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# Impact of Ultra-Processed Food and Sugar-Sweetened Beverage Consumption on Type 2 Diabetes Risk in Young Adults

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#### **Abstract**

Ultra-processed foods (UPFs) and sugar-sweetened beverages (SSBs) have risen sharply in global diets, especially among young adults. This review examines evidence linking UPF and SSB consumption to type 2 diabetes (T2D) risk in younger populations. Epidemiological studies consistently show higher T2D incidence with greater UPF and SSB intake. For example, a European cohort found a 17% higher T2D risk per 10% increase in UPF share. Similarly, a meta-analysis reported a ~27% rise in T2D risk per daily SSB serving. Mechanistically, UPFs and SSBs tend to be high in rapidly digested sugars and fats, promoting excess energy intake, weight gain, and insulin resistance. Additives (e.g. carrageenan, emulsifiers) and byproducts (acrylamide, bisphenols) in UPFs may further drive inflammation and metabolic dysfunction. Young adults consume more SSBs than older groups, and early-onset T2D is rising globally. Reducing UPF and SSB consumption - through public health policies, education, or substituting healthier options - could lower young-adult diabetes risk.

Keywords: ultra-processed foods, sugar-sweetened beverages, type 2 diabetes, young adults, dietary risk.

#### Introduction

Type 2 diabetes mellitus (T2DM) is increasingly affecting younger populations worldwide. Traditionally a disease of middle-aged and older adults, T2DM is now rising sharply in adolescents and young adults, driven by obesity and poor diet. Obesogenic diets high in ultra-processed foods (UPFs) and added sugars play a central role. UPFs - defined by the NOVA system as industrial formulations with multiple ingredients (sugars, fats, additives) and minimal whole foods - have proliferated in modern diets. Examples include sweetened cereals, snack cakes, processed meats, frozen meals, and soft drinks. Sugar-sweetened beverages (SSBs), such as sodas and sweetened juices, are a major UPF category. Young adults consume more UPFs and SSBs than older age groups, contributing a large fraction of daily calories (Tischmann, R., et al., 2023). For instance, CDC data show U.S. youth drink more caloric beverages than children or older adults, and global surveys confirm highest SSB intake in younger adults.

Excess intake of UPFs and SSBs is strongly linked to weight gain and metabolic abnormalities. Liquid sugars in SSBs add calories without satiety, often leading to excess energy intake and adiposity (Hall, K. D., et al., 2019). Fructose-heavy UPFs (e.g. HFCS-sweetened sodas, snacks) promote hepatic fat synthesis and insulin resistance. Moreover, UPFs often contain additives (e.g. emulsifiers, preservatives) and contaminants (e.g. acrylamide, bisphenol A) that may disrupt metabolism and inflammation. Given these concerns, researchers have investigated whether high UPF/SSB consumption raises T2DM risk in young adults. This paper reviews current evidence - including cohorts, meta-analyses, and mechanistic studies - linking UPF and SSB consumption to T2DM in younger populations, and discusses implications for prevention.

#### Ultra-Processed Foods and T2D Risk

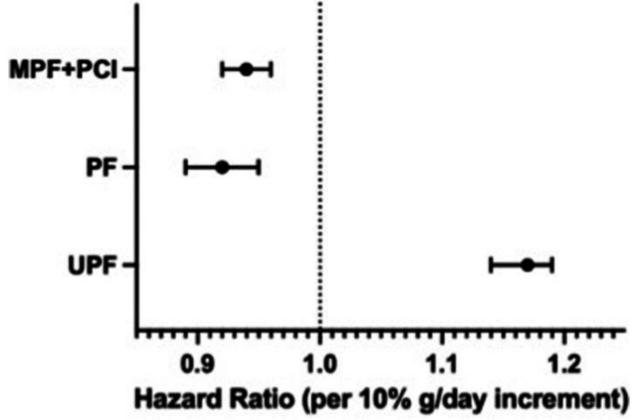
Ultra-processed foods (NOVA group 4) are industrial products containing added sugars, refined fats, artificial ingredients, and minimal whole food content. Common examples include sweetened breakfast cereals, packaged snacks, reconstituted meats (hot dogs, chicken nuggets), sugary yogurts, and ready-to-eat frozen meals. By contrast, Group 1 (unprocessed/minimally processed) includes fresh vegetables, fruits, grains, and meats, and Group 3 (processed foods) includes canned vegetables, simple breads, and cheeses. Modern diets in many countries are dominated by UPFs. In the U.S., UPFs contribute over 50% of daily energy intake. Young adults (ages ~20-40) especially rely on convenience foods and take-out, leading to high UPF intake. **Epidemiological Evidence:** Multiple prospective studies have linked higher UPF intake to greater T2DM risk. In the NutriNet-Santé cohort (France), each 10-percentage-point increase in dietary UPF proportion was associated with a 15% higher T2DM

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risk (adjusted hazard ratio HR=1.15, 95% CI 1.06-1.25). This remained significant after adjusting for BMI, lifestyle, and nutritional quality. Similarly, the EPIC (European Prospective Investigation into Cancer) analysis of 311,892 adults found that a 10% rise in UPF share corresponded to 17% higher T2DM incidence (HR $\approx$ 1.17). These studies controlled for confounders and suggest a dose-response relationship. Other cohorts agree: for example, a Spanish SUN project (N=20,060) reported 53% higher T2D hazard in the highest vs. lowest UPF tertile, and a UK Biobank analysis (N $\approx$ 21,700) found 44% higher odds in the top vs. bottom quartile of UPF intake. A meta-analysis of five longitudinal studies confirmed the overall trend: higher UPF diets significantly elevate T2DM risk.

**Table 1** Key prospective studies linking ultra-processed food intake to type 2 diabetes risk. All studies used NOVA classification to quantify UPF proportion. HR = hazard ratio: OR = odds ratio: O = quartile.

Study (Year)	Population / Cohort	Sample (n)	UPF Measure	Diabetes Outcome (Effect)
Srour et al. 2019 (France)	NutriNet-Santé prospective cohort	104,707 adults	% of diet from UPFs	HR ≈1.15 per 10% ↑ in UPF (95% CI: 1.06-1.25)
Dicken et al. 2024 (Europe)	EPIC prospective cohort	311,892 adults	% of diet from UPFs	HR 1.17 per 10% ↑ in UPF (95% CI: 1.14-1.19)
Levy et al. 2020 (UK)	UK Biobank prospective cohort	21,730 adults	UPF quartiles (Q1-Q4)	HR_Q4 vs Q1 = 1.44 (95% CI: 1.04- 2.02)
Llavero-Valero et al. 2021 (Spain)	SUN cohort (health professionals)	20,060 adults	UPF tertiles (T1- T3)	HR_T3 vs T1 = 1.53 (95% CI: 1.06- 2.22)
Nardocci et al. 2021 (Canada)	Canadian Cross- sectional survey	13,608 adults	% of energy from UPFs	OR_T3 vs T1 = 1.37 (95% CI: 1.01- 1.85)



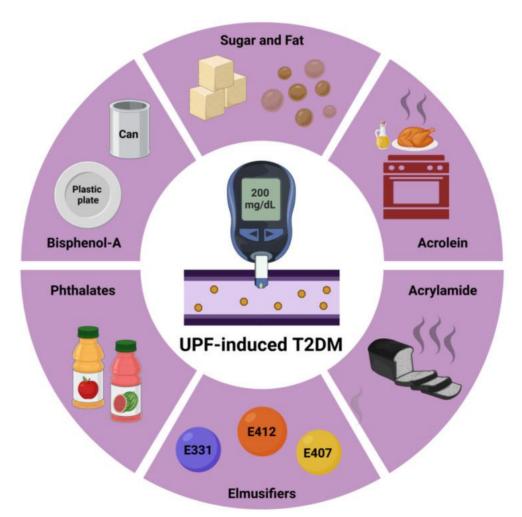
**Figure 1** Hazard ratios (HR) for incident type 2 diabetes per 10% increase in dietary intake share of each NOVA group (MPF+PCI = minimally processed foods + processed culinary ingredients, PF = processed foods, UPF = ultra-processed foods). Ultra-processed foods (right) show a marked positive association with T2D risk. Adapted from Dicken et al. (2024).

These findings imply that UPFs are a powerful modifiable risk factor for T2D. The magnitude of effect is comparable to classic dietary risks (e.g. refined grains, sugar) and remains after accounting for body weight. However, these studies largely involve adults of mixed ages; there is limited data specifically on young adults. Still, because young adults often have high UPF exposure and are entering risk age for metabolic disease, these results likely apply.

### **Mechanisms Linking UPFs to Diabetes**

Several dietary and biological mechanisms explain why UPFs may increase T2DM risk. First, UPFs are energy-dense and hyperpalatable. A controlled feeding trial showed that subjects eating an ultra-processed diet consumed ~508 more calories per day and gained ~0.9 kg in two weeks, whereas on an unprocessed diet they lost ~0.9 kg (Hall, K. D., et al., 2019). Excess energy intake leads to weight gain, central adiposity, and insulin resistance - key pathways to T2D (Hall, K. D., et al., 2019). UPFs also tend to have high glycemic load: refined starches and sugars in UPFs cause rapid glucose spikes and demand on pancreatic insulin release. Regular high glycemic load can induce beta-cell stress and insulin resistance over time. Fructose (from HFCS, fruit juices) in many UPFs is uniquely lipogenic: it is rapidly metabolized in the liver to fat, promoting non-alcoholic fatty liver disease, elevated triglycerides, and visceral fat deposition, all of which impair insulin action.

Beyond macronutrients, UPFs contain additives and contaminants that may affect metabolism. Emulsifiers like carrageenan (E407) and carboxymethylcellulose (E466) are common in processed foods. Preclinical studies show carrageenan can trigger intestinal inflammation and increase CRP/IL-6 levels, which are linked to insulin resistance. In France's NutriNet cohort, high carrageenan intake was associated with higher T2D risk. Phthalates and bisphenol-A (from packaging) are known endocrine disruptors; higher phthalate levels correlate with obesity and insulin resistance. Acrylamide and acrolein toxic byproducts formed in high-fat UPFs (fried potatoes, chips, baked goods) have been implicated in metabolic dysfunction. Dietary acrylamide intake in a Tehran cohort was linked to an increased T2D risk in women.



**Figure 2** Key components of ultra-processed foods implicated in T2DM risk. Excess added sugar and saturated fat, combined with additives (e.g. emulsifiers E331, E412, E407) and contaminants (acrylamide, acrolein, bisphenol-A, phthalates), may promote insulin resistance and inflammation. (Adapted from Kounatidis 2025.)

These mechanisms suggest that UPFs are harmful beyond their calories. Even at equal caloric intakes, a diet high in UPFs can alter energy balance and metabolism. Therefore, dietary guidelines increasingly emphasize limiting UPFs.

#### Sugar-Sweetened Beverages and T2D Risk

Prevalence and Young Adult Consumption: Sugar-sweetened beverages (SSBs) e.g. sodas, energy drinks, sweetened teas, fruit drinks are a major source of added sugars. Their consumption remains high in young adults. For example, a U.S. report found 63% of youth (ages 2-19) and 49% of adults consumed an SSB on a given day (2011-2014). Among adults, males and younger age groups drink the most (Tischmann, R., et al., 2023). Global data show young (20-39 years) adults have higher SSB intake than older adults. This is critical because adolescent and young-adult T2DM is rising; one CDC analysis found T2DM incidence in U.S. youths (<20 years) doubled from 9 to 18 per 100,000 from 2002 to 2018 (Tischmann, R., et al., 2023). High SSB intake likely contributes to this rise.

**Epidemiological Evidence:** Many cohorts link frequent SSB intake with higher T2DM incidence. A pooled analysis of three large U.S. cohorts (Nurses' Health Studies I & II, Health Professionals) found that increasing sugary beverage consumption by ≥0.5 servings per day over 4 years was associated with a 16% higher subsequent T2D risk (HR ≈1.16, 95% CI 1.01-1.34). A meta-analysis of 34 prospective studies reported that each additional daily serving of SSB increased T2D risk by ~27% (pooled RR ≈1.27, 95% CI 1.15-1.41). In the Coronary Artery Risk Development in Young Adults (CARDIA) study, young adults consuming >2 servings/day of SSB had a 6% higher T2D risk per serving (HR 1.06) while artificially sweetened beverage (ASB) intake carried a 12% higher risk per serving. Ding et al. (2024) followed a Chinese cohort for ~12 years and found those drinking >2 SSBs per day had a 17% higher T2D hazard (HR 1.17, 95% CI 1.05-1.31) compared to non-drinkers. These associations persist after adjusting for BMI, suggesting direct effects of SSBs beyond weight gain.

**Table 2** Key studies of sugar-sweetened beverages (SSBs) and type 2 diabetes risk. SSB = sugar-sweetened beverages; ASB = artificially sweetened beverages. HR = hazard ratio; RR = relative risk; \* indicates pooled result from multiple cohorts.

Study (Year)	Population / Cohort	Sample (n)	Exposure	Diabetes Outcome (Effect)
Hirahatake et al. 2019 (USA)	CARDIA cohort (young adults)	4,134	SSB, ASB (serv/day)	SSB >2/day: HR=1.06 (per serving); ASB >2/day: HR=1.12
Meng et al. 2021 (Meta-analysis)	34 prospective studies (global)	~600,000	SSB (per 1 serving/day)	RR≈1.27 (95% CI: 1.15-1.41) per serving
Drouin-Chartier et al. 2019 (USA)	Nurses'/HPFS (pooled women+men)	~192,000	↑0.5 serving/day SSB	HR=1.16 (95% CI: 1.01-1.34) for +0.5 serving/day
Ding et al. 2024 (China)	Chinese cohort (mixed adults)	37,168	SSB >2/day	HR=1.17 (95% CI: 1.05-1.31) for >2 SSB/day vs none

Notably, risk extends to artificially sweetened beverages (ASBs). Meta-analyses indicate that each ASB serving also raises T2DM risk (pooled RR ≈1.13 per daily serving). However, this may partly reflect reverse causation (people at risk switching to ASBs). Pure fruit juices (though natural) can also contribute, as they provide similar sugars.

Recent global analyses quantify the SSB impact: in 2020, an estimated 2.2 million new T2DM cases ( $\approx$ 10% of all new cases) were attributable to SSBs. Importantly, younger adults bear more of this burden: the proportional SSB-attributable T2DM risk is higher in younger vs. older adults. In Latin America, where SSB intake is very high, up to 24% of new T2D cases were linked to SSBs.

# **Mechanisms Linking SSBs to Diabetes**

Sugar-sweetened beverages drive T2DM through several pathways. First, they provide high calories in liquid form, which do not trigger strong satiety cues. A single 12-oz soda (~150 kcal) daily can theoretically add ~5 lbs/year if uncompensated. Over time this leads to weight gain and obesity, the principal risk factor for T2D. Clinical trials support this: replacing sugar drinks with non-caloric beverages in children reduced weight gain and fat accumulation. Conversely, habitual SSB intake is linked to greater adiposity in longitudinal studies.

Second, the rapid digestion of SSB sugars spikes blood glucose and insulin acutely. Frequent spikes may cause pancreatic beta-cell stress and eventual dysfunction. Fructose, prevalent in sodas (HFCS) and fruit drinks, is metabolized in the liver to glyceraldehyde-3-phosphate (see Figure 4) and then to acetyl-CoA, fueling de novo lipogenesis. This leads to fat accumulation in liver and muscle, worsening insulin resistance. High fructose intake also raises uric acid, which can induce inflammation and vascular dysfunction. Over time, these metabolic effects impair glucose regulation independent of weight.

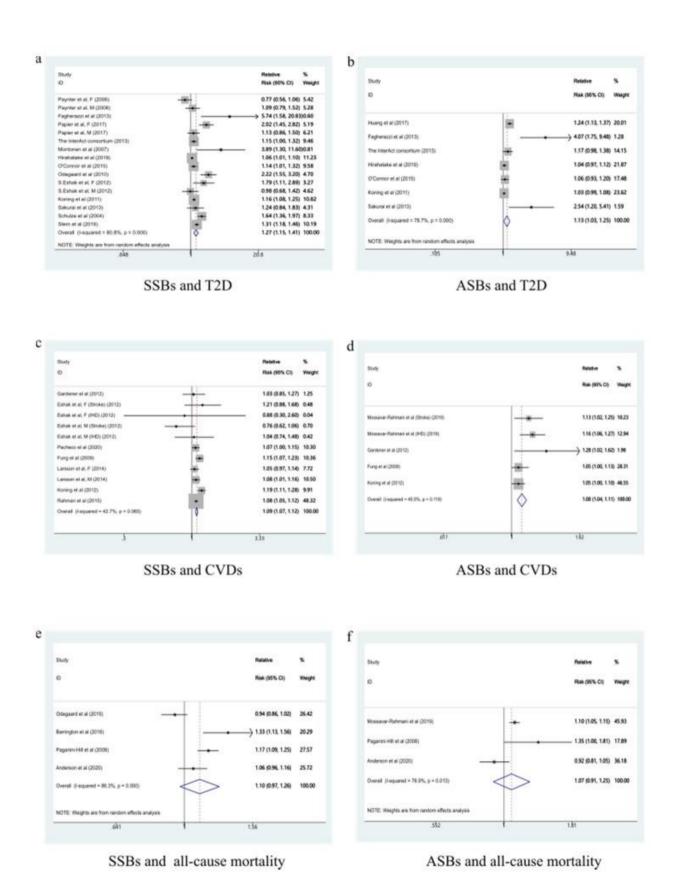
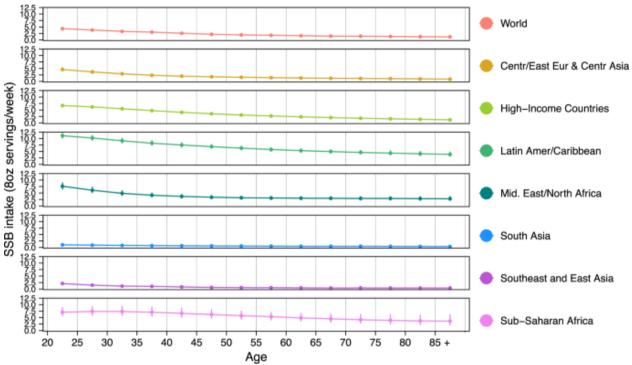


Figure 3 Forest plot from meta-analysis (Meng et al. 2021) showing the pooled relative risk (RR) for type 2 diabetes per one daily serving of sugar-sweetened beverages (SSBs). The diamond summary indicates a ~1.27-fold higher T2D risk per SSB serving.



**Figure 4** Average weekly sugar-sweetened beverage (SSB) intake (8-oz servings) by adult age and region in 2018. Younger adults (20-39 years) consume substantially more SSBs than older groups in all regions (e.g. ~10/week in Latin America).

Data from Lara-Castor et al. (2023).

Third, high SSBs can displace healthier foods (like whole fruit, milk), reducing fiber and micronutrient intake. Chronic excess sugar intake also raises lipids and blood pressure, compounding metabolic syndrome. Over time, these diet-driven changes trigger systemic inflammation and atherosclerosis, accelerating T2D development.

In young adults, these effects may manifest quickly. Early adulthood is a period of accelerating insulin resistance (due to weight gain or lifestyle) and metabolic imprinting. High SSB and UPF intake during these years likely accelerates pathogenesis of T2D, contributing to earlier onset.

# **Public Health Implications**

The evidence strongly suggests that reducing UPF and SSB consumption could prevent many T2DM cases, especially among the young. Policy measures like soda taxes, warning labels, and restricting sales in schools have shown some success in lowering SSB intake. For example, a Cochrane review found that environmental interventions (taxes, improved availability) can reduce SSB consumption and improve weight outcomes. Educational campaigns and improved food labeling can also shift choices. Clinically, replacing SSBs with water, tea, or coffee (without added sugar) is recommended; one study found that substituting one SSB daily with water reduced T2DM risk by ~5%.

For UPFs, guidelines advise emphasizing unprocessed whole foods. Interventions could include front-of-pack labeling (e.g. warning logos on high-UPF products) and promoting home cooking. Some countries have considered regulating levels of additives. Given that UPFs are often cheaper and heavily marketed, attention to socioeconomic and cultural factors is needed. In practice, individuals can limit UPFs by reading labels, avoiding "hyper-palatable" snacks, and choosing fresh or minimally processed staples.

#### Conclusion

Heavy consumption of ultra-processed foods and sugar-sweetened beverages is consistently linked to higher risk of developing type 2 diabetes, including among young adults. Cohort studies show that diets rich in UPFs substantially raise T2DM incidence, and each extra daily SSB significantly increases risk. Mechanisms involve excess calories, rapid sugar absorption, lipogenesis, and pro-inflammatory additives. These effects are especially concerning for young adults, who consume large quantities of these foods and may develop early-onset diabetes with long-term health consequences. Preventive efforts should focus on reducing UPF/SSB intake through public policy, education, and healthier dietary patterns.

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