BMC Public Health

RESEARCH

Open Access

Impact of ultra-processed foods consumption on the burden of obesity and type 2 diabetes in Belgium: a comparative risk assessment

Claudia Gutierrez-Ortiz[1*], Leonor Guariguata[2], Claire Dénos[3,4], José L. Peñalvo[1,5] and Stefanie Vandevijvere[6]

Abstract

Background A high consumption of ultra-processed foods (UPFs) has been related to several chronic diseases such as obesity and type 2 diabetes. This study aimed to estimate the proportion of the burden of obesity and type 2 diabetes in Belgium that could be attributed to the consumption of UPFs.

Methods A comparative-risk assessment framework was used. A literature search and a dose-response meta-analysis between UPFs intake and obesity and type 2 diabetes were performed. The risks were extracted from the curve dose-response meta-analysis and extrapolated to the mean of UPFs consumption by strata (5-year age category/sex/region) using the 2014/2015 Belgian Food Consumption Survey. The population attributable fraction and the attributable burden were then calculated for the prevalence of obesity and type 2 diabetes, and disability-adjusted life-years (DALYs) for type 2 diabetes.

Results From the meta-analysis, for every 10% increase in UPFs intake (g/day), there was a 13% increase in risk of developing type 2 diabetes (RR:1.13, 95%IC 1.12-1.15, $p<0.001$) and for every 10% increase of energy intake from UPFs (kcal/day), there was a 5% increase in risk of developing obesity (RR:1.05, 95%IC 0.99-1.13, $p=0.1321$). Using these estimates, 21% of cases (277 056/1 286 454) of obesity in Belgium in 2014/2015 could be attributed to the UPFs intake. Similarly, for type 2 diabetes in Belgium in 2014/2015, 23% of cases (53 348/227 502) and 24% of DALYs (7 998/34 034) were attributed to UPFs consumption.

Conclusions The sizeable impact of UPFs on the burden of obesity and type 2 diabetes in Belgium emphasizes the need to design and implement policies to address UPF consumption in Belgium.

Keywords Comparative risk assessment, Population attributable fraction, Ultra-processed foods, Type 2 diabetes, Obesity

*Correspondence:
Claudia Gutierrez-Ortiz
ceciliagut09@gmail.com
Full list of author information is available at the end of the article

Introduction

The food industry plays a central role in the production, preservation, manufacturing, distribution, marketing, and sale of food products [1]. Initially, food processing aimed to prevent spoilage and enhance food safety. However, over time, it has evolved to prioritize convenience, palatability, and affordability for consumers [2]. To better understand the impact of food processing on health, the NOVA classification was developed, categorizing foods based on their degree of processing rather than solely on nutrient composition or quality [1]. This system classifies foods into four groups: (1) unprocessed or minimally processed foods, (2) processed culinary ingredients, (3) processed foods, and (4) ultra-processed foods (UPFs) [3]. The NOVA classification has become widely used in research investigating the links between food processing and health outcomes [4].

UPFs, which include ready-to-eat meals, sugar-sweetened beverages, sweet biscuits, and artificially sweetened products, constitute an increasing proportion of the global food supply [3]. These products are typically high in saturated fats, added sugars, and salt while being low in dietary fiber. Additionally, they are often energy-dense, nutritionally unbalanced, and lack essential micronutrients [1]. Numerous studies have demonstrated that a high intake of UPFs is associated with an increased risk of cardiometabolic diseases, including dyslipidemia, hypertension, obesity, and type 2 diabetes mellitus (T2DM), as well as cardiovascular and cerebrovascular diseases [5]. UPF consumption has also been linked to cancer, mental health disorders, and higher overall mortality [5, 6]. The mechanisms underlying these associations include the adverse nutritional profile of UPFs, the presence of food additives, emulsifiers, and artificial sweeteners, as well as potential metabolic and inflammatory effects [7].

Despite the accumulating evidence on the negative health impacts of UPFs, their consumption remains high worldwide. In low- and middle-income countries (LMICs), UPFs intake is rising rapidly [1]. The range across Europe of UPFs intake is between 12.2% and 40.8% of daily energy intake, with Belgium reporting an average consumption of 28.8% [8]. Another study estimates that 36% of all food and beverages consumed by the Belgian population are classified as UPFs, accounting for approximately 50% of total daily energy intake [9].

Despite the growing concern regarding UPF consumption, its contribution to the burden of disease has not yet been comprehensively estimated in large-scale studies. Quantifying this burden is essential to better understand the magnitude of the public health challenge and to inform the development of targeted policies aimed at reducing UPF consumption. Regulatory measures, such as restricting the marketing of UPFs and limiting their availability and accessibility, could be instrumental in mitigating their impact.

This study aims to quantify the population attributable fraction and attributable burden of UPF consumption in obesity and T2DM among adults in Belgium using a comparative risk assessment. By providing country-specific burden estimates, this research will contribute to the growing body of evidence on UPFs and support public health planning efforts to address their rising consumption.

Methods

This study is based on the Comparative Risk Assessment (CRA) methodology, in which the current level of exposure is compared to a "counterfactual" or theoretical scenario of lower consumption [10, 11]. The CRA provides a practical approach to estimating the burden of disease associated with an exposure in the absence of long-term longitudinal studies [8]. The counterfactual scenario, formally referred to as the theoretical-minimum-risk exposure level (TMREL), serves as the reference against which the current scenario is compared, providing an estimate of the additional risk experienced by the population due to the current exposure [11].

To gather all the necessary inputs for the CRA, several steps were taken. First, a literature review was conducted to assess the association between UPFs consumption and various health outcomes. Second, the dose-response relationship between UPFs —or energy intake from UPFs—and the development of obesity and T2DM was quantified using meta-analyses, as these were the outcomes with sufficient evidence. Third, the proportion of the burden of obesity and T2DM attributable to UPFs consumption was calculated. Finally, the burden of obesity and T2DM due to UPFs consumption in the Belgian population in 2014 was estimated.

Literature search

A literature search was conducted in Medline, ScienceDirect, Web of Science, Scopus, Embase, and Google Scholar for studies published between January 2013 and September 2023. The search terms used were "Ultra-processed food" and "Risk". Studies were initially screened by title and abstract, followed by a full-text evaluation to determine eligibility. First, our review summarized all available evidence on UPFs consumption and chronic diseases. Then, we assessed the evidence and identified diseases with sufficient data to perform a dose-response meta-analysis, considering clinical relevance and the availability of data sources for Belgium (See Supplementary Table 1).

Eligibility criteria

The eligibility criteria followed the PICOS framework explained in Table 1. These criteria were based on the research question: *What is the dose-response relationship between ultra-processed food (UPF) consumption and the risk of chronic diseases (e.g., type 2 diabetes, obesity, cardiovascular disease, and cancer) in adults, based on prospective cohort studies?*

Chronic diseases considered in the literature search were those with available burden estimates, such as disability-adjusted life-years (DALYs) or case counts. Children, adolescents, and pregnant women were excluded. The selection of the diseases included in the meta-analysis was based on the strength of the evidence and their clinical relevance. Of the health outcomes retrieved, only T2DM and obesity were identified as eligible for this study. One clinical trial assessed weight gain and glucose intolerance in healthy participants who followed either a diet high in UPFs or an unprocessed diet for 2 weeks [12]. Participants on the UPFs diet exhibited significant weight gain and notable changes in appetite-related hormones. However, due to the short trial period, glucose tolerance showed no significant differences. Regarding clinical relevance, obesity is a major risk factor for several diseases, such as hypertension, T2DM, cancer, etc. [13]. Therefore, this study focused on the association between UPFs consumption and the risk of obesity (BMI $\geq$ 30 kg/m^2) and T2DM.

Critical appraisal of cohort studies

We performed a risk of bias assessment of the studies included in the dose-response meta-analysis using the Newcastle-Ottawa Scale adapted for cohort studies, which ranges from 0 to 9 points. Studies scoring $\geq$7 were considered of good quality, those scoring between 4 and 6 were classified as fair quality, and those scoring <4 were considered of low quality [14].

Comparative risk assessment model inputs

Exposure-outcome function

A dose-response meta-analysis was conducted using a two-stage random-effects analysis in R version 4.3.2 (*package dosesresmeta).* The analysis involved two stages: first, the regression coefficient for each study was retrieved; second, the total coefficient was calculated by aggregating the weighted averages of the individual study coefficients [15]. All studies reporting hazard ratios (HRs) assumed a proportional hazards model, where the HR was constant over the follow-up period. Linearity analysis was assessed using a restricted maximum likelihood (REML) model, while non-linearity analysis was conducted using a restricted cubic spline with three knots (0.1, 0.5, 0.9) [16].

The measurement of UPFs consumption lacks standardization across studies and is reported in various units, *e.g.* g/day, kcal/day, or serving/day. Therefore, the units for UPFs consumption were determined based on their frequency in the cohort studies: For T2DM, UPFs consumption was calculated by dividing the daily UPFs intake (g/day) by total food intake (g/day). For obesity, UPFs consumption (%) was calculated by dividing the daily energy intake from UPFs (kcal) by total daily energy intake (kcal/day). Our meta-analysis evaluated UPF consumption as a percentage of total food intake per day, expressed in g/day and kcal/day, depending on the units reported in cohort studies.

Ultra processed foods consumption

The 2014 Belgian Food Consumption Survey (FCS), a nationally representative study of 3 146 participants aged 3 to 64 years [17], was used to estimate current UPF intake in Belgium. For this study, only participants aged 20 years and older were included to align with the meta-analysis results [18].

The FCS 2014–2015 protocol and questionnaire are publicly available online [19]. Dietary intake was assessed using GloboDiet, a standardized computerized 24-hour dietary recall program, conducted on two non-consecutive days [18].

Table 1 Eligibility criteria based on PICO framework

Population	Adults with ≥18 years old
Intervention	Ultra-processed food intake
Comparator	Lower UPF intake or reference category
Outcome	Chronic diseases
Others	Study design: prospective cohort studies reporting RR or HR as primary effect measures. Case-control, cross-sectional, and ecological studies were excluded

Abbreviations: RR relative risk, *HR* hazard ratio

Participants were stratified into 54 groups based on 5-year age categories, sex, and region (Flanders, Brussels-Capital, and Wallonia). Food items were classified according to the NOVA system, and the mean UPF consumption for each stratum was weighted using population sampling factors to ensure representativeness at the national level.

Burden estimates by diseases

Type 2 diabetes burden The Belgian National Burden of Disease Study (BeBOD) estimates the number of people living with diabetes and corresponding DALYs stratified by age, sex, and region. The BeBOD methodology is described in detail elsewhere [20].

Briefly, BeBOD defines diabetes mellitus using administrative and electronic medical records, incorporating indicators such as medication use, physician diagnosis, and hospital discharge data [20]. Diabetes cases and related DALYs are classified according to BeBOD guidelines, which define diabetes *as "a group of metabolic diseases characterized by hyperglycemia resulting from defects in insulin secretion, insulin action, or both."* BeBOD also considers the diagnostic codes E10–E14, covering insulin-dependent diabetes, non-insulin-dependent diabetes, malnutrition-related diabetes, other specific types of diabetes mellitus, and unspecified diabetes mellitus.

BeBOD accounts for patients with diabetes who experience disability or premature death due to complications. These include chronic conditions that require daily medication and may cause concern, as well as microvascular complications such as neuropathy and ophthalmopathy. However, macrovascular complications—including coronary artery disease, stroke, and peripheral arterial disease—are classified separately under cardiovascular diseases [20].

Obesity burden Two datasets were used to estimate the burden of obesity: (i) 2014 FCS data, which provided obesity prevalence by sex, region, and 5-year age categories. In the 2014 FCS, weight and height were objectively measured [17]. (ii) STATBEL, the Belgian Statistical Office, which provides population data by region, 5-year age groups, and sex. The available 2014 STATBEL data were multiplied by the obesity prevalence from the 2014 FCS to estimate the total number of obesity cases in Belgium [21].

Comparative-risk assessment model

Population attributable fractions (PAFs) represent the proportion of cases for a given outcome or risk factor that can be attributed to a specific level of exposure in a population [10].

PAFs can be calculated using different formulas, depending on the exposure-outcome relationship under study. In this study, exposure was assessed as average UPF consumption (g/day and kcal/day) by age group, sex, and region. A continuous version of the PAFs equation was applied [10]. The TMREL was defined as the level of UPF consumption associated with no risk, as determined by the density function [10].

Relative risks (RRs) for each exposure level were extracted from the dose-response meta-analysis curve and applied to age/sex/region-specific UPFs consumption levels to calculate strata-specific PAFs. The attributable burden (AB) was obtained by multiplying the PAF by the total number of DALYs or cases. The final aggregate analysis provided results for the entire Belgian population, with the total rate (RT) expressed per 1 000 000 inhabitants.

Policy scenarios

Two theoretical policy scenarios were evaluated for each disease: For T2DM, a reduction of 100 grams in total UPF consumption and a relative reduction of 10% in daily UPF intake.

For obesity, a reduction of 100 kcal in total energy from UPF intake and a relative reduction of 10% in daily UPF intake.

Results

Literature search

A total of 4 023 articles were identified after removing duplicates in the period 2013 −2023 (see Supplementary, Figure 1). From the literature search, 148 studies were evaluated in full text, 55 studies were included in the literature synthesis (see Supplementary, Table 2) and 93 studies were excluded (see Supplementary, Table 3).

The units used to express UPF consumption varied across the literature, with grams per day being commonly employed in T2DM studies and percentages of daily caloric intake in obesity investigations. Due to limitations in data accessibility, unit conversion was not possible. Consequently, for T2DM and obesity, we used percentages of UPF consumption in g/day and kcal/day, respectively. As a result, six studies were excluded: two T2DM studies that assessed total UPF consumption in grams [22, 23] and three obesity studies that reported UPF intake in g/day or serving/day instead of kcal/day [24–26]. Additionally, a study examining comorbid depressive symptoms alongside UPF consumption in T2DM patients was excluded [27] as depressive status has been associated with an increased risk of T2DM [28]. Notably, after requesting additional data from the

authors, one study provided UPF consumption information in the required units for our analysis across three distinct cohorts, allowing for its direct inclusion without the need for unit conversion [29]. Ultimately, three articles with a total of five cohorts on T2DM were included, while two out of four five studies were incorporated into the obesity analysis (Table 2).

In the T2DM meta-analysis, the five primary studies were UK Biobank, Nutrinet Santé, Nurses' Health Study (NHS), Nurses' Health Study II (NHSII), and the Health Professionals Follow-up Study (HPFS). The last three studies were from the US.

Among the total 325 073 participants, 78% (254 191) were women. The follow-up duration was longer in the US cohort studies, ranging from an average of 26 to 32 years, whereas the more recent UK and France cohorts had follow-up durations of 5.4 years and 6 years, respectively. Food intake was assessed using FFQs (n=3) and repeated 24-hour recalls (n=2). T2DM diagnoses were self-reported and confirmed through a medical database (n=1), a validated algorithm (n=1), and a validated questionnaire (n=3).

In contrast, the obesity meta-analysis included two cohort studies: the Brazilian Longitudinal Study of Adult Health (ELSA) from Brazil and UK Biobank from the United Kingdom, with follow-up durations of 3.8 years and 5 years, respectively. There were 34 486 participants, 55% (18 309) of whom were female. Food intake assessment was conducted using FFQs and repeated 24-hour recalls, while obesity diagnoses were based on baseline and follow-up body mass index (BMI) and annual weight measurements.

Dose-response meta-analysis

Type 2 diabetes

Our findings show a statistically significant linear dose-response association between UPF intake and an increased risk of T2DM. Specifically, for every 10% increase in UPF consumption in grams per day (g/day), the risk of T2DM increased by 13% (RR: 1.13, 95% CI: 1.12–1.15, $p < 0.001$). The meta-analysis included five prospective cohort studies, with a total of 325 073 participants. Heterogeneity was assessed using the I^2 statistic, which indicated no evidence of heterogeneity among the included studies ($I^2 = 0\%$, $p = 0.785$). The model fit was evaluated using the coefficient of determination ($R^2 = 0.924$, Adjusted $R^2 = 0.920$) (Figs. 1 and 2). Additionally, a non-linear dose-response relationship was examined using restricted cubic splines, revealing significant evidence of a non-linear association ($p < 0.001$; p for non-linearity < 0.001).

Sensitivity analysis for T2DM Sensitivity analyses were conducted using a leave-one-out approach, excluding one study at a time. The results remained consistent with the primary analysis, with the estimated risk of T2DM ranging from 13.4% to 13.8% for every 10% increase in UPF consumption. Notably, excluding the three US-based cohort studies resulted in a higher estimated risk of 16% per 10% increase in UPF consumption (RR: 1.16, 95% CI: 1.07–1.25, $p < 0.001$).

Obesity

The dose-response meta-analysis for obesity indicated that each 10% increase in UPF consumption in kcal per day was associated with a 5% increased risk of obesity (RR: 1.05, 95% CI: 0.99–1.13, $p = 0.1321$, $I^2 = 85.4\%$, $p = 0.008$) (Figs. 3 and 4).

There was no evidence of a non-linear association ($p = 0.3025$; p for non-linearity = 0.354). Due to the limited number of studies included in the analysis, a sensitivity analysis was not performed.

Critical appraisal with Newcastle-Ottawa

Most studies (4 out of 5) were rated as good quality, with scores ranging from 0.7 to 0.8. In contrast, one study was rated as fair quality, with a score of 5. A detailed assessment can be found in the Supplementary Material, Table 4.

The ultra-processed food consumption among adults in Belgium

A total of 1 155 adult participants were included in the study. Of these, 52% were female, 57% resided in the Flemish region, 35% in the Walloon region, and 8% in the Brussels-Capital region (Table 3).

The distribution of UPF consumption varied depending on the unit of measurement. When expressed as a percentage of total food weight (g/day %), the distribution was asymmetric, with a higher concentration of individuals consuming lower proportions of UPFs (see Supplementary, Figure 2). In contrast, when measured as a percentage of total energy intake (kcal/day %), the distribution appeared more symmetric, suggesting a more even spread across the population (see Supplementary, Figure 3). This difference likely arises because ultra-processed foods tend to be more energy-dense than unprocessed foods [1], meaning that a small proportion of total weight can contribute disproportionately to total energy intake.

Among sex and age categories, males aged 20 to 29 years in the Flemish and Walloon regions had the highest consumption of UPFs compared to other age groups and females. Similarly, females aged 30 to 34 years in the

Table 2 Characteristics of studies included in meta-analysis

Author (year)	Country	Cohort study & follow-up	Characteristics of participants	UPF survey	Diagnoses of DM	Analysis method	RR or HR (95% CI)	Covariates adjusted
Type 2 diabetes mellitus								
Chen *et al.* (2023) [29]	United States	NHS (32 years)	71 871 women aged 30–55 years	Self-administered 130-items semi-quantitative FFQ, every 2–4 years	Self-reports by participants identified through follow-up questionnaires and confirmed by a validated supplementary questionnaire	Cox proportional hazards regression	Quintiles: Q1 (Reference) Q2 HR: 1.06 (95%CI 0.98–1.15) Q3 HR: 1.17 (95%CI 1.08–1.26) Q4 HR: 1.27 (95%CI 1.18–1.37). Q5 HR: 1.36 (95%CI 1.26–1.46) For each 10% HR 1.13 (95% CI 1.11–1.15)	Age, race, family history of diabetes, history of hypercholesterolemia at baseline, history of hypertension at baseline, baseline BMI, smoking status, physical activity, oral contraceptive use, postmenopausal hormone use, physical examination in the past 2 years, neighborhood income, total alcohol consumption, and total energy intake
Chen *et al.* (2023) [29]	United States	NHSII (26 years)	87 918 women aged 25–44 years	Self-administered 130-items semi-quantitative FFQ, every 2–4 years	Self-reports by participants identified through follow-up questionnaires and confirmed by a validated supplementary questionnaire	Cox proportional hazards regression	Quintiles: Q1 (Reference) Q2 HR: 1.23 (95% 1.12–1.34). Q3 HR: 1.33 (95%CI 1.22–1.45) Q4 HR:1.37 (95%CI1.26-1.50) Q5 HR:1.63 (95%CI1.50-1.76 For each 10% HR 1.11 (95%CI 1.09–1.13)	Age, race/ethnicity, family history of diabetes, history of hypercholesterolemia at baseline, history of hypertension at baseline, baseline BMI, smoking status, physical activity, postmenopausal hormone use, oral contraceptive use, physical examination, neighborhood income, total alcohol consumption, and total energy

Table 2 (continued)

Author (year)	Country	Cohort study & follow-up	Characteristics of participants	UPF survey	Diagnoses of DM	Analysis method	RR or HR (95% CI)	Covariates adjusted
Chen *et al.* (2023) [29]	United States	HPFS (30 years)	38 847 men aged 40–75 years	Self-administered 130-items semi-quantitative FFQ, every 2–4 years	Self-reports by participants identified through follow-up questionnaires and confirmed by a validated supplementary questionnaire	Cox proportional hazards regression	Quintiles: Q1 (Reference) Q2 HR:1.08 (95%CI 0.96–1.21). Q3 HR: 1.16 (95%CI 1.04–1.30). Q4 HR: 1.22 (95%CI 1.09–1.37). Q5 HR:1.41 (95%CI 1.26–1.58) For each 10% HR 1.09 (95%CI 1.06, 1.13)	Age, race/ethnicity, family history of diabetes, history of hypercholesterolemia at baseline, history of hypertension at baseline, baseline BMI, smoking status, physical activity, physical examination, neighborhood income, total alcohol consumption, and total energy
Levy *et al.* (2021) [30]	UK	UK Biobank (5.4 years)	21 730 participants aged 40–79 years, 52.9% were women	Web-based, self-administered questionnaire in the previous 24 hours at baseline and within 3 years after	Self-report and nurse-interview data. Derived an algorithm to identify people with or without incident type 2 diabetes	Cox proportional hazards regression	Quartiles: Q1 (Reference) Q2 HR: 0.98 (95%CI 0.68–1.39) Q3 HR: 1.10 (95% CI 0.76–1.55) Q4 HR: 1.44 (95%CI 1.04–2.02) For each 10% HR 1.12 (95%CI 1.04–1.20)	Age, family history of diabetes, sex, ethnicity, Index of Multiple Deprivation, physical activity level, current smoking status,total energy intake, BMI at baseline
Srour *et al.* (2020) [31]	France	Nutrinet Santé (6.0 years)	104 707 participants aged more than 18 years, 79.2% were women	3 non-consecutive validated web-based 24-hour dietary records at baseline and every 6 months, randomly assigned over a 2-week period (2 weekdays and 1 weekend day)	Self-questionnaires linked to medical databases of the SNIIRAM	Cox proportional hazards regression	For each 10% HR 1.15 (95%CI 1.06–1.25)	Age, sex, educational level, baseline BMI, physical activity level, smoking status, alcohol intake, number of 24-hour dietary records, energy intake without alcohol, family history of diabetes, overall nutritional quality of the diet, number of 24-hour dietary records, energy intake, FSAm-NPS DI score, and family history of type 2 diabetes

Table 2 (continued)

Author (year)	Country	Cohort study & follow-up	Characteristics of participants	UPF survey	Diagnoses of DM	Analysis method	RR or HR (95% CI)	Covariates adjusted
Obesity								
Rauber *et al.* (2021) [32]	England, Scotland and Wales	UK Biobank (5.0 years)	22 659 participants aged 40–69 years and 52.1% were women	Web-based 24-hour dietary records at the end of the recruitment between the next two years (4 times).	BMI at baseline and follow-up.	Cox proportional hazards regression	Quartiles: Q1 (Reference) Q2 HR: 1.21 (95%CI 1.00–1.47) Q3 HR: 1.17 (95%CI 0.97–1.42) Q4 HR: 1.62 (95%CI 1.35–1.94). For each 10% HR:1.11 (95%CI 1.07–1.15)	Sex, Index of Multiple Deprivation, physical activity, smoking status, sleep duration, BMI at baseline
Canhada *et al.* (2017) [33]	Brazil	ELSA-Brasil (3.8 years)	11 827 participants aged 35–74 years, and 55% were women	FFQ at baseline. (Evaluate diet in the last 12 months).	The annual weight gain in BMI. At baseline and after a mean 3.8 years	Poisson regression	Quartiles: Q1 (Reference) Q2 RR: 1.12 (95%CI 0.92–1.35) Q3 RR 1.03 (95% CI 0.85–1.26) Q4 RR 1.11 (95%CI 0.91–1.36) For each 15% RR 1.13 (95%CI 1.01–1.26)	Age, sex, race, centre, income, school achievement, smoking, and physical activity

Abbreviations: NHS Nurses' Health Study, *NHSII* Nurses' Health Study II, *HPFS* Health Professional Follow-Up Study, *RR* Relative risk, *HR* Hazard ratio, *FFQ* Food Frequency Questionnaire, *BMI* Body Mass Index, *FSAm-NPS DI score* Nutrient Profiling System of the British Food Standards Agency Dietary Index Score, *ELSA-Brasil* Brazilian Longitudinal Study of Adult Health Cohort

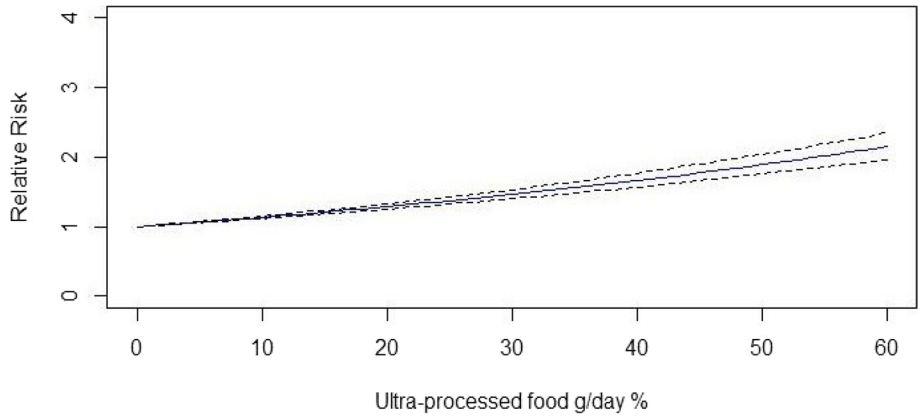

Fig. 1 Curve dose-response meta-analysis of T2DM

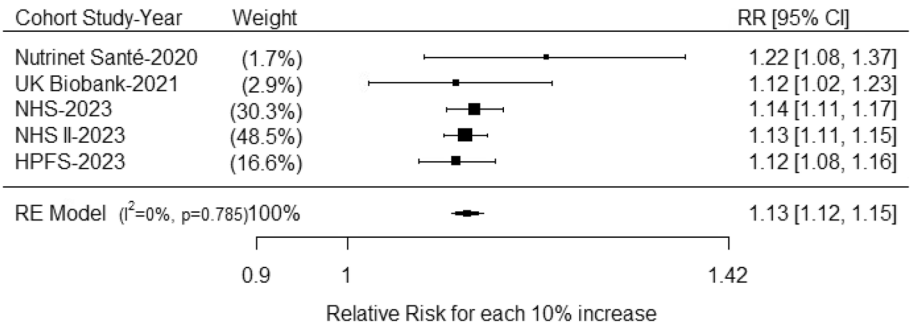

Fig. 2 Forest plots dose-response meta-analysis of T2DM

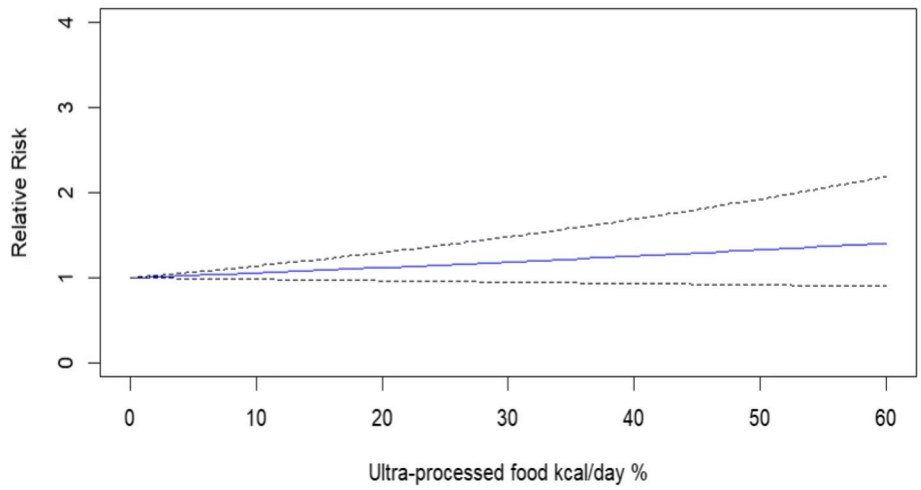

Fig. 3 Curve dose-response meta-analysis of obesity

Walloon region had the highest UPF consumption among women. In contrast, the lowest UPFs was observed in females aged 60–64 years.

Population attributable fraction and attributable burden

We extracted the RR from the dose-response meta-analysis and extrapolated it to the Belgian study population. The dose-response curves (Figs. 1 and 3) indicate that

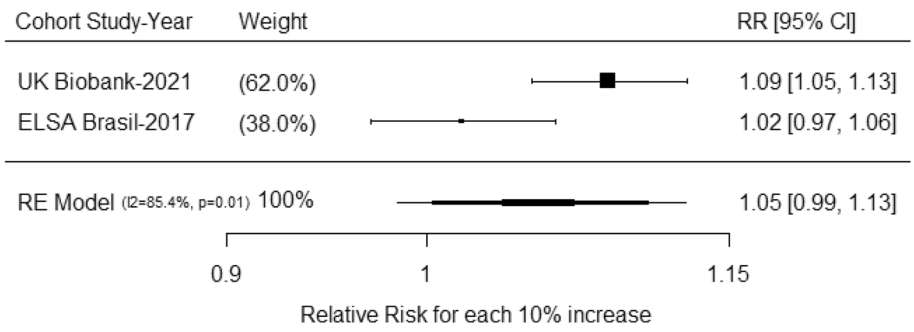

Fig. 4 Forest plots dose-response meta-analysis of obesity

Table 3 Characteristics of food consumption survey 2014

Characteristics	Belgium *N*=1 155	Flanders 660 (57%)	Brussels-Capital 92 (8%)	Wallonia 403 (35%)
Age (years)	43 (32–53)	44 (33–54)	38 (31–52)	43 (32–54)
Male (n)	552 (48%)	317 (48%)	45 (49%)	190 (47%)
Female (n)	603 (52%)	343 (52%)	47 (51%)	213 (53%)
% UPF consumption (g/day)	24.02%	24.66%	19.54%	24.33%
%UPF consumption (kcal/day) percentage by Kcal/day	45.83%	46.78%	40.90%	45.66%

Age is expressed as median (IQR) and %UPF consumption are expressed as mean

zero UPF consumption is the only scenario with no associated risk, meaning that the TMREL is zero.

In our analysis, the highest UPF consumption increased the risk of developing T2DM by 3.5 times compared to individuals with no UPF consumption (RR: 3.57, 95% CI: 3.08–4.14). For obesity, the highest UPF consumption was associated with a 72% increased risk compared to individuals with no UPF consumption (RR: 1.72, 95% CI: 1.16–2.55).

Following the calculation of PAFs and AB by categories, an aggregate analysis yielded results for the entire population. In T2DM, our findings show that UPFs consumption in Belgium was responsible for 23% of cases (53 348 /227 502) and 24% of DALYs (7 998/34 034) of T2DM recorded in 2014. Also, the population rate of T2DM was 8 250 cases per 1 000 000 habitants and 1 236 DALYs per 1 000 000 habitants.

The proportion of T2DM cases and corresponding DALYs attributed to UPFs among regions was higher in Flanders with 25% of cases (26 267/ 106 289) and 25% DALYs (3 893/ 15 690), followed by 23% of cases (22 024/ 97 588) and 23% DALYs (3 381/ 14 891) in Wallonia, and 21% of cases (5 057/23 626) and 21% DALYs (725/3 452) in the Brussels-Capital Region. Between sexes, 26% of cases (31 690/124 130) and 25% of DALYs (5 039/19 813) are attributed to UPFs in males, and 21% of cases (21 658/103 372) and 21% of DALYs (2 959/14 221) are attributed to UPFs in females. Regarding age categories, the group aged 20 to 24 years showed the highest proportion of cases (918/2 866) and DALYs (97/303), with 32% attributed to UPFs consumption for both cases and DALYs. As age increased in both sex groups, the proportion of T2DM attributed to UPFs consumption decreased (Table 4).

On the other hand, 21% of cases (277 056/1 286 454) of obesity in Belgium in 2014 can be attributed to UPFs consumption. Here, the population rate was 4 175 per 1 000 000 inhabitants. Among regions, the proportion of cases attributed to UPFs intake was higher in Flanders with 22% of cases (157 577/720 595) and in Wallonia with 21% of cases (105 831/493 949), followed by the Brussels-Capital Region with 19% of cases (13 648/71 910). Between sexes, 22% of cases (141 289/647 161) are attributed to UPF consumption in males and 21% of cases (135 767/639 293) in females. Regarding age categories, the group aged between 20 to 24 years showed the highest proportion of cases (14 400/56 822) with 25% attributed to UPF consumption (Table 5).

In the two theoretical scenarios (see Supplementary Table 5 and Table 6), a hypothetical reduction of 100 grams across all population strata could reduce 18% of cases and 20% of DALYs of T2DM from the total

Table 4 Population attributable fraction and attributable burden of T2DM with 95%CI in the TMREL scenario

Population	**Total value**	**PAF (95%CI)**	**AB (95%CI)**
All sex, regions, age groups	227 502 cases 34 034 DALYs	23% (21%–26%) 24% (21%–26%)	53 348 (47 880-58 670) 7 998 (7 179 – 8 796)
Region	**Total value**	**PAF (95%CI)**	**AB (95%CI)**
Flanders	106 289 cases 15 690 DALYs	25% (22%–27%) 25% (22%–27%)	26 267(23 583-28 871) 3 893 (3 495-4 278)
Wallonia	97 588 cases 14 891 DALYs	23% (20%–25%) 23% (20%–25%)	22 024 (19 759 – 24 239) 3 381 (3 033-3 720)
Brussels-capital	23 626 cases 3 452 DALYs	21% (19%–24%) 21% (19%–23%)	5 057 (4 538-5 560) 725 (650–797)
Sex	**Total value**	**PAF (95%CI)**	**AB (95%CI)**
Male	124 130 cases 19 813 DALYs	26% (23%–28%) 25% (23%–28%)	31 690 (28 478-34 807) 5 039 (4 528-5 535)
Female	103 372 cases 14 221 DALYS	21% (19%–23%) 21% (19%–23%)	21 658 (19 402-23 863) 2 959 (2 650-3 261)
Age group	**Total value**	**PAF (95%CI)**	**AB (95%CI)**
20–24 years	2 866 cases 303 DALYs	32% (29%–35%) 32% (29%–35%)	918 (828–1003) 97 (88–106)
25–29 years	4 188 cases 516 DALYs	32% (29%–35%) 32% (29%–35%)	1 351 (1 221-1 476) 166 (150–181)
30–34 years	6 998 cases 971 DALYs	30% (27%–33%) 30% (27%–33%)	2 104 (1 896-2 305) 292 (264–320)
35–39 years	8 350 cases 1 173 DALYs	28% (25%–30%) 28% (25%–31%)	2 318 (2 084-2 541) 328 (295–359)
40–44 years	14 061 cases 2 079 DALYs	24% (21%–26%) 24% (22%–26%)	3 343 (3 000–3 677) 498 (447–548)
45–49 years	23 234 cases 3 692 DALYs	26% (23%–28%) 26% (23%–28%)	5 958 (5 360-6 539) 952 (857–1045)
50–54 years	38 823 cases 5 819 DALYs	23% (21%–26%) 24% (21%–26%)	9 107(8 176-10 018) 1 374 (1 234-1 512)
55–59 years	56 759 cases 8 747 DALYs	24% (22%–26%) 24% (22%–26%)	13 627 (12 227-14 973) 2 102(1 886-2 310)
60–64 years	72 225 cases 10 735 DALYs	20% (18%–22%) 20% (18%–22%)	14 623(13 087-16 138) 2 188 (1 958-2 414)

TMREL Theoretical minimum risk exposure level is equal to zero consumption of ultra-processed food, *PAF* Population Attributable Burden, *AB* Attributable Burden, *95%CI* 95% Confidence Interval

estimated burden (44 282 cases and 6 645 DALYs), whereas a hypothetical reduction of 100 kcal across all population strata could reduce 19% of cases of obesity from the total estimated burden (221 349 cases). On the other hand, in the hypothetical scenario with a 10% relative reduction in daily UPF intake, it could avert 14% of cases and 14% of DALYs of T2DM from the estimated burden (31 894 cases and 4 790 DALYs), and 17% of cases of obesity from the estimated burden (245 439 cases) (Figs. 5 and 6).

By subgroup analysis, the policy would benefit similar strata for each disease. For type 2 diabetes and obesity, males aged 20 to 29 years and residents of Flanders would benefit the most (Supplementary Table 5 and Table 6).

Discussion

Our analysis provides a comprehensive estimation of the burden of obesity and T2DM attributable to UPF consumption in Belgium. We found that UPF intake was associated with a 13% increased risk of T2DM for every 10% increase in consumption. The burden estimates suggest 8 250 cases per million inhabitants, corresponding to a proportional burden of 23%, and 1 236 DALYs per million inhabitants, representing 24% of the total burden. For obesity, the meta-analysis indicated a 5% increased risk per 10% increase in UPF consumption, though results were not statistically significant. The estimated burden for obesity was 4 175 cases per million inhabitants, accounting for 21% of total cases in Belgium in 2014.

Table 5 Population attributable fraction and attributable burden of obesity with 95%CI in the TMREL scenario

Population	**Total value**	**PAF (95%CI)**	**AB (95%CI)**
All sex, regions, age	1 286 454 cases	21% (6%–34%)	277 056 (83 270–439 871)
Region	**Total value**	**PAF (95%CI)**	**AB (95%CI)**
Flanders	720 595 cases	22% (6%–34%)	157 577 (47 534-250 546)
Wallonia	493 949 cases	21% (6%–34%)	105 831 (31 718-167 721)
Brussels-capital	71 910 cases	19% (5%–30%)	13 648 (4 018–21 604)
Sex	**Total value**	**PAF (95%CI)**	**AB (95%CI)**
Male	647 161 cases	22% (7%–34%)	141 289 (43 004–223 464)
Female	639 293 cases	21% (6%–34%)	135 767 (40 266-216 407)
Age group	**Total value**	**PAF (95%CI)**	**AB (95%CI)**
20–24 years	56 822 cases	25% (8%–39%)	14 400 (4 431-22 406)
25–29 years	88 329 cases	25% (7%–39%)	22 176 (6 642-34 830)
30–34 years	82 752 cases	23% (7%–36%)	19 540 (5 719-30 368)
35–39 years	128 084 cases	23% (7%–37%)	30 425 (8 929-47 896)
40–44 years	176 520 cases	20% (6%–32%)	34 885 (10 005–56 068)
45–49 years	193 832 cases	22% (7%–34%)	41 991 (13 115-67 005)
50–54 years	155 536 cases	21% (6%–33%)	32 208 (9 809-51 109)
55–59 years	189 319 cases	21% (6%–33%)	39 412 (11 512-62 934)
60–64 years	215 260 cases	20% (6%–31%)	42 019 (13 108-67 255)

TMREL Theoretical minimum risk exposure level is equal to zero consumption of ultra-processed food, *PAF* Population Attributable Burden, *AB* Attributable Burden, *95%CI* 95% Confidence Interval

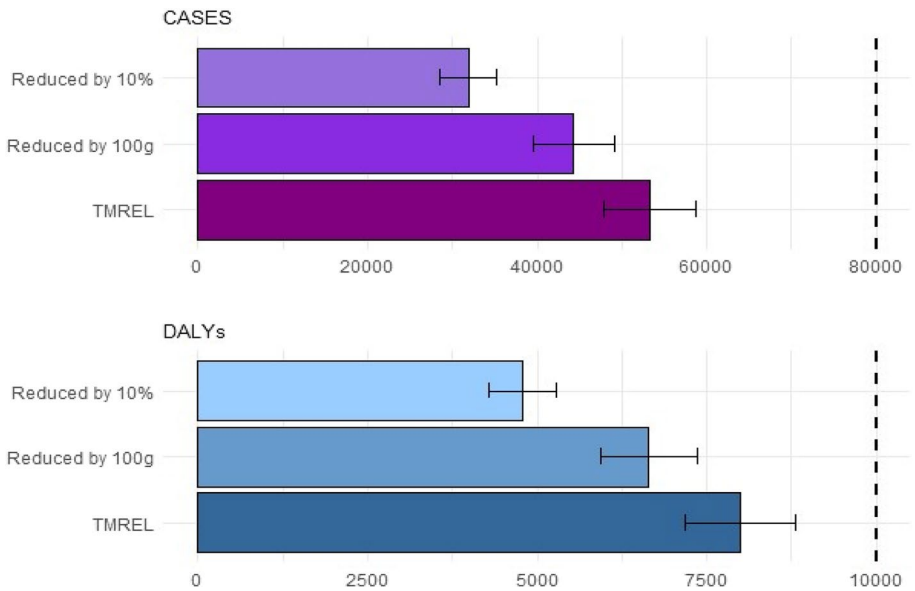

Fig. 5 Estimated number of cases and DALYs of T2DM under different UPF reduction scenarios in Belgium. This figure shows the total number of obesity cases expected under three scenarios: (1) a 10% reduction in UPF consumption (2) a reduction of 100 kcal per day from UPFs, and (3) TMREL (theoretical-minimum-risk exposure level). The differences between these scenarios highlight the potential number of cases and DALYs that could be prevented with lower UPF intake

Our dose-response meta-analysis for T2DM aligns closely with a prior meta-analysis of seven cohort studies, which reported a RR of 1.12 (95% CI: 1.10–1.13) [29], and is somewhat comparable to another meta-analysis that included five studies (four cohort and one cross-sectional), reporting an RR of 1.15 (95% CI: 1.06–1.26) [34]. Three studies from these prior analyses were excluded due to differences in study design (cross-sectional) [35], units of analysis (absolute amount) [22], or analytical approach (logistic regression) [36]. Regarding

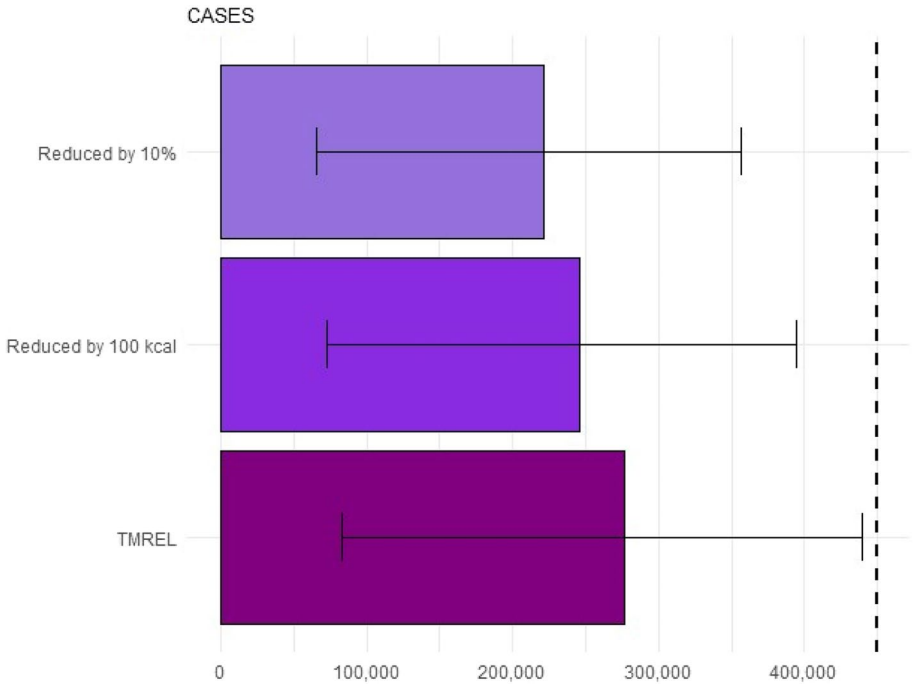

Fig. 6 Estimated number of obesity cases under different UPF reduction scenarios in Belgium. This figure shows the total number of obesity cases expected under three scenarios: (1) a 10% reduction in UPF consumption (2) a reduction of 100 kcal per day from UPFs, and (3) TMREL (theoretical-minimum-risk exposure level). The differences between these scenarios highlight the potential number of cases that could be prevented with lower UPF intake

confounding factors, all selected models were adjusted for age, sex, family history of T2DM, physical activity, baseline BMI, and total calorie intake. Additionally, three out of five included studies had follow-up periods ranging from 26 to 32 years, but information on the time of diagnosis was lacking.

For obesity, the dose-response meta-analysis suggested a 5% increased risk, but results were not significant, and high heterogeneity was observed. This could be due to limited data in our analysis. Moreover, individuals with obesity may underreport calorie intake and exhibit impaired metabolic efficiency [37]. Additionally, the short follow-up periods (3.8 to 5 years) may have been insufficient, as sustained small dietary changes affect body weight over longer durations (e.g., 10 years) [37]. A previous meta-analysis found that a 10% increase in UPF consumption was associated with a 7% increased risk of obesity (HR 1.07, 95% CI: 1.03–1.11), based on five cross-sectional and two cohort studies [38]. These last two cohort studies were included in our meta-analysis showing slight similarity.

This study also explored two theoretical policy impact scenarios—absolute and relative reductions in UPF consumption. Both scenarios demonstrated substantial decreases in burden estimates. However, no specific policies targeting UPF intake currently exist.

Our study is the first to estimate the burden of T2DM attributable to UPF consumption, though prior research has linked UPFs to cardiovascular diseases and obesity [39, 40]. Globally, dietary risk factors contribute to 34.9% of T2DM-related DALYs, with the Global Burden of Disease (GBD) study identifying 11 dietary risk factors. Among these, processed meat consumption significantly contributes to T2DM burden [41]. In Belgium, processed meat was associated with a 36.1% proportional burden and a rate of 1 379 T2DM cases per million inhabitants in 2018. Some processed meats, such as sausages, fall into processed foods or UPFs [42]. Therefore, UPFs should be considered a global dietary risk for T2DM.

Regarding obesity, a study in Brazil attributed 28.6% of the increase in obesity prevalence over seven years to UPFs consumption [40]. Globally, 51.9% of T2DM-related DALYs are attributed to high BMI, the primary risk factor among others, including dietary risks [43]. Given that UPFs partially contribute to obesity, their effect on T2DM should also consider mediation factors for a more accurate risk estimation [10].

Research has assessed the health risks of specific UPF subtypes (e.g., ready-to-eat meals, sweets, desserts, bread, cereals, yogurt) [3, 29]. Adding nutritional information on individual UPFs could enhance the understanding of their health impacts. The NOVA

classification should complement ingredient declarations and nutritional quality scores, as recognizing the extent and purpose of food processing provides better insight into non-essential components.

Existing food policies primarily target specific compounds, such as sugar taxation, front-of-package (FOP) labeling, marketing bans, sodium and trans-fat limits, and school food regulations [44]. Expanding these policies to encompass UPFs would require an expert-led operational definition of UPFs for regulatory purposes [45]. Although current policies have improved consumption patterns, their long-term effects on chronic disease development require further study, as long-term monitoring is essential. The food industry often uses the "lack of effect" argument to oppose such policies [44].

Future studies should include participants with at least 10 years of UPF exposure before a T2DM diagnosis to better assess causality, given that prediabetes—a reversible stage—has an average duration of 10 years [46, 47]. Additionally, further research should assess social determinants (e.g., income, education, food access, security) of UPF consumption, the economic and environmental burden of UPFs, and policy impacts.

The limitations of our study are: First, our study is based on 2014 Food Consumption Survey (FCS) data, and UPF consumption may have increased since then [48]. However, a comparison with the 2004 survey showed no significant changes in UPF intake among individuals aged 15–64 years [49]. Second, while FFQ and 24-hour recall are validated dietary assessment tools [50, 51], they have inherent limitations, including recall bias, social desirability bias, and challenges in assessing long-term dietary intake. These factors should be considered when interpreting dietary data in epidemiological studies [52]. Third, greater access to primary cohort data on obesity and its conversion into comparable units could improve our meta-analysis estimates.

The strengths of our study include a robust dose-response meta-analysis, demonstrating consistency with previous research and supporting the extrapolation of relative risk estimates to UPF consumption levels. We are the first to estimate the contribution of UPF consumption to the burden of obesity and T2DM in Belgium, offering stratified population data based on the Belgian Food Consumption Survey. Our findings underscore the need for UPF-related policies and provide a benchmark for comparison with the upcoming 2024 Food Consumption Survey.

Conclusion

Our findings suggest that higher UPF consumption is associated with an increased risk of obesity and T2DM. In 2014, UPF intake was responsible for 23% of T2DM cases and 24% of related DALYs, as well as 21% of obesity cases.

These burden estimates highlight the urgent need for policy interventions to limit UPF consumption. Despite ongoing debates on causality, the strong associations observed in longitudinal studies support the implementation of recommended public health policies to mitigate the impact of UPFs on chronic diseases.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12889-025-22304-3.

Supplementary Material 1.

Acknowledgements
Not applicable.

Authors' contributions
LG has made a substantial contribution to the conception of the study. All authors participated in the study design, data analysis, and interpretation. They have drafted the manuscript, revised it, and approved the submitted version.

Funding
The authors want to thank the Federal Public Service of Health, Food Chain Safety and Environment to provide funding for the national food consumption surveys of 2004 and 2014/2015.

Data availability
The datasets generated and analyzed during the current study are not publicly available because these data fall under the protection of privacy concerning the processing of personal data. The datasets are available from the authors upon reasonable request and according to certain conditions.

Declarations

Ethics approval and consent to participate
The Belgian Food Consumption Survey 2014-2015 was conducted in accordance with the ethical principles in the Declaration of Helsinki. The study protocol received approval from the Ethical Committee of the University of Ghent. Written informed consent was obtained from all participants prior to their involvement in the survey.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

Author details
[1]Global Health Institute, Faculty of Medicine and Health Sciences, University of Antwerp, Antwerp 2610, Belgium. [2]KU Leuven Access-to-Medicines Research Centre, Vlamingenstraat 83, Leuven 3000, Belgium. [3]Sustainable Systems Engineering (STEN), Department of Green Chemistry and Technology, Ghent University, Coupure Links 653, Gent 9000, Belgium. [4]Socio-environmental dynamic research group (SONYA), Université Libre de Bruxelles, Avenue Antoine Depage 30, Brussels 1050, Belgium. [5]National Center for Epidemiology, Carlos III Health Institute, Avenida Monforte de lemos 5, Madrid 28029, Spain. [6]Department of Epidemiology and Public Health, Scientific Institute of Public Health (Sciensano), J.Wytsmanstraat 14, Brussels 1050, Belgium.

Received: 12 November 2024 Accepted: 12 March 2025
Published online: 22 March 2025

References

1. Monteiro CA, Cannon G, Moubarac JC, Levy RB, Louzada MLC, Jaime PC. The UN Decade of Nutrition, the NOVA food classification and the trouble with ultra-processing. Public Health Nutr. 2018;21(1):5–17.
2. Monteiro CA. Nutrition and health. The issue is not food, nor nutrients, so much as processing. Public Health Nutr. 2009;12(5):729–31.
3. Cordova R, Viallon V, Fontvieille E, Peruchet-Noray L, Jansana A, Wagner KH, et al. Consumption of ultra-processed foods and risk of multimorbidity of cancer and cardiometabolic diseases: a multinational cohort study. Lancet Reg Health Eur. 2023;35:100771.
4. Crino M, Barakat T, Trevena H, Neal B. Systematic review and comparison of classification frameworks describing the degree of food processing. Nutr Food Technol. 2017;3(1):138.
5. Lane MM, Davis JA, Beattie S, Gómez-Donoso C, Loughman A, O'Neil A, et al. Ultraprocessed food and chronic noncommunicable diseases: a systematic review and meta-analysis of 43 observational studies. Obes Rev. 2021;22(3):e13146.
6. Elizabeth L, Machado P, Zinöcker M, Baker P, Lawrence M. Ultra-processed foods and health outcomes: a narrative review. Nutrients. 2020;12(7):1955.
7. Almarshad MI, Algonaiman R, Alharbi HF, Almujaydil MS, Barakat H. Relationship between ultra-processed food consumption and risk of diabetes mellitus: a mini-review. Nutrients. 2022;14(12):2366.
8. Mertens E, Colizzi C, Peñalvo JL. Ultra-processed food consumption in adults across Europe. Eur J Nutr. 2022;61(3):1521–39.
9. Dénos CVS, Boone L, Cooreman-Algoed M, De Bauw M, Achten WMJ, et al. Contribution of ultra-processed food and animal-plant protein intake ratio to the environmental impact of Belgian diets. Sustain Prod Consum. 2024;51:584–98.
10. Plass D, Hilderink H, Lehtomäki H, Øverland S, Eikemo TA, Lai T, et al. Estimating risk factor attributable burden - challenges and potential solutions when using the comparative risk assessment methodology. Arch Public Health. 2022;80(1):148.
11. Ezzati M. Comparative risk assessment. In: International encyclopedia of public health. 2008. p. 806–818.
12. Hall KD, Ayuketah A, Brychta R, Cai H, Cassimatis T, Chen KY, et al. Ultra-processed diets cause excess calorie intake and weight gain: an inpatient randomized controlled trial of ad libitum food intake. Cell Metab. 2020;32(4):690.
13. Dixon JB. The effect of obesity on health outcomes. Mol Cell Endocrinol. 2010;316(2):104–8.
14. Ma LL, Wang YY, Yang ZH, Huang D, Weng H, Zeng XT. Methodological quality (risk of bias) assessment tools for primary and secondary medical studies: what are they and which is better? Mil Med Res. 2020;7(1):7.
15. Shim SR, Lee J. Dose-response meta-analysis: application and practice using the R software. Epidemiol Health. 2019;41:e2019006.
16. Crippa A, Discacciati A, Bottai M, Spiegelman D, Orsini N. One-stage dose-response meta-analysis for aggregated data. Stat Methods Med Res. 2019;28(5):1579–96.
17. Bel S, Van den Abeele S, Lebacq T, Ost C, Brocatus L, Stiévenart C, et al. Protocol of the Belgian food consumption survey 2014: objectives, design and methods. Arch Public Health. 2016;74:20.
18. Park MK, Freisling H, Huseinovic E, Winkvist A, Huybrechts I, Crispim SP, et al. Comparison of meal patterns across five European countries using standardized 24-h recall (GloboDiet) data from the EFCOVAL project. Eur J Nutr. 2018;57(3):1045–57.
19. Sciensano. National Food Consumption Survey: study protocol. Brussels: Sciensano. [Internet]. Available from: https://www.sciensano.be/en/projects/national-food-consumption-survey#study-protocol.
20. De Pauw R, Gorasso V, Scohy A, Van den Borre L, Devleesschauwer B. Belgian National Burden of Disease Study. Guidelines for the calculation of Disability-Adjusted Life Years in Belgium. Brussels, Belgium: Sciensano; 2023. Deposit number: D/2023.14.440/67.
21. Statistics Belgium. Demographic data of Belgium. [Internet]. Available from: https://bestat.statbel.fgov.be/bestat/crosstable.xhtml?datasource=65ee413b-3859-4c6f-a847-09b631766fa7.
22. Llavero-Valero M, Escalada-San Martín J, Martínez-González MA, Basterra-Gortari FJ, de la Fuente-Arrillaga C, Bes-Rastrollo M. Ultra-processed foods and type-2 diabetes risk in the SUN project: a prospective cohort study. Clinical Nutrition. 2021;40(5):2817–24.
23. Canhada SL, Vigo Á, Levy R, Luft VC, da Fonseca MJM, Giatti L, et al. Association between ultra-processed food consumption and the incidence of type 2 diabetes: the ELSA-Brasil cohort. Diabetol Metab Syndr. 2023;15(1):233.
24. Cordova R, Kliemann N, Huybrechts I, Rauber F, Vamos EP, Levy RB, et al. Consumption of ultra-processed foods associated with weight gain and obesity in adults: a multi-national cohort study. Clin Nutr. 2021;40(9):5079–88.
25. Beslay M, Srour B, Méjean C, Allès B, Fiolet T, Debras C, et al. Ultra-processed food intake in association with BMI change and risk of overweight and obesity: a prospective analysis of the French NutriNet-Santé cohort. PLoS Med. 2020;17(8):e1003256.
26. Mendonça RD, Pimenta AM, Gea A, de la Fuente-Arrillaga C, Martinez-Gonzalez MA, Lopes AC, et al. Ultraprocessed food consumption and risk of overweight and obesity: the University of Navarra Follow-Up (SUN) cohort study. Am J Clin Nutr. 2016;104(5):1433–40.
27. Sen A, Brazeau AS, Deschênes S, Ramiro Melgar-Quiñonez H, Schmitz N. The role of ultra-processed food consumption and depression on type 2 diabetes incidence: a prospective community study in Quebec, Canada. Public Health Nutr. 2023;26(11):2294–303.
28. Liu K, Zhou D, Chen L, Hao S. Depression and type 2 diabetes risk: a Mendelian randomization study. Front Endocrinol (Lausanne). 2024;15:1436411.
29. Chen Z, Khandpur N, Desjardins C, Wang L, Monteiro CA, Rossato SL, et al. Ultra-processed food consumption and risk of type 2 diabetes: three large prospective U.S. cohort studies. Diabetes Care. 2023;46(7):1335–44.
30. Levy RB, Rauber F, Chang K, Louzada M, Monteiro CA, Millett C, et al. Ultra-processed food consumption and type 2 diabetes incidence: A prospective cohort study. Clin Nutr. 2021;40(5):3608–14.
31. Srour B, Fezeu LK, Kesse-Guyot E, Alles B, Debras C, Druesne-Pecollo N, et al. Ultraprocessed food consumption and risk of type 2 diabetes among participants of the NutriNet-Santé prospective cohort. JAMA Intern Med. 2020;180(2):283–91.
32. Rauber F, Chang K, Vamos EP, Louzada MLD, Monteiro CA, Millett C, et al. Ultra-processed food consumption and risk of obesity: a prospective cohort study of UK Biobank. Eur J Nutr. 2021;60(4):2169–80.
33. Canhada SL, Luft VC, Giatti L, Duncan BB, Chor D, da Fonseca MDM, et al. Ultra-processed foods, incident overweight and obesity, and longitudinal changes in weight and waist circumference: the Brazilian Longitudinal Study of Adult Health (ELSA-Brasil). Public Health Nutr. 2020;23(6):1076–86.
34. Moradi S, HojjatiKermani MA, Bagheri R, Mohammadi H, Jayedi A, Lane MM, et al. Ultra-Processed food consumption and adult diabetes risk: a systematic review and dose-response meta-analysis. Nutrients. 2021;13(12):4410.
35. Nardocci M, Polsky JY, Moubarac JC. Consumption of ultra-processed foods is associated with obesity, diabetes and hypertension in Canadian adults. Can J Public Health. 2021;112(3):421–9.
36. Duan MJ, Vinke PC, Navis G, Corpeleijn E, Dekker LH. Ultra-processed food and incident type 2 diabetes: studying the underlying consumption patterns to unravel the health effects of this heterogeneous food category in the prospective lifelines cohort. BMC Med. 2022;20(1):7.
37. Willett W, Stampfer MJ. Total energy intake: implications for epidemiologic analyses. Am J Epidemiol. 1986;124(1):17–27.
38. Moradi S, Entezari MH, Mohammadi H, Jayedi A, Lazaridi AV, Kermani MAH, et al. Ultra-processed food consumption and adult obesity risk: a systematic review and dose-response meta-analysis. Crit Rev Food Sci Nutr. 2023;63(2):249–60.
39. Nilson EAF, Ferrari G, Louzada M, Levy RB, Monteiro CA, Rezende LFM. The estimated burden of ultra-processed foods on cardiovascular disease outcomes in Brazil: a modeling study. Front Nutr. 2022;9:1043620.
40. Louzada ML, Steele EM, Rezende LFM, Levy RB, Monteiro CA. Changes in obesity prevalence attributable to ultra-processed food consumption in Brazil between 2002 and 2009. Int J Public Health. 2022;67:1604103.
41. O'Hearn M, Lara-Castor L, Cudhea F, Miller V, Reedy J, Shi P, et al. Incident type 2 diabetes attributable to suboptimal diet in 184 countries. Nat Med. 2023;29(4):982–95.
42. World Health Organization. Red and processed meat in the context of health and the environment: many shades of red and green: information brief. Geneva: World Health Organization; 2023.

43. Safiri S, Karamzad N, Kaufman JS, Bell AW, Nejadghaderi SA, Sullman MJM, et al. Prevalence, Deaths and Disability-Adjusted-Life-Years (DALYs) due to type 2 diabetes and its attributable risk factors in 204 countries and territories, 1990–2019: results from the global burden of disease study 2019. Front Endocrinol (Lausanne). 2022;13:838027.
44. Popkin BM, Barquera S, Corvalan C, Hofman KJ, Monteiro C, Ng SW, et al. Towards unified and impactful policies to reduce ultra-processed food consumption and promote healthier eating. Lancet Diabetes Endocrinol. 2021;9(7):462–70.
45. Touvier M, da Costa Louzada ML, Mozaffarian D, Baker P, Juul F, Srour B. Ultra-processed foods and cardiometabolic health: public health policies to reduce consumption cannot wait. BMJ. 2023;383:e075294.
46. Bertram MY, Vos T. Quantifying the duration of pre-diabetes. Aust N Z J Public Health. 2010;34(3):311–4.
47. Lamparter J, Raum P, Pfeiffer N, Peto T, Höhn R, Elflein H, et al. Prevalence and associations of diabetic retinopathy in a large cohort of prediabetic subjects: the Gutenberg health study. J Diabetes Complications. 2014;28(4):482–7.
48. Moodie R, Stuckler D, Monteiro C, Sheron N, Neal B, Thamarangsi T, et al. Profits and pandemics: prevention of harmful effects of tobacco, alcohol, and ultra-processed food and drink industries. Lancet. 2013;381(9867):670–9.
49. Vandevijvere S, De Ridder K, Fiolet T, Bel S, Tafforeau J. Consumption of ultra-processed food products and diet quality among children, adolescents and adults in Belgium. Eur J Nutr. 2019;58(8):3267–78.
50. Sierra-Ruelas É, Bernal-Orozco MF, Macedo-Ojeda G, Márquez-Sandoval YF, Altamirano-Martínez MB, Vizmanos B. Validation of semiquantitative FFQ administered to adults: a systematic review. Public Health Nutr. 2021;24(11):3399–418.
51. Timon CM, van den Barg R, Blain RJ, Kehoe L, Evans K, Walton J, et al. A review of the design and validation of web- and computer-based 24-h dietary recall tools. Nutr Res Rev. 2016;29(2):268–80.
52. Dao MC, Subar AF, Warthon-Medina M, Cade JE, Burrows T, Golley RK, et al. Dietary assessment toolkits: an overview. Public Health Nutr. 2019;22(3):404–18.

Publisher's Note