INTRODUCTION

The World Health Organisation (WHO) considers non-communicable diseases (NCDs) to be any non-transmissible chronic disease. Historically, preventative strategies have targeted risk factors for five major NCDs: obesity, type 2 diabetes (T2D), cardiovascular disease (CVD), respiratory disorders (namely, asthma and chronic obstructive pulmonary disease [COPD]), and cancer. Mental health disorders are now also included considering their co-occurrence and similar risk factor profiles.[1] NCDs account for ~90% of mortality in the United Kingdom (UK), with lower life-expectancy linked with greater deprivation and lower socioeconomic status.[2]

Despite being considered economically advanced, the United Kingdom has relatively poor social mobility compared to its European neighbors, suggesting wider disparities in health equality between contrasting socioeconomic groups.[3,4] One explanation for this health inequality is the increasingly industrialized global food system, with a shift towards ultra-processed foods (UPFs) instead of home-grown whole foods.[5] UPFs were first categorized by the NOVA classification system, defined as industrial reformulations of food generated through compounds extracted, derived, or synthesized from high yield crops or remnants of intense animal agriculture[6] (Table 1).

Although UPFs are not new, they have persistent broad appeal for many reasons including their interplay between macronutrient composition and artificial additives that enhance palatability, esthetics, shelf life, relative low cost, convenience, and marketability.[8] Alongside the United States, the United Kingdom consumes the most UPFs globally,[9] with school lunches of British children containing almost 80% UPFs,[10] exaggerated further in lower income households.[11] Concurrently, NCD incidence and prevalence is rising in the United Kingdom,[2] widely accepted to be driven by diets characterized by poor nutritional quality and high energy density.[12] Comprehensive meta-analyses have demonstrated associations between greater UPF consumption and increased risk of a wide range of NCDs[13] including obesity,[14] T2D,[15] cancer,[13] CVD,[13] non-alcoholic fatty liver disease (NAFLD),[16] dementia,[17] and all-cause mortality.[18]

Given that poor diet quality is known to increase mortality from chronic disease,[19] it is imperative and timely to review trends in UPF consumption in the United Kingdom and how they are associated with poorer health on a biological, epidemiological, and sociological level, to inform future clinical and political decisions aiming to improve the public health of the United Kingdom.

2 | WHAT ARE UPFS?

The concept of UPFs was first conceived by Carlos Monteiro, a Brazilian physician who identified an apparent paradox between decreasing sugar and oil sales and increasing obesity and T2D prevalence.[6] He determined that the observed reductions in consumption of sugar and oil were misleading, rather these ingredients were being reformulated into pre-prepared, packaged food products. Reformulation of food ingredients arose from an attempt to appropriately feed an increasing global human population. Although initially successful, curbing the malnutrition pandemic in less economically advanced countries, transnational food companies subsequently saw the potential profitability from these products and began increasing their marketability with packaging and advertising campaigns.[20] This marketing is often targeted at children and adolescents in particular, using attention-grabbing and emotionally salient techniques,[21] and has been shown to have robust, detrimental impacts on food choice and consumption in this population[22] as well as wider sociocultural effects on diet in adults.[23] As a result of this commercialization, the malnutrition pandemic has been progressively replaced by an ever-increasing obesity burden.[20]

In 2009, these reformulated products were formally defined as UPFs by Monteiro and his team with the creation of the NOVA classification system.[6] Despite an array of classification systems later becoming available for grouping foods based on their level of processing, NOVA remains the most specific, coherent, comprehensive, and workable tool and, as such, is endorsed by both the United Nations and WHO.[24] NOVA's categorical spectrum includes group 1 (unprocessed/minimally processed) through to group 4 (ultra-processed), with processed culinary ingredients and processed foods making up groups 2 and 3, respectively.[6] A detailed breakdown of these categories is shown in Table 1. Briefly, unprocessed/minimally processed foods include anything from a freshly picked coriander to a carrot or quinoa. Processed culinary ingredients include butter, salt, sugar, oil, and flour that are used to enhance the flavor of foods in group 1. Processed foods include anything that has been preserved, pickled, fermented, or salted, such as canned tomatoes, pickles, or smoked fish. Finally, UPFs in group 4 are typically centered around processed ingredients from group 2 that have undergone an entire reformulation of their food matrix through the addition of artificial colors, emulsifiers, flavorings, and other additives, to enhance palatability. An easy way of identifying UPFs is through inspection of their packaging labels, which highlights ingredients seldom found in domestic kitchens such as soy protein isolate and chemical binding agents.[6]

Policy makers argue that NOVA is too simplistic to dictate dietary guidelines and that UPFs are not necessarily inadequate in macro- and micronutrients.[7] Furthermore, they hail the low cost and high availability of UPFs as benefiting the least socioeconomically privileged by increasing access to food and potentially reducing malnutrition. However, NOVA remains a useful classification tool in research to enable comparable and reproducible evidence surrounding the association between UPFs and NCDs. Recent meta-analyses have been limited by a scarcity of experimental studies using the NOVA classification system to provide an overview of the processing level of an entire diet and consequently have resorted to using multiple methods of defining UPFs, which increases between-study heterogeneity and the risk of bias.[14,15]

3 | SOCIAL DETERMINANTS OF UPF CONSUMPTION IN THE UNITED KINGDOM

The United Kingdom and the United States have the highest percentage of total energy intake originating from UPFs, ~57.8%.[9] This has increased from ~30% in 2000 and ~40% in 2010, with the most commonly consumed UPFs in the United Kingdom being fine bakery wares, sugar sweetened beverages, and sausages.[25] The food environment increases the desirability of UPFs in the United Kingdom through a complex interaction of personal and societal domains including real and perceived economic costs, availability, time constraints, conflicting health information, and cooking traditions.[26]

TABLE 1 NOVA classification of ultra-processed foods.[7]

NOVA	Definition	Examples	Photos
1—Unprocessed or minimally processed foods	Unprocessed foods are obtained directly from plants or animals. There is no alteration following their removal from nature. Minimally processed foods are natural foods that have been submitted to cleaning, removal of inedible or unwanted parts, fractioning, grinding, drying, fermentation, pasteurization, cooling, freezing, or other processes that may subtract part of the food but that do not add oils, fats, sugar, salt, or other substances to the original food.	• Natural, packaged, cut, chilled, or frozen vegetables, fruits, potatoes, and other roots and tubers • Bulk or packaged grains such as brown, white, parboiled, and wholegrain rice, corn kernel, or wheat berry • Grains of wheat, oats, and other cereals • Dried or fresh pasta, couscous, and polenta made from water and the grits/flakes/flours described above • Lentils, chickpeas, beans, and other legumes • Nuts, peanuts, and other seeds without salt or sugar • Fresh and dried herbs and spices (e.g., oregano, pepper, thyme, and cinnamon) • Fresh, chilled, or frozen meat, poultry, fish and seafood, whole or in the form of steaks, fillets, and other cuts • Fresh or pasteurized milk; yoghurt without sugar	  

(Continues)

TABLE 1 (Continued)

NOVA	Definition	Examples	Photos
2—Processed culinary ingredients	Processed culinary ingredients are extracted from natural foods by pressing, grinding, crushing, pulverizing, and refining. They are found domestically and commercially to season and cook food.	• Oils made from seeds, nuts, and fruits, to include soybeans, corn, oil palm, sunflower, or olives • White, brown, and other types of sugar and molasses obtained from cane or beet • Honey and syrup from honeycombs and maple trees • Starches extracted from corn and other plants • Butter, lard, and coconut fat • Refined or coarse salt, mined, or from seawater	
3—Processed foods	Processed foods are manufactured with the addition of salt, sugar, oil, or other NOVA group 2 substances to NOVA group 1 foods to enhance food preserve and taste. They are derived directly from foods and are recognized as versions of the original foods. Most processed foods have two or three ingredients.	• Canned or bottled legumes or vegetables preserved in salt (brine) or vinegar or by pickling • Tomato extract, pastes, or concentrates (with salt and/or sugar) • Fruits in sugar syrup (with or without added antioxidants) • Salted or sugared nuts and seeds • Bacon and beef jerky • Canned fish such as tuna or sardines • Freshly made cheeses • Freshly made (unpackaged) breads made of wheat flour, yeast, water, and salt	

TABLE 1 (Continued)

NOVA	Definition	Examples	Photos
4—Ultra-processed foods	Ultra-processed foods are industrial formulations made from by-products of high yield crops or intense animal agriculture. They are designed to be cheap, palatable, and have long shelf-lives. Manufacturing techniques include extrusion, molding, and pre-processing by frying.	• Fatty, sweet, savory, or salty packaged snacks including biscuits, ice cream, confectionaries such as chocolates and candies, and pastries such as sausage rolls • Sugar sweetened beverages and energy sports drinks and sweetened juices • Canned, packaged, dehydrated (powdered), and other "instant" soups, noodles, sauces, desserts, drink mixes, and seasonings • Sweetened and flavored yoghurts, including fruit yogurts • Margarines and spreads • Pre-prepared (packaged) meat, fish, and vegetables including burgers, hot dogs, and sausages • Pre-prepared poultry and fish "nuggets" and "sticks" • Packaged breads • Breakfast cereals and bars • Meal replacement shakes	

Food environments prove even more difficult to navigate for those from low income households[27]; 72% of the most deprived decile of the UK population were living with overweight or obesity in 2021, compared with 58% of the least deprived.[28] Therefore, perhaps the most important domain contributing towards the upward trend in UPF consumption across England, Scotland, Wales, and Northern Ireland is the higher (both real and perceived) cost associated with adherence to healthier dietary patterns.[29] For the first time in two decades, child poverty rates have risen in absolute terms.[30] In addition, deprived populations are disproportionately exposed to UPF marketing,[31] and promotional strategies often appeal directly to people with budget constraints, for example through discount promotions that create the perception that purchasing UPFs is an easy way to save money.[32] In contrast to the abundance of UPF adverts, similar advertising campaigns for minimally or unprocessed foods are rare.[33] Indeed, the potential health benefits of restricting unhealthy food and beverage advertising on UK television between child viewing hours are clear, with substantially greater benefit for lower socioeconomic groups.[34]

While marketing techniques increase the perceived desirability of UPFs, this is compounded by the physical food environment that is dominated by unhealthy food outlets.[26] Clustering and co-location of fast-food outlets and other harmful commodities in areas of deprivation has been observed.[35] Again, the issues of access to nutritious food are amplified by socioeconomic status, with people living in more deprived areas often having to commute via multiple public transport lines to reach the nearest supermarket, necessitating a taxi journey home due to carrying shopping bags, resulting in an extra expense[36] and further exposure to UPF food marketing in outdoor settings.[37]

Significant research attention has been paid to the detrimental impact of UPFs on health in Latin American nations, namely Brazil, where the NOVA system originated.[9] This has culminated in the Brazilian government advising citizens to avoid UPFs completely.[38] Conversely, the National Health Service (NHS) updated its nutrition guidelines in 2016 to the new "Eatwell guide," replacing the "Eatwell plate" from 2007. The Eatwell guide classifies low-fat margarines and packaged cereals as healthy options, contradicting NOVA's that would list these products in the least healthy group 4.[6,39] In addition, the United Kingdom is now seeking to reduce energy intake by 20% through reformulation and portion-size approaches,[40] and the Nutrient Profile Model underpinning the food advertising legislation (both current and proposed) would also consider some UPF products (e.g., wholemeal bread) as healthy and allow it to continue to be advertised[41]; hence, confusion surrounding what is "healthy" may partly explain increasing UK UPF consumption.[9]

Another contributing factor may be that memories of, and the value attributed to, traditional national cooking methods remain stronger in other countries compared to the United Kingdom. Our extensive relationship with UPFs could mean the current generation associates a childhood dinner with meals such as spaghetti hoops on toast rather than a whole food healthy alternative such as stews and curries containing whole vegetables, legumes, and cereals.

4 | ASSOCIATION BETWEEN UPF AND NCD IN THE UNITED KINGDOM

4.1 | Obesity

The substantial growth of ultra-processed products parallels the increase in obesity. Meta-analysis demonstrates UPF consumption increases the risk of having overweight by 2%, and obesity by 26%, with a dose–response relationship.[14] In the United Kingdom, this association has been assessed extensively (Table 2). Between 2008 and 2016, data from 6143 participants in the UK National Diet and Nutrition Survey Rolling Programme, a nationally representative repeat cross-sectional survey of dietary habits and nutrient intakes, highlighted that high (vs. low) UPF consumption increased the risk of obesity by 90% (OR = 1.90 [95% CI 1.39–2.61]) after adjustment for sociodemographic and lifestyle characteristics. Every 10% increase in UPF intake was associated with a further 18% likelihood of obesity (1.18 [1.08–1.28]).[42] These findings are consistent with UK Biobank data, wherein consumption of UPFs was also found to be positively associated with overweight and obesity.[43] In a 2017 study, Monteiro demonstrated a positive relationship between the prevalence of obesity among male adults versus household availability of UPF in 19 European countries between 1991 and 2008; the United Kingdom had the highest prevalence of both obesity and UPF consumption (Figure 1). The regression model used by Monteiro predicted that for every single percentage point increase in total energy intake attributed to UPFs, obesity prevalence would increase by 0.25 percentage points. In other words, a 40% higher total household dietary energy intake coming from UPFs (as occurs in the United Kingdom vs. Portugal) would translate to a 10% greater obesity prevalence (United Kingdom 24.5%, Portugal 15.2% prevalence).[48]

The mechanisms underlying the association of UPFs with weight gain, and thus overweight/obesity, were investigated in a randomized, controlled, cross-over study of 20 weight-stable men and women, mean age 31.2 years and mean body mass index (BMI) 27 kg/m^2. Participants were admitted to a metabolic research facility and randomized to receive either ultra-processed or unprocessed diets for 14 days immediately followed by the alternate diet for 14 days; both diets were matched for calories, energy density, macronutrients, sugar, sodium, and fiber with participants free to consume as much food as desired on each of their respective diets. The UPF diet was associated with a significantly greater daily energy intake (508 ± 106 kcal/day; p = <0.01) than with the unprocessed diet, with increased consumption of both carbohydrate (280 ± 54 kcal/day) and fat (230 ± 53 kcal/day), but similar protein intake. Weight gain was positively correlated with energy intake, with participants gaining 0.9 kg while consuming UPFs and losing 0.9 kg (p = 0.007) consuming the unprocessed diet.[49] A follow on study by the same research group is assessing the impact of UPFs on energy metabolism in adults.[50] Albeit a short-term mechanistic study, the findings from this study support a causal relationship between UPFs and weight gain/overweight/obesity and are consistent with real-world UK data.

TABLE 2 Description of studies conducted in the United Kingdom using NOVA to evaluate ultra-processed food association with various non-communicable disease.[42–47]

ID	Sample	Population (age/sex)	Study design (follow-up)	Adjustment	Outcome
Obesity					
Rauber et al.[42]	National Diet and Nutrition Survey Rolling Programme	6143 males/females, aged 19–96 years	Cross-sectional	Age, sex, ethnicity, region, survey year, and social class occupation, physical activity, smoking, sleep duration and following a diet to lose weight, total energy intake, and consumption of fruits and vegetables	• High (vs. *low*) UPF consumption increased the risk of obesity by 90% (OR = 1.90 [95% CI 1.39–2.61]) • Every 10% increase in UPF intake was associated with a further 18% likelihood of obesity (1.18 [1.08–1.28])
Rauber et al.[43]	UK Biobank	22,659 males/females, mean age 55.9 years	Prospective cohort (5 years)	Age, sex, quintiles of the Index of Multiple Deprivation, level of physical activity (low/moderate/high), current smoking status (smoker/non-smoker), sleep duration (≤ 6 h/day, 7–8 h/day, ≥ 9 h/day), body mass index (BMI), waist circumference	• High (vs. *low*) UPF consumption increased the risk of obesity by 79% (OR = 1.79 [95% CI 1.06–3.03]) • Every 10% increase in UPF intake was associated with a further 6% likelihood of abdominal obesity (1.06 [1.03–1.08])
Diabetes					
McNaughton et al.[44]	Whitehall II study	7339 males and females, aged 35–55 years	Retrospective cohort (11.6 years)	Age, sex, energy misreporting (energy intake-to-energy expenditure ratio), ethnicity, employment grade, health behaviors (smoking, alcohol use, and physical activity), blood pressure, and BMI	• High (vs. *low*) intake of UPF increased the risk of T2D by 51% (HR 1.51 [95% CI 1.10–2.09])
Levy et al.[45]	UK Biobank	21,730 males and females, mean age 55.8 years	Prospective cohort (5.4 years)	Age, sex, ethnicity (White/Black/other), family history of T2D (yes/no), current smoking status (smoker/non-smoker), BMI (kg/m^2), quintiles of the Index of Multiple Deprivation (IMD), and physical activity level (low/moderate/high)	• High (vs. *low*) intake of UPF increased the risk of T2D by 44% (1.44 [1.04–2.02]) • Every 10% increase in UPF intake was associated with a further 12% likelihood of T2D (1.12 [1.04–1.20])
Cardiovascular disease					
Chen et al.[46]	UK Biobank	60,298 males and females, median age 57 years	Prospective cohort (10.9 years)	Quartiles of UPF consumption, age, sex, ethnicity, years of education, smoking status, total energy intake, obesity status, sleep duration, total energy intake, physical activity, protein, total fat, carbohydrates, alcohol, fiber, saturated fat, monounsaturated fat, polyunsaturated fat, trans-fat, hypertension, diabetes, and dyslipidemia	• High (vs. *low*) intake of UPFs increased the risk of CVD by 17% (HR 1.17 [1.09–1.26]), including a 16% increased risk of CHD (1.16 [1.07–1.25]), a 30% increased risk of stroke (1.30 [1.13–1.50]) • High (vs. low) intake of UPFs increased risk of cardiovascular associated deaths by 22% (1.22 [1.09–1.36])

(Continues)

TABLE 2 (Continued)

ID	Sample	Population (age/sex)	Study design (follow-up)	Adjustment	Outcome
Cancer					
Chang et al.[47]	UK Biobank	197,426 males and females, mean age 58 years	Prospective cohort (9.8 years)	Age, sex, ethnicity, smoking status, physical activity, average household income, highest educational attainment, alcohol intake, and additionally stratified by height, family history of cancer, IMD, and geographical region, BMI, and total energy intake	• Every 10% increase in UPF consumption was associated with an increased incidence of overall cancer risk (HR 1.02 [95% CI 1.01–1.04]), ovarian-specific cancer risk (1.19 [1.08–1.30]) • Every 10% increase in UPF consumption increased risk of overall (1.06 [1.03–1.09]), ovarian-specific (1.30 [1.13–1.50]), and breast-specific (1.16 [1.02–1.32]) cancer-related mortality

Abbreviations: CVD, cardiovascular disease; CHD, coronary heart disease; T2D, type 2 diabetes; UPF, ultra-processed food.

4.2 | NAFLD

The prevalence of NAFLD mirrors that of obesity, representing the most prevalent liver disease in the United Kingdom and globally.[51] NAFLD is also increasingly common in people not living with obesity but who have unfavorable body composition with visceral adiposity.[52] To date, there has been a paucity of UK research assessing the association between NOVA-based UPF consumption and NAFLD. Our own meta-analysis demonstrates a dose–response association between UPFs and NAFLD, with high (vs. low) intake of UPFs associated with a 42% increased risk of NAFLD (RR 1.42 [95% CI 1.16–1.75]).[16]

4.3 | T2D

Patients with obesity and NAFLD are susceptible to development of T2D, explained later by the twin cycle hypothesis.[53] T2D prevalence in the United Kingdom rose from 1.4 million to 3.1 million (1996–2010) and is expected to affect almost 5 million people by 2025.[54] Meta-analysis demonstrates high UPF consumption increases the risk of T2D by 31%, with a dose–response relationship[15]; UK-based studies are shown in Table 2. Data from 10,308 men and women (aged 35–55 years) from civil service departments in London were included in the Whitehall II study.[44] After adjusting for sociodemographic and lifestyle variables, the high (vs. low) intake of UPF increased the risk of T2D by 51% (HR 1.51 [95% CI 1.10–2.09]). Similar analysis of 21,730 adults in the UK Biobank highlighted high (vs. low) intake of UPF increased the T2D risk by 44% (1.44 [1.04–2.02]) after adjusting for confounding variables.[45] The European Prospective Investigation into Cancer (EPIC) study analyzed 340,234 individuals from eight European countries, including the United Kingdom, and found a 16% increased risk of T2D with high intake of UPFs; the smaller effect size was explained by other European countries following a more traditional Mediterranean diet on average and therefore skewing the quartiles of UPF intake.[55]

4.4 | CVD

In 2020, CVD affected ~6.4 million people in the United Kingdom.[28] The only UK study to assess the association between UPFs and CVD (UK Biobank) concluded that high (vs. low) intake of UPFs increased the risk of CVD by 17% (HR 1.17 [1.09–1.26]), including a 16% increased risk of coronary heart disease (CHD) (1.16 [1.07–1.25]) and a 30% increased risk of stroke (1.30 [1.13–1.50]) after adjusting for sociodemographic and lifestyle variables (Table 2). The risk of cardiovascular associated deaths also increased by 22% (1.22 [1.09–1.36]) in those who consumed high (vs. low) UPF rich diets.[46] In the NutriNet-Santé French cohort, high (vs. low) UPF intake was associated with increased risk for CHD (HR for absolute 10% increase: 1.13 [1.02–1.24]), CVD (HR for absolute 10% increase: (1.12 [1.05–1.20]), and stroke (HR for absolute 10% increase: 1.11 [1.01–1.21]), even after adjusting for diet quality.[56]

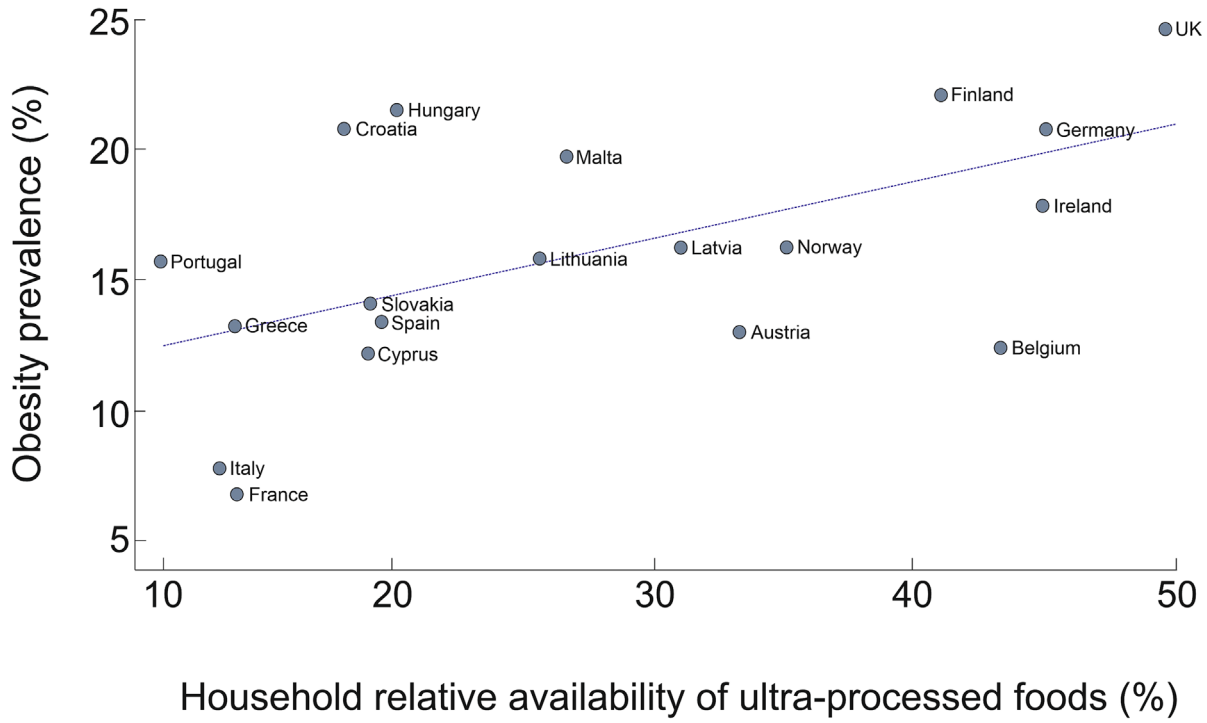

FIGURE 1 An adapted figure redrawn from Monteiro et al. meta-regression analysis of household availability of ultra-processed foods and obesity in 19 European countries, as published in Public Health Nutrition in 2018.[48]

4.5 | Cancer

Currently, 1.8 million UK citizens are living with cancer, largely preventable with lifestyle changes (e.g., reducing smoking and changing diet).[28] A UK Biobank study of 97,426 males and females evaluated the association between UPFs and cancer concluding UPFs may be positively associated with an increased burden and mortality related to overall, and some site-specific, cancers after adjusting for baseline sociodemographic characteristics, smoking status, physical activity, BMI, alcohol, and total energy intake. For every 10% increase in UPF consumption, there was an associated increased incidence of overall (HR 1.02 [95% CI 1.01–1.04]) and ovarian-specific (1.19 [1.08–1.30]) cancer, as well as an associated increased risk of overall (1.06 [1.03–1.09]), ovarian (1.30 [1.13–1.50]), and breast (1.16 [1.02–1.32]) cancer-related mortality.[47] A second study of 450,111 EPIC study participants, also adjusting for sex, smoking, education, physical activity, height, and diabetes, concluded that substituting 10% of NOVA group 4 foods for an equal amount of NOVA group 1 foods would be associated with a 4% reduced risk of overall cancer (HR 0.96 [95% CI 0.95–0.97]), 20% reduced risk of head and neck cancers (0.80 [0.75–0.85]), 43% reduced risk of esophageal squamous cell carcinoma (0·57 [0.51–0.64]), 12% reduced risk of CRC (0.88 [0.85–0.92]), 23% reduced risk of hepatocellular carcinoma (0.77 [0.68–0.87]), and 7% reduced risk of postmenopausal breast cancer (0.93 [0.90–0.97])[57] (Table 2). A cohort of 104,980 French adults in the NutriNet-Santé study concluded that every 10% increase in dietary UPFs was associated with a 12% increase in overall (1.12 [1.06–1.18]), and 11% increase in breast (1.11 [1.02–1.22]), cancer risk after adjustment for comparable confounding variables to the UK Biobank study.[58] Finally, a case–control analysis of 7834 Spanish patients reported an association between UPF consumption and colorectal cancer (CRC), with every 10% increase in UPF consumption being associated with an 11% increased risk of CRC (OR 1.11 [95% CI 1.04–1.18]). However, the study did not find any significant associations between increased UPF consumption and risk of breast or prostate cancer.[59]

4.6 | Respiratory disease

Asthma and COPD remain the most common non-communicable respiratory diseases in the United Kingdom. Asthma affects 5.4 million people in the United Kingdom (1.1 million children and 4.3 million adults), which, despite declining prevalence since early 1990s, is still most prevalent in socioeconomically deprived regions.[28] No experimental study has assessed the association between UPFs and asthma in the United Kingdom, but two cross-sectional Brazilian studies report differing conclusions. Melo et al. concluded consumption of UPFs was associated with asthma and wheezing in adolescents, a 27% increased risk of asthma (OR 1.27 [95% CI 1.15–1.41]) and 42% increased risk of wheezing (1.42 [1.35–1.50]) when comparing the highest versus the lowest quintiles of UPF consumption.[60] Conversely, Machado Azeredo et al. concluded that there was no such association with asthma or wheeze among adolescents.[61] There are no published studies assessing the association between UPFs and COPD in the United Kingdom or globally.

4.7 | Mental health disorder

Approximately 25% of the UK population experience a mental health disorder, with over 15% reporting symptoms of such disorders, including anxiety and depression, each week.[62] Although no studies in the United Kingdom have clearly demonstrated an association between UPFs and mental health disorder, there have been efforts to assess the association in Europe, where several studies have evaluated the impact UPFs have on depressive disorders.[63] One study of 26,730 French participants in the NutriNet-Santé cohort concluded that there was a 21% increased risk of incident depressive symptoms (HR 1.21 [95% CI = 1.15–1.27]) associated with every 10% increase in UPF quantity after adjusting for sociodemographic characteristics, BMI, and lifestyle factors.[64] Similarly, a study of 14,907 Spanish participants from the Seguimiento Universidad de Navarra (SUN) cohort also revealed a positive association between UPFs and depression. Participants in the highest quartile of UPF consumption had a 33% greater risk of developing depression (1.33 [1.07–1.64]) compared with those in the lowest quartile after adjusting for confounders.[65]

4.8 | Dementia

In the United Kingdom, just under 1 million people are living with dementia. This will exceed 1 million by 2030 and 1.5 million by 2050.[66] This is largely attributable to aging populations, although an unpublished meta-analysis from our team demonstrates that high (vs. low) intake of UPFs is associated with a 44% increased risk of dementia (RR 1.44 [95% CI 1.09–1.90]) [Henney et al. 2023 unpublished data]. A single UK Biobank study, using the NOVA classification system, assessed the association between UPFs and dementia in the United Kingdom (Table 2), and a further UK Biobank study assessed the association between processed meats and dementia.[67,68] Data from the 72,083 participants, after adjusting for confounding variables, demonstrated that for every 10% increase in intake of UPFs, there was a 26% increase in all-cause dementia risk (HR 1.25 [95% CI 1.14–1.37]). Specifically, Alzheimer dementia risk increased by 14% (1.14 [1.00–1.30]) and vascular dementia by 28% (1.28 [1.06–1.55]), respectively.[69]

5 | MECHANISTIC EXPLANATIONS FOR UPF ASSOCIATION WITH NCDS

5.1 | Nutritional properties of UPF

5.1.1 | Energy density

The causal relationship between UPF consumption and excess energy intake has been recently highlighted.[49,70] Weight gain promotes NAFLD through visceral adipose tissue (VAT) and ectopic fat (liver and fat) expansion as proposed by the twin cycle hypothesis with consequent insulin resistance and impaired insulin secretion.[53]

The high energy density of UPFs is likely explained by the macro- and micronutrient composition of these foods. When people consume energy dense foods, they fail to adjust overall energy intake and consequently overfeed.[71] Despite the clear association between obesity and NCDs, total energy intake and BMI can confound associations between disease risk and macro- and micronutrient composition of UPFs.[72] Various experimental studies demonstrate that UPFs remain associated with NCDs even independently of energy intake and BMI,[13–15,18] suggesting other components of UPFs must also contribute to disease development.

5.1.2 | Macronutrient composition

UPFs are typically high in total fat (largely from saturated and trans-fat) and refined carbohydrates and low in fiber and protein.[6]

Controlled-feeding studies have demonstrated associations between dietary saturated fatty acids (SFAs) and increased low-density lipoprotein (LDL) cholesterol, known to increase the risk of CHD.[73] In addition, saturated and trans-fats carry high energy density and hence contribute to excess adiposity and increased risk of overweight and obesity. Conversely, UPFs are low in monounsaturated (MUFA) and polyunsaturated fatty acids (PUFAs) that are found in rich supplies in Mediterranean diets.[6] Such diets, high in M/PUFAs, are associated with improved health outcomes including reduced morbidity and mortality associated with CVD.[74]

Refined carbohydrates are independently associated with CVD[75] and accelerated endothelial pathogenesis resulting in vascular dysfunction exacerbated by increased advanced glycation end products (AGEs) that promote oxidative stress.[76] In addition, the rapid gastric emptying of refined carbohydrates alters nutrient flow and their interaction with the gastrointestinal tract endocrine mucosa, which can result in overfeeding through alteration of gut-brain (and adipose tissue-brain) signaling to the arcuate nucleus of the hypothalamus. This cross-talk is mediated via the vagal nerve and the systemic circulation through secretion of such hormones/peptides as cholecystokinin (CCK), glucagon-like peptide-1 (GLP-1), glucagon-like peptide-2 (GLP-2), and gastric inhibitory peptide (GIP).[77] Thus, UPFs may modulate energy intake by altering the peripheral signals that promote satiety/suppress appetite or stimulate hunger. This was illustrated in the study by Hall et al., where consumption of an unprocessed diet was characterized by an increase in the appetite-suppressing hormone, PYY, and a reduction in the hunger hormone ghrelin, as compared with both the ultra-processed diet and baseline. In addition, active GLP-1 was significantly reduced following high UPF consumption.[49] High consumption of refined carbohydrates is also more harmful when consumed later in the day.[78] Moreover, excessive refined carbohydrate intake promotes hepatic de novo lipogenesis, which, if chronic and recurrent, increases intra-hepatic triglyceride deposition,[79] returning

attention to the self-perpetuating twin cycle hypothesis that eventually produces T2D.

UPFs high in refined carbohydrates are typically low in fiber, which acts as the protective outer layer of the original grain. An umbrella review of 18 meta-analyses found that high fiber intake reduced the risk of all-cause mortality, with particular benefit to CVD and gastrointestinal cancer risk. The health benefits of fiber likely stem from a multitude of biological mechanisms including reduced inflammation, improved diversity and function of the gastrointestinal microbiome, and being associated with higher intake of vitamins, minerals, and phytoestrogens.[80]

5.1.3 | Micronutrient composition

UPFs, especially pre-packaged ready meals, are typically high in salt, as well as being deficient in potassium and other minerals such as calcium and magnesium.[6] High dietary salt is associated with elevated blood pressure,[81] thereby increasing CVD risk, including CHD and stroke.[82] Meta-analysis suggests that an additional 5 g of dietary salt daily increases the risk of stroke by 23% and other CVDs by 17%.[83] In addition, salt worsens endothelial dysfunction, through suppression of nitric oxide release.[84] Endothelial dysfunction increases the risk of NCDs including CVD, T2D, cancer, and dementia.[84–86] Higher dietary potassium intake may attenuate the impact of endothelial dysfunction that increases the risk of NCDs in high sodium diets and has therefore been shown to reduce the risk of a similar battery of diseases.[87–89] Other micronutrients such as calcium and magnesium have also been shown to have health benefits.[90]

5.2 | Non-nutritional properties of UPF

5.2.1 | Food matrix and the gut microbiome

In addition to nutritional quality, other unique features of UPFs may contribute to the association with NCDs, including their food matrix (defined as the nutritional and non-nutritional components of food and their molecular interactions). Ultra-processing often involves damaging protective food structures and consequent alterations to the food matrix, which, in turn, have an impact upon nutrient bioavailability, digestion kinetics, glycemic control, and satiety, largely through damaging alterations to the gut microbiota.[13]

The gut microbiota is recognized as a metabolic organ, playing a crucial role in homeostasis in terms of nutrition, immune regulation, and systemic inflammation. Gut dysbiosis is associated with several NCDs.[91] Ultra-processing of foods, such as the refinement of carbohydrates, involves breaking down cellular structures and accelerating nutrient exposure through removal of fiber, as well as reducing dietary polyphenols, which can alter the gut microbiome.[92] Favorable microbial diversity and function promotes cardiometabolic benefits, largely through weight loss and improved insulin sensitivity[93] as a result of short-chain fatty acids and bile flow improving energy balance regulation through stimulation of gastrointestinal L-cells to secrete satiety and incretin hormones.[94] Hence, the deleterious impact of UPFs on the gastrointestinal microbiome causes dysfunction of these pathways and may shift towards favoring overfeeding, hyperglycemia, and the development of chronic diseases.[95]

5.2.2 | Artificial additives

Associations between UPF and health outcomes have been observed independent of the overall poorer nutritional quality of UPF, suggesting that other non-nutritional components also influence UPFs association with NCDs. During the manufacturing process of UPFs, metabolically damaging chemical modification, cosmetic additives, and artificial packaging have potential to worsen health outcomes.[6] Acellular nutrients, pathogen associated molecular patterns (PAMPs), and food additives (e.g., artificial sweeteners and emulsifiers) have been shown to create a selection ground for microbes, which promote inflammation.[95] There is a sparsity of studies in this area, however, with conflicting results and hence further research in this area may provide interesting mechanistic insights.[96] To date, preclinical studies suggest artificial emulsifiers, including carboxymethylcellulose and polysorbate-80, can induce cardiometabolic disturbance through alteration of the gastrointestinal microbiome in rodent models.[97] Similar associations between artificial sweeteners and the gut microbiota have also been demonstrated, implicating metabolic dysfunction; although human data have been collected here.[98] In addition, bisphenol-A is a hormone disruptor commonly used as an artificial component of UPF packaging. Bisphenol-A driven disruption to endocrine signaling has been associated with a multitude of metabolic diseases such as obesity, T2D, and NAFLD.[99–101]

Other possible drivers of NCDs in diets rich in UPFs include carcinogenic compounds produced via intense cooking processes of certain macronutrients. Acrylamide, produced when starchy carbohydrate rich foods, such as potato crisps and breakfast cereals, are cooked at high temperatures, has been associated with CVD risk through hormone disruption.[102] Similarly, acrolein, produced when cooking fats at high temperatures, has been associated with CVD risk.[103]

6 | WHAT CONSTITUTES A DIET LOW IN UPFS?

Several dietary patterns are minimally processed and have been well researched, most notably the Mediterranean diet and the Dietary Approaches to Stop Hypertension (DASH) pattern of eating.

The Mediterranean diet is characterized largely by plant-based eating with daily intake of whole grains, olive oil, fruits, vegetables, beans and other legumes, nuts, herbs, and spices, with smaller quantities of animal proteins, which should be preferably fish and seafood. A Mediterranean diet is characterized by low UPF consumption[104,105] and a low NCD prevalence.[106] The UK diet generally lacks Mediterranean components.[107] An updated review of randomised controlled

trials (RCTs) assessing health outcomes for those following a Mediterranean diet demonstrates efficacy in reduction in the prevalence of obesity/metabolic disease, CVD, and cancer largely through anti-inflammatory and antioxidant effects.[108]

Similarly, the DASH pattern of eating emphasizes intake of fruit, vegetables, whole grains, nuts and legumes, and low-fat dairy while deemphasizing red and processed meat, sugar-sweetened beverages, and sodium salt consumption. DASH is associated with a lower incidence of CVD and a plethora of other cardiometabolic benefits, in people with and without diabetes, according to a recent systematic review and meta-analysis.[109] Even modest adherence to DASH reduces the risk of all-cause and cause-specific mortality.[110] Promisingly, DASH is well accepted following advice within UK populations.[111]

There have been limited attempts to shift away from UPFs and promote consumption of less processed food in robust clinical trial settings. The effectiveness of a minimally processed food-based counseling intervention for weight gain in overweight pregnant women, based on NOVA, was assessed during one RCT in Brazil; 350 overweight, adult pregnant women were randomly assigned to control or intervention groups (which consisted of three counseling sessions that encouraged the consumption of minimally processed foods, following the NOVA food classification system). Significantly, women who received the intervention had a 44% lower chance of developing gestational weight gain that those in the control group (OR 0.56 [95% CI 0.32, 0.98]).[112]

7 | FUTURE DIRECTIONS

The United Kingdom has an urgent need for epidemiological and experimental research to enhance understanding of the impact of food processing and formulation on health. Research should emphasize identification of specific factors comprising UPFs (e.g., energy density, artificial additives, and food matrix alteration) that are causally involved in health outcomes.

Whereas long-term RCT assessing the impact of UPF on hard clinical outcomes is unethical, short-term RCTs on intermediate clinical endpoints are feasible; this was evident from the studies of Hall et al., demonstrating ultra-processed diets lead to increased energy intake and weight gain compared with an unprocessed diet.[49] Utilization of large databases, such as the UK Biobank, may also offer high quality observational evidence.

To date, there is limited human study on additive exposure and long-term health outcomes, particularly when these artificial additives are combined in cocktail as seen with UPFs. Furthermore, food processing encompasses a broad spectrum of techniques, and it may be possible to apply these for the improvement of population (dietary) health if these effects are better understood. For example, the impact of food texture and food matrix integrity on health could have relevance for healthier food innovation.

With better data and improved understanding of mechanism, we would have stronger evidence to enable appropriate implementation of measures at both policy (e.g., prohibition or reduction in safe levels of specific substances or processing methods) and consumer level (e.g., improvement in food labeling), to improve the health of the UK population.

8 | CONCLUSION

The alarming trend in the increasing consumption of UPFs in the United Kingdom is strongly associated with the rising prevalence in NCDs, including obesity, metabolic and CVD, and cancer. This graded association reflects increasing health inequalities across socioeconomic groups in the United Kingdom, representing a major public health challenge for policy makers. It is imperative that policy makers implement measures that favor the availability, affordability, and consumption of minimally processed foods in order to reverse this trend. Promoting optimal physical and mental health enhances quality of life at both an individual and a population level.

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CONFLICT OF INTEREST STATEMENT

None declared.

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