Action on ultra-processed foods needs robust evidence

Should we regulate or even ban ultra-processed foods (UPFs)? This question is asked with increasing frequency, spurred by popular media interest in research that associates these foods with adverse health outcomes. With evidence suggesting that more than 50% of the energy consumed in UK diets is derived from UPFs, some voices urge immediate action.

The most widely used system for classifying food processing, the Nova classification, defines foods as UPFs based on how they are processed and their use of industrially derived ingredients, without considering the health effects of these processes or ingredients. The UPF classification therefore covers a disparate group of foods that differ markedly in their ingredients and additives, the methods used to prepare them, and their nutritional composition and energy content.

From a regulatory perspective, this definition presents a quandary. Some aspects of UPF definitions relate to the use of ingredients, such as flavourings, rather than processing per se. Moreover, many commonly consumed UPFs are also high in fat, sugar, and salt (HFSS), and meet existing definitions of unhealthy foods. Data from the UK suggest that 59% of the energy from UPFs comes from foods already classified as HFSS. Of the remaining 41% of energy from UPFs that is not HFSS, most comes from foods such as bread and potato products. Dairy products are the leading sources of HFSS, non-UPF energy.

Will regulating UPFs provide population benefit beyond regulating HFSS foods? The argument that UPFs should be regulated for health reasons is founded on several assumptions. First, that specific features of UPFs cause negative health outcomes beyond their nutrient profiles. Second, that a definition of UPFs can incorporate these specific features alone. Third, that inducing such a shift does not cause or exacerbate health inequalities, will improve health outcomes, and is cost-effective.

Evidence on the clinical effects of UPFs is almost entirely drawn from observational studies of varying populations, designs, and statistical methods. Disentangling the specific UPF features that cause harm from this vantage point is extremely difficult. In one large cohort study, associations between UPF consumption and mortality disappeared when stratified by the nutritional quality of diets or controlling for pack-years of smoking. Associations also varied substantially by UPF class, with ready-to-eat UPF animal products conveying the greatest risk and some other UPF products having no associations. That much of the epidemiological evidence on UPFs is contradictory and poor quality is too often dismissed—this evidence base demands epistemological humility.

Only a handful of controlled trials have explored the short-term effects of UPFs on energy intake. In the first published trial, participants had higher energy intake on an ultra-processed diet than on a minimally processed diet largely matched for macronutrient composition. This is the strongest evidence to date that stark differences in processing, additives, and ingredients between diets might alter energy intake, yet the causal mechanisms remain elusive, long-term outcomes are uncertain, and key questions persist. Would such effects be seen if comparing UPF and processed food diets matched for nutrient composition, for instance?

The vacuum of good evidence on UPFs speaks to a broader problem in nutritional science: a reluctance of funders to invest in high-quality, expensive, randomised controlled trials. Powering trials for short-term or medium-term surrogate outcomes is necessary to guide policy, particularly when some proposed interventions disproportionately affect more disadvantaged sectors of society. The ambitious UPDATE trial is assessing 8-week weight loss on UPF or minimally processed diets, and clinical and financial outcomes following a 6-month behavioural intervention to reduce UPF consumption. Mechanistic research is also feasible and ethical, as demonstrated by the ADDapt trial, which is assessing the effect of dietary emulsifier content on Crohn's disease symptoms. Conducting trials to investigate each factor potentially underlying UPF-specific harms might not be practical, but focus can be directed towards areas with the strongest epidemiological and preclinical evidence.

Pleas to overhaul the food system to mitigate the specific harms of UPFs are well meaning but overly reliant on limited data. To move the debate forwards, a strengthened, specific, and actionable UPF definition, drawing upon vastly improved causal evidence, is crucial. While those necessary data are collected, efforts would be better focused on driving through policies targeting established nutritional drivers of ill-health and reducing economic barriers to healthy food.

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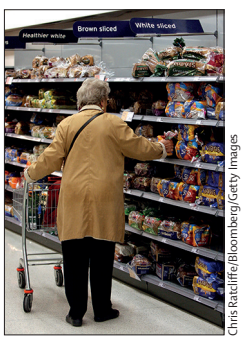

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For **UPF consumption in the UK** see *Nutrients* 2021; **13:** 2778

For the **overlap between UPFs and HFSS** see *medRxiv* 2024; published online Aug 24. https://doi.org/10.1101/2024.08.27.24312650 (preprint)

For the **cohort study** see *BMJ* 2024; **385:** e078476

For **more on the conflicting epidemiological evidence** see *Eur J Clin Nutr* 2024; published online Sept 26. https://doi.org/10.1038/s41430-024-01515-8

For the **first trial of UPFs on weight gain** see *Cell Metab* 2019; **30:** 67–77; and *Diabetes Obes Metab* 2024; **26:** 5431–43

For **more on the UPDATE trial** see *BMJ Open* 2024; **14:** e079027

For the **ADDapt trial** see https://clinicaltrials.gov/study/NCT04046913