Food Toxicology and Safety

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The Inverted Burden: Food Toxicology, Regulatory Capture, and the Case for Precautionary Food Safety

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Author's Note

This paper exists because of Goal 10.

Goal 10 states: *Food contains only things proven safe. If it's not proven safe, it doesn't go in food. The precautionary principle applied to what you eat.*

That sounds like a radical position. It is not. It is the position we already apply to every other substance that enters the human body under controlled conditions. Pharmaceuticals must pass years of clinical trials. Pesticides require toxicological assessment before agricultural use. Building materials must meet safety standards before installation. Even novel psychoactive substances, in New Zealand, must be proven safe before sale.

Food is the exception. The one substance that enters every human body, multiple times daily, from birth to death, across every demographic, in every country, without medical supervision, without dose control, without informed consent – is the one substance that does not require proof of safety before market entry.

You provide? The food you're providing is poison.

That is not rhetoric. It is a description. The man who works double shifts to put food on the table for his family is, in most industrialised countries, putting food on the table that contains substances no one has proven are safe, approved by the companies selling them, in quantities no one monitors, for durations no one tracks. When his children develop allergies, when his partner

develops an autoimmune condition, when he develops type 2 diabetes at fifty, these are treated as personal health outcomes – genetics, lifestyle choices, bad luck. They are not. They are the predictable result of a food system that defaults to permission rather than precaution, that treats the human population as an experimental cohort, and that has been running this experiment for seventy years against two hundred thousand years of evolutionary dietary history.

The results of the experiment are in. This paper documents them.

A zookeeper who fed an animal a substance not yet proven safe would be fired. We do it to ourselves three times a day.

Abstract

The modern food supply operates under an inverted burden of proof: substances are permitted in food until demonstrated harmful, rather than excluded until demonstrated safe. This paper argues that this inversion is ethically indefensible, scientifically unjustifiable, and causally linked to the epidemic-scale chronic disease burden of industrialised populations.

We examine the regulatory frameworks governing food safety across four jurisdictions – the United States (FDA/GRAS), the European Union (EFSA), Australia and New Zealand (FSANZ), and international standards (Codex Alimentarius) – and demonstrate that each operates on a spectrum from near-total industry self-certification (US GRAS) to imperfect precaution (EU), with none approaching the evidentiary standard applied to pharmaceuticals. We document the GRAS (Generally Recognized As Safe) loophole, under which approximately 1,000 chemicals have been self-affirmed as safe by their manufacturers without any notification to the FDA, and under which 100% of GRAS expert panels between 1997 and 2012 had financial conflicts of interest (Neltner et al., 2013).

Drawing on cross-cultural epidemiological evidence from traditional populations – including the Kitava study (n=1,200; zero acne, near-zero cardiovascular disease, diabetes, and cancer), Okinawan longevity data, Inuit disease profiles, and the Tsimane (n=705; lowest atherosclerosis ever recorded) – we demonstrate that Homo sapiens consuming species-appropriate diets do not develop the diseases currently treated as inevitable features of human ageing. These populations are not genetically privileged; when they adopt Western diets, Western disease rates emerge within a single generation.

We examine specific classes of concern: the NOVA ultra-processed food classification and the landmark NIH inpatient trial (Hall et al., 2019) demonstrating 500 excess calories per day from ultra-processed versus minimally processed diets; the glyphosate evidence base and its regulatory contestation; the seed oils debate; food contact material contamination (BPA, PFAS, phthalates); emulsifier-driven gut microbiome disruption (Chassaing et al., 2015); food dye behavioural effects in children (McCann et al., 2007); and Advanced Glycation End-products as a mechanistic pathway from processed food to chronic disease.

We conduct a detailed comparison of substances banned or restricted in the EU but legal in the United States and Australia, demonstrating that identical evidence bases produce divergent regulatory outcomes depending on the structural relationship between regulator and regulated. We critique the Australian food safety authority FSANZ for its reliance on applicant-supplied data, its

limited independent research capacity, and its alignment with US-style post-market surveillance rather than EU-style precaution.

We propose that New Zealand's Psychoactive Substances Act 2013 – which reversed the burden of proof for novel psychoactive substances – provides a directly transferable legislative template for food regulation. The central argument is simple: food should not contain anything not yet proven safe beyond reasonable doubt.

Keywords: food safety, precautionary principle, GRAS, regulatory capture, ultra-processed food, NOVA classification, FSANZ, food additives, glyphosate, seed oils, endocrine disruptors, food toxicology

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Chapter 1: Introduction – The Seventy-Year Experiment

Every animal on Earth eats what it evolved to eat.

Lions eat what lions ate a hundred thousand years ago. Salmon eat what salmon ate a hundred thousand years ago. Elephants, sparrows, beetles, whales. Every organism in every ecosystem

on this planet consumes the food that shaped its biology over millions of years of evolutionary adaptation.

Every animal except one.

Homo sapiens, in the last approximately seventy years, has undergone the most radical dietary transformation in the history of any species. The food consumed by a typical human in an industrialised nation in 2026 bears almost no resemblance to the food consumed by any human at any point in the preceding two hundred thousand years of the species' existence. The shift is not a matter of degree. It is categorical. The majority of calories consumed in the modern Western diet come from substances that did not exist in any human diet before the mid-twentieth century: refined seed oils, high-fructose corn syrup, synthetic emulsifiers, artificial preservatives, flavour enhancers, colourings, stabilisers, and ultra-processed food matrices that no human metabolism was ever exposed to during the evolutionary period in which human biology was formed (Monteiro et al., 2013).

Ultra-processed foods now constitute 50-60% of total caloric intake in the United States, United Kingdom, Canada, and Australia (Monteiro et al., 2019; Machado et al., 2019). More than half of what the average person in these countries eats is food that did not exist in any human diet at any point in the preceding two hundred thousand years. In Australia specifically, ultra-processed foods account for approximately 42% of total energy intake in adults and up to 50% in children (Machado et al., 2019). These are not fringe foods consumed by negligent individuals. They are the default diet of the industrialised world.

Seventy years. Against two hundred thousand. Against millions, if you count the broader evolutionary lineage. We took a system that worked – a diet that produced the biology we currently inhabit – and replaced it with something entirely novel, entirely untested at scale, in the blink of an evolutionary eye.

The experiment ran. You do not have to speculate about the results. They are in.

Obesity: epidemic. 42% of American adults are obese (CDC, 2020). The rate was under 15% in 1970. In Australia, 31.7% of adults are obese and a further 35.6% are overweight – two thirds of the adult population (ABS, 2022). Type 2 diabetes: epidemic. Global prevalence has quadrupled since 1980 (WHO, 2016). Heart disease: the leading cause of death worldwide. Cancer: rates climbing across virtually every industrialised population.

In seventy years.

No other species has done this. No other species has replaced its evolutionary diet with a novel industrial product and experienced the consequences at population scale. We are running an experiment on eight billion people with no control group, no monitoring, no consent, and no exit.

Actually – that is not quite right. There is a control group. The control group is the traditional populations. Kitava. Okinawa. The Inuit on their ancestral diet. The Tsimane. They are the control group, and they do not have the diseases. They are the same species eating a different diet, and they are fine. The experiment has a control group and the control group is healthy. The experimental group – us – is sick. The variable is the food.

This is not a small-sample anomaly. It is not a cherry-picked comparison. It is a pattern observed across every traditional population ever studied, on every continent, across every macronutrient ratio, in every climate – from the Arctic to the tropics, from high-fat diets to high-carbohydrate diets, from fishing cultures to farming cultures. The common variable is never the macronutrient

ratio. It is never the specific food. It is always the same thing: the absence of industrial food processing and synthetic chemical additives. The presence of food that is recognisably food – things that grew in the ground or swam in the sea or walked on the land, prepared by methods that human beings have used for tens of thousands of years.

This paper examines the system that decides what goes into that food. It is not what you think it is.

1.1 The Public Assumption

The public assumption is that food additives are tested before they reach the market – that somewhere, a team of scientists in a government laboratory examines each substance, determines it is safe, and then grants permission for it to enter the food supply.

This assumption is wrong.

The system that regulates what goes into food in most industrialised countries does not require proof of safety before market entry. It requires proof of harm before removal. The burden is inverted. The substance is innocent until proven guilty. Your body is not.

1.2 What This Paper Does

This paper documents the inversion across four jurisdictions. It examines the specific mechanisms by which the burden of proof is reversed – the GRAS framework in the United States, the applicant-funded assessment model in Australia and New Zealand, and the imperfect but measurably more precautionary approach of the European Union. It presents the evidence base for specific classes of concern: ultra-processed food, glyphosate, seed oils, food contact materials, emulsifiers, food dyes, artificial sweeteners, Advanced Glycation End-products, and endocrine disruptors. It documents the traditional populations that demonstrate, through their existence, that the Western disease burden is not a feature of being human but a feature of eating what the Western food supply provides. It presents the legislative precedent from New Zealand for reversing the burden of proof. And it makes the case for Goal 10: food contains only things proven safe.

The case is not difficult to make. The difficulty is in accepting what it means: that the system you trusted to keep you safe was never designed to keep you safe. It was designed to keep the market moving.

Chapter 2: The Precautionary Principle and Its Inversion

2.1 The Principle Stated

The precautionary principle, as formulated in the 1992 Rio Declaration on Environment and Development, states: "Where there are threats of serious or irreversible damage, lack of full scientific certainty shall not be used as a reason for postponing cost-effective measures to prevent environmental degradation" (United Nations, 1992). The principle has been adopted in various forms across international environmental law, public health policy, and chemical regulation. Its logic is straightforward: when the potential consequences of an action are severe and the evidence base is incomplete, the default position should be caution rather than permission.

The precautionary principle is not an exotic innovation. It is the default assumption in every domain where competent adults manage risk. An engineer does not install an untested structural beam and wait for the building to collapse before conducting a safety review. A pilot does not fly an uninspected aircraft and wait for an engine failure before checking the engine. A veterinarian does not administer an untested compound to an animal and wait for symptoms before evaluating toxicity.

Only in food regulation is the precautionary principle inverted. The default position is permission. A substance may be added to the food supply unless evidence demonstrates it is harmful. The burden falls not on the manufacturer to prove safety, but on regulators, researchers, and ultimately the consuming public to prove danger – after exposure has already occurred, often across decades and millions of people.

2.2 Precautionary Principle vs Post-Market Surveillance

Two fundamentally different models exist for regulating substances that interact with human biology:

The precautionary model (pre-market proof of safety): A substance is prohibited by default. Before it can be sold, the proponent must demonstrate – through independent testing, peer-reviewed research, and regulatory review – that it is safe for human consumption at the doses and durations at which it will actually be consumed. If the evidence is insufficient or ambiguous, the substance is not permitted. The cost of generating evidence falls on the proponent. This is how pharmaceutical regulation works. This is how New Zealand's Psychoactive Substances Act works. This is, in principle, how the EU's food additive framework works.

The post-market surveillance model (prove harm after exposure): A substance is permitted by default. After it enters the market, regulators monitor for signals of harm. If sufficient evidence accumulates – typically over years or decades, and typically contested by the industries that profit from the substance – regulatory action may follow. The cost of generating evidence falls on regulators and, ultimately, on the exposed population. This is how the US GRAS system works. This is, in practice, how much of Australia's food regulation functions despite formal pre-market assessment requirements.

The distinction is not academic. It is the difference between a system that protects people from unknown risks and a system that exposes people to unknown risks and waits to see what happens. Post-market surveillance is not precaution. It is an autopsy system. It waits for the bodies, then argues about whether the bodies count.

2.3 The Asymmetry of Evidence Requirements

The evidentiary asymmetry is stark. To introduce a substance into the food supply, a manufacturer needs relatively minimal evidence of safety – in many cases, no independent evidence at all. To remove a substance from the food supply, regulators need overwhelming evidence of harm, sustained across multiple studies, sufficient to withstand legal challenge by well-resourced corporate defendants, and sufficiently specific to attribute harm to the particular substance rather than to "lifestyle factors" or "confounders."

This asymmetry is not accidental. It reflects the distribution of resources and incentives. The entities that profit from introducing substances into the food supply are concentrated, well-funded,

and highly motivated. The entities that bear the cost – individual consumers – are dispersed, poorly informed, and have no mechanism for collective action on the timescale relevant to chronic disease.

The result is a ratchet effect: substances accumulate in the food supply over time, with each addition facing low barriers and each removal facing high ones. The modern processed food supply, containing thousands of additives that did not exist a century ago, is the predictable output of this ratchet.

2.4 The Pharmaceutical Comparison

The contrast with pharmaceutical regulation illuminates the absurdity. Before a pharmaceutical compound can be administered to patients, it must pass through:

- **Preclinical testing:** Laboratory and animal studies to establish basic safety and mechanism of action.
- **Phase I trials:** Small-group studies (20-100 people) to establish safety, dosage, and side effects.
- **Phase II trials:** Larger studies (100-300 people) to assess efficacy and further evaluate safety.
- **Phase III trials:** Large-scale studies (1,000-3,000+ people) to confirm efficacy, monitor side effects, compare with existing treatments.
- **Regulatory review:** Comprehensive assessment by an independent agency (FDA, TGA, EMA).
- **Phase IV (post-market surveillance):** Ongoing monitoring after approval.

This process typically takes 10-15 years and costs $1-2 billion. It is considered the minimum acceptable standard for a substance that will be administered to patients under medical supervision, at specific doses, for defined durations, with informed consent.

Food additives – substances administered to every person in a population, without medical supervision, at uncontrolled doses, for a lifetime, without informed consent – face no comparable requirement. A substance consumed by a small population of patients under controlled conditions faces more stringent safety requirements than a substance consumed by billions of people under uncontrolled conditions from birth to death.

The disparity is not defensible on any rational basis. The thing you swallow once, under medical guidance, has to prove it will not hurt you. The thing you swallow three times a day, every day, forever, does not.

Chapter 3: The GRAS Loophole – The Manufacturer Marks Its Own Homework

3.1 The Mechanism

In the United States, the mechanism for the inversion of the precautionary principle has a name: GRAS, or Generally Recognized As Safe. Under the 1958 Food Additives Amendment to the Federal Food, Drug, and Cosmetic Act, food additives must receive FDA approval before use – unless they are "generally recognized, among qualified experts, as having been adequately shown to be safe under the conditions of their intended use."

This exception has swallowed the rule.

The GRAS process operates as follows:

1. A manufacturer identifies a substance it wishes to add to food products.
2. The manufacturer conducts or commissions a safety evaluation, typically using its own funding and, frequently, its own scientists or consultants with financial ties to the company.
3. An expert panel – selected and paid by the manufacturer – reviews the evidence and determines whether the substance qualifies as GRAS.
4. The manufacturer may (but is not required to) notify the FDA of this determination.
5. If the FDA is notified, it may (but is not required to) review the submission. The FDA does not "approve" GRAS determinations; it merely indicates whether it has "no questions" about the manufacturer's conclusion.
6. If the FDA is not notified – and there is no legal requirement for notification – the substance enters the food supply with no regulatory review whatsoever.

3.2 The Numbers

Neltner et al. (2011) documented the scale of the problem in *Comprehensive Reviews in Food Science and Food Safety*. They found that as of 2010, approximately 10,000 substances were allowed in food. Of these, roughly 3,000 had been determined to be GRAS – and the majority of those GRAS determinations were made by the manufacturers themselves, without FDA review or public notification. Between 1997 and 2012, the FDA received approximately 1,000 voluntary GRAS notifications. It rejected zero. Not one.

A follow-up study by Neltner et al. (2013) in *JAMA Internal Medicine* examined the conflicts of interest in the GRAS determination process. Among GRAS determinations made between 1997 and 2012, every single determination was made by an expert panel with financial ties to the manufacturer. The rate of financial conflict of interest was 100%. This is not a system that produces independent safety assessments. It is a system in which the company selling the substance hires experts to certify that the substance is safe, and the regulator does not review the determination.

3.3 The Invisible Thousand

The Pew Charitable Trusts, which has tracked GRAS reform for over a decade, estimated in 2013 that approximately 1,000 chemicals had been self-affirmed as GRAS by their manufacturers without any notification to the FDA whatsoever. These are substances in the food supply that the regulatory agency responsible for food safety has never evaluated and may not know exist.

Read that again. There are approximately one thousand chemical substances in the food you eat that the FDA – the agency whose entire purpose is to ensure your food is safe – has never reviewed and may not even be aware of. They are there because the companies that sell them decided they were safe, told no one, and put them in your food.

A 2010 investigation by the US Government Accountability Office (GAO) confirmed the inadequacy of the GRAS process, finding that the FDA did not systematically reconsider the safety of substances already designated as GRAS when new scientific information emerged, and that the voluntary nature of the notification process meant the FDA was unaware of many GRAS determinations being made by industry (GAO, 2010).

3.4 The System Is Working as Designed

The GRAS system is not broken. It is working exactly as designed. It was designed by and for the food industry, and it performs its designed function: it allows substances to enter the food supply with minimal friction, at minimal cost to the manufacturer, and with minimal regulatory oversight. The system is optimised for throughput, not for safety. It is a pipeline, not a filter.

The fox does not merely guard the henhouse. The fox decides which animals are safe for the hens to live with.

Chapter 4: Regulatory Comparison – EU, US, Australia, and Codex Alimentarius

4.1 The European Union: Imperfect Precaution

The European Union operates under a fundamentally different regulatory philosophy from the United States. Regulation (EC) No 178/2002 explicitly invokes the precautionary principle as a foundational element of EU food law. Regulation (EC) No 1333/2008 on food additives requires pre-market authorisation: a food additive may not be placed on the market or used in food unless it is included in the EU's positive list and has been evaluated by the European Food Safety Authority (EFSA).

The EU system is not perfect. Industry still funds much of the safety testing. The revolving door between regulatory agencies and food corporations exists in Europe as it does in the United States. EFSA has been subject to recurring criticism regarding conflicts of interest on its scientific panels, reliance on industry-supplied data, and delays in acting on emerging safety concerns (Robinson et al., 2013; Corporate Europe Observatory, 2017).

But the outcomes are measurably different. The structural difference – pre-market authorisation versus post-market surveillance – produces a different food supply. The EU has restricted or banned substances that remain freely used in US and Australian food. The divergence is not scientific. It is structural. The EU's regulatory architecture defaults toward caution. The US system defaults toward permission. Australia falls somewhere between, closer to the US in practice despite formal similarities to the EU in process.

4.2 The United States: GRAS and Beyond

As documented in Chapter 3, the US system is the most extreme case of inverted burden of proof. The GRAS framework allows industry self-certification. The FDA's enforcement capacity is limited by chronic underfunding – the agency is responsible for regulating approximately 80% of the US food supply with a budget and staffing level that do not permit comprehensive oversight. Pre-market review is the exception rather than the rule for substances entering the food supply.

The FDA's statutory authority is reactive: it can act against substances shown to be harmful, but it has limited authority to require pre-market demonstration of safety for substances classified as GRAS. Legislative efforts to reform the GRAS system have repeatedly failed, opposed by food industry lobbying that consistently ranks among the largest lobbying expenditures in Washington.

4.3 Codex Alimentarius: The Lowest Common Denominator

The Codex Alimentarius Commission, established jointly by the FAO and WHO in 1963, sets international food standards that influence national regulation globally. Its standards are referenced in WTO trade dispute resolution, giving them practical force even where they are not formally binding.

Codex processes have been criticised for excessive industry influence. Industry representatives participate in national delegations and in the scientific committees that inform Codex decisions. The standards produced reflect the lowest common denominator of political acceptability rather than the best available evidence on safety. For countries like Australia that reference Codex standards in their own regulatory processes, this creates a ceiling on precaution: domestic regulation is constrained by international standards that are themselves constrained by industry participation.

4.4 The Comparison Table

The same substances, the same research base, different regulatory conclusions:

Substance	EU Status	US Status	Australia Status
Titanium dioxide (E171)	Banned in food (2022)	Permitted	Permitted
Potassium bromate	Banned	Permitted in bread	Banned
BHA (butylated hydroxyanisole)	Restricted	Widely used	Permitted
Azodicarbonamide	Banned	Permitted (bread/flour)	Permitted with limits
Red 40, Yellow 5, Yellow 6	Require warning labels	No warning required	Permitted, no warning
Ractopamine (livestock drug)	Banned	Permitted	Permitted
rBGH/rBST (growth hormone)	Banned	Permitted in dairy	Banned
Brominated vegetable oil (BVO)	Banned	Permitted (beverages)	Banned
Olestra/Olean	Not permitted	Permitted	Not permitted
Chlorine-washed chicken	Not permitted	Standard practice	Not standard
Atrazine (herbicide)	Banned	Widely used	Permitted with limits
Neonicotinoids (3 types)	Banned outdoor use (2018)	Permitted	Permitted
Formaldehyde in hair products	Banned	Permitted	Being phased out

These are not marginal disagreements about ambiguous data. These are cases where regulatory bodies, examining the same evidence base, reached opposite conclusions. The divergence is explained not by science but by structure: which system the regulator operates within, which burden of proof applies, and which interests are best resourced to influence the outcome.

Chapter 5: FSANZ – Australia's Regulatory Architecture

5.1 Structure and Mandate

Food Standards Australia New Zealand (FSANZ) is the bi-national statutory authority responsible for developing food standards for Australia and New Zealand. Established under the Food Standards Australia New Zealand Act 1991 (and successor legislation), FSANZ develops and maintains the Australia New Zealand Food Standards Code, which governs food composition, labelling, food safety, and the approval of food additives, processing aids, and novel foods.

FSANZ is not the same as the FDA. It does not enforce food standards – enforcement is the responsibility of state and territory governments in Australia, and of the New Zealand Ministry for Primary Industries. FSANZ develops the rules. Others enforce them. This separation creates an accountability gap: the body that writes the standards is not the body that monitors compliance, and the bodies that monitor compliance operate with varying levels of resourcing and political will across eight Australian jurisdictions.

5.2 The Approval Process

FSANZ's food additive approval process formally requires pre-market assessment. An applicant (typically the food manufacturer or ingredient supplier) submits an application that includes the identity and specification of the substance, its proposed use, and safety data. FSANZ conducts a risk assessment based on the submitted data, supplemented by its own literature review.

In principle, this sounds like a precautionary system. In practice, it has several structural weaknesses:

Applicant-supplied data. The safety evidence evaluated by FSANZ is predominantly generated and supplied by the applicant – the entity with a direct financial interest in approval. This is the same structural conflict that undermines the GRAS system, wrapped in a more formal procedural framework. FSANZ does not routinely commission independent safety studies. Its assessments rely on data produced by or for the parties seeking approval.

Limited independent research capacity. FSANZ is a small agency with limited scientific staff compared to the scope of its mandate. It regulates food standards for two countries with a combined population of approximately 30 million but does not have the in-house research capacity to independently verify the safety data submitted by applicants. It relies on literature review and risk assessment rather than independent experimental validation.

Reference to international assessments. FSANZ frequently references assessments conducted by JECFA (the Joint FAO/WHO Expert Committee on Food Additives) and other international bodies. While this leverages a broader evidence base, it also imports the limitations and industry-influence dynamics of those bodies. If JECFA's assessment is compromised by industry participation, FSANZ's reliance on that assessment imports the compromise.

Slow response to emerging evidence. When new evidence emerges suggesting that a previously approved substance may pose risks, the FSANZ review process is slow. Re-evaluation requires a formal proposal, public consultation, and Ministerial Council review. This process can take years, during which the substance remains in the food supply.

5.3 Australia-Specific Concerns

Several issues are specific to the Australian context:

Glyphosate residues. Australia is one of the largest users of glyphosate globally, both in agriculture and in domestic settings. FSANZ sets maximum residue limits (MRLs) for glyphosate in food, based on assessments that have been criticised for relying on industry-supplied data and for not adequately accounting for chronic low-dose exposure. The Australian Pesticides and Veterinary Medicines Authority (APVMA) regulates glyphosate use, but its assessments have similarly been criticised for industry alignment.

Ultra-processed food prevalence. Australia has one of the highest rates of ultra-processed food consumption in the world. Machado et al. (2019) found that ultra-processed foods account for approximately 42% of energy intake in the Australian diet. The Health Star Rating system – Australia's front-of-pack labelling scheme – has been criticised for allowing ultra-processed foods to receive high ratings by manipulating formulations to score well on the algorithm while remaining fundamentally ultra-processed.

No mandatory added sugar disclosure. Unlike several other jurisdictions, Australian food labelling does not require the disclosure of added sugars as a separate line item. Total sugars are listed, but the consumer cannot distinguish between naturally occurring sugars (in fruit, dairy) and added sugars. This is a labelling gap that obscures one of the most significant dietary risk factors.

Health Star Rating gaming. The Health Star Rating system, introduced in 2014 as a voluntary front-of-pack labelling scheme, was designed to help consumers identify healthier food choices. In practice, the algorithm can be gamed by manufacturers who add fibre or protein to otherwise nutritionally poor products to improve their star rating, or who reformulate minimally to cross a threshold. Nutri-Grain, a product that is essentially refined grains and sugar, has received a Health Star Rating of 4 out of 5. The system measures nutrients in isolation, not the degree of processing – and as the NIH trial (Hall et al., 2019) demonstrated, it is the degree of processing, not the macronutrient profile, that drives overconsumption.

5.4 What FSANZ Gets Right

It would be dishonest to present FSANZ as equivalent to the FDA's GRAS system. FSANZ does conduct pre-market assessment. It has banned several substances that the US permits (potassium bromate, BVO, rBGH). Its labelling requirements, while imperfect, are more comprehensive than US requirements. Its approach to novel foods includes mandatory pre-market approval.

But "better than the US" is a low bar. The question is not whether FSANZ is worse than the FDA. The question is whether FSANZ meets the standard that the precautionary principle requires: that no substance enters the food supply without independent, rigorous, pre-market demonstration of safety at the doses and durations at which it will actually be consumed. It does not.

5.5 Australian Dietary Disease Burden

The consequences of Australia's food regulatory approach are visible in the population health data:

- **Obesity:** 31.7% of Australian adults are obese, 35.6% are overweight. Two-thirds of the adult population is above healthy weight (ABS, 2022). The rate has been climbing steadily for four decades.

- **Type 2 diabetes:** 1.3 million Australians have diagnosed type 2 diabetes, with an estimated additional 500,000 undiagnosed. Prevalence has tripled since the 1980s (AIHW, 2022).
- **Cardiovascular disease:** Remains the leading cause of death in Australia, responsible for approximately 25% of all deaths (AIHW, 2022).
- **Colorectal cancer:** Australia has among the highest rates of colorectal cancer in the world – a cancer type specifically linked to processed meat (IARC Group 1) and ultra-processed food consumption.
- **Food allergy:** Australia has among the highest rates of childhood food allergy in the world, with hospital admissions for anaphylaxis increasing approximately 350% in the past two decades (Mullins et al., 2015). While the causes of this increase are debated, the temporal correlation with increasing food additive exposure and ultra-processed food consumption is notable.

These outcomes are not inevitable features of Australian life. They are the output of a food system. The food system is regulated by FSANZ. FSANZ permits substances in the food supply that have not been independently demonstrated to be safe at the doses at which they are consumed. The connection is not speculative. It is structural.

5.6 The APVMA and Pesticide Residues

Food safety in Australia is further complicated by the division of regulatory responsibility. While FSANZ sets standards for food composition and labelling, the Australian Pesticides and Veterinary Medicines Authority (APVMA) regulates agricultural chemicals, including pesticide residue limits in food.

The APVMA has been subject to sustained criticism for its proximity to the agricultural chemical industry. In 2014, the APVMA was controversially relocated from Canberra to the regional city of Armidale in New South Wales – a move widely criticised as politically motivated and which resulted in the loss of approximately 80% of its experienced scientific staff (Senate Rural and Regional Affairs and Transport References Committee, 2017). The rebuilding of institutional expertise has been slow.

The APVMA's pesticide reassessment programme – the process by which approved chemicals are periodically re-evaluated in light of new evidence – has been criticised as chronically under-resourced and slow. Chemicals that have been banned or restricted in the EU remain approved in Australia, in some cases years after international regulators have acted. The structural explanation is the same: the burden of proof falls on the regulator to demonstrate harm, and the regulator lacks the resources to do so on the timescale at which evidence accumulates.

Chapter 6: Ultra-Processed Food – The NOVA Classification and the NIH Trial

6.1 The NOVA Classification

The NOVA food classification system, developed by Carlos Monteiro and colleagues at the University of Sao Paulo, classifies foods not by nutrient content but by the nature and extent of processing. This represents a paradigm shift in nutritional science: from asking "what nutrients does this food contain?" to asking "what has been done to this food?"

NOVA defines four groups:

Group 1: Unprocessed or minimally processed foods. The edible parts of plants or animals, or foods altered by processes such as drying, crushing, grinding, roasting, boiling, pasteurization, or fermentation – processes that do not add substances but may remove parts of the food. Examples: fresh fruit, vegetables, eggs, meat, fish, milk, grains, legumes, nuts.

Group 2: Processed culinary ingredients. Substances extracted from Group 1 foods or from nature by processes such as pressing, refining, grinding, or milling. Used in kitchens to prepare Group 1 foods. Examples: oils, butter, sugar, salt, flour, starches.

Group 3: Processed foods. Products made by adding Group 2 ingredients to Group 1 foods, using preservation methods such as canning, bottling, or fermentation. Examples: canned vegetables, cheese, bread, cured meat.

Group 4: Ultra-processed foods. Industrial formulations made mostly or entirely from substances derived from foods and additives, with little if any intact Group 1 food. They are manufactured using a sequence of industrial processes (hence "ultra-processed"), many requiring sophisticated equipment and technology. They typically contain ingredients not found in domestic kitchens: hydrogenated oils, hydrolysed proteins, modified starches, emulsifiers, humectants, flavour enhancers, colourings, and other cosmetic additives. Examples: soft drinks, mass-produced bread and baked goods, reconstituted meat products, instant noodles, packaged snacks, ice cream, confectionery.

The NOVA distinction matters because it captures something that nutrient-level analysis misses: the overall character of the food as a product of industrial processing, including the presence of substances (emulsifiers, colourings, flavour enhancers, preservatives) that are not captured by macronutrient profiles.

6.2 The NIH Inpatient Trial

In 2019, Kevin Hall and colleagues at the National Institutes of Health published what may be the single most important nutrition experiment of the decade. It was a randomized, controlled, crossover feeding study – the gold standard of dietary research – published in *Cell Metabolism* (Hall et al., 2019).

Twenty adults were admitted to the NIH Clinical Center for four weeks. For two weeks, they ate a diet of ultra-processed foods (as defined by NOVA). For two weeks, they ate a diet of minimally processed foods. The diets were matched for presented calories, macronutrient composition, sugar, fat, sodium, and fibre. The only variable was the degree of processing.

The result: participants eating ultra-processed food consumed approximately 500 more calories per day and gained approximately 1 kg over two weeks. On the minimally processed diet, they ate less and lost approximately 1 kg. Same people, same available nutrition, radically different outcomes.

The mechanism is not fully understood. The participants did not report the ultra-processed food as more palatable. They ate faster on the ultra-processed diet – approximately 50 calories per minute versus 35 on the minimally processed diet. The current hypotheses include: altered satiety signalling (the body does not register ultra-processed calories the same way), differences in food texture affecting eating rate, and disruption of gut-brain communication by emulsifiers and other processing agents.

What makes this study powerful is its design. It is not observational. It is not a food frequency questionnaire. It is a controlled, inpatient feeding study where every meal was provided and every calorie was tracked. The 500-calorie-per-day difference was not a statistical artefact. It was a measured, replicated, consistent effect.

6.3 Epidemiological Evidence

The Hall trial sits within a broader epidemiological literature that consistently associates ultra-processed food consumption with adverse health outcomes:

- **Cancer:** Fiolet et al. (2018), in the NutriNet-Sante cohort (n=104,980), found that a 10% increase in ultra-processed food consumption was associated with a 12% increase in overall cancer risk and an 11% increase in breast cancer risk.
- **Cardiovascular disease:** Srour et al. (2019), in the same cohort, found that ultra-processed food consumption was associated with increased risk of cardiovascular disease, coronary heart disease, and cerebrovascular disease.
- **All-cause mortality:** Rico-Campa et al. (2019), in the SUN cohort (n=19,899), found that consumption of more than four servings per day of ultra-processed food was associated with a 62% increase in all-cause mortality.
- **Depression:** Adjibade et al. (2019) found that higher ultra-processed food consumption was associated with increased depressive symptoms.
- **Obesity:** Mendonca et al. (2016) found that high ultra-processed food consumption was associated with a 26% increased risk of developing overweight or obesity over a median 8.9-year follow-up.

These are observational studies, and individual studies have the limitations inherent to observational epidemiology. But the consistency of the association – across multiple cohorts, multiple countries, multiple health outcomes, and now supported by the Hall RCT – makes the case for a causal relationship between ultra-processed food consumption and chronic disease substantially stronger than the case against it.

6.4 The Australian Ultra-Processed Diet

Australia is not an outlier in ultra-processed food consumption. It is a typical case. Machado et al. (2019), analysing data from the Australian Health Survey, found that ultra-processed foods contributed 42% of total energy intake in Australian adults, with the figure rising to approximately 50% in children and adolescents. The most consumed ultra-processed products were industrialised bread, confectionery, biscuits, pastries, cakes, soft drinks, and reconstituted meat products.

The Australian dietary guidelines, while recommending "whole foods" in principle, do not use the NOVA classification and do not address ultra-processing as a distinct dietary risk factor. The concept of ultra-processing is absent from FSANZ's regulatory framework. Australia regulates individual additives in isolation but does not regulate the overall degree of food processing. This is like regulating the ingredients of cigarettes one chemical at a time while ignoring the act of smoking.

Chapter 7: The Substances – What Is in Your Food and Who Put It There

7.1 Additives That Give You Nothing

Here is the part that should make you angry.

If these additives made your food taste better – if they made it more nutritious, more satisfying, more nourishing – you could at least understand the trade-off. Risky but delicious. Dangerous but essential.

They do not. They give you nothing.

Artificial colourings exist to make food look brighter. That is it. They do not affect taste. They do not affect nutrition. They make the red redder and the yellow yellower so it looks more appealing on the shelf. Preservatives extend shelf life. Not your life. Shelf life. Emulsifiers change texture. They make things smoother, creamier, more uniform. The benefit in every case is to the supply chain, not to you.

Would you believe the potentially carcinogenic ingredients give you nothing? Nothing by way of taste or experience. Nothing. It is not like you are eating something dangerous but delicious. Or dangerous but nutritious. No. These are additives that contribute absolutely zero to the experience of eating and actively damage the body. And they are permitted because someone found it cheaper to include them than to not.

7.2 The Additive Count

The number of substances permitted in food varies by jurisdiction but is measured in the thousands. In the United States, approximately 10,000 substances are permitted in food, including direct additives, colour additives, GRAS substances, and substances used in food contact materials (Neltner et al., 2011). The EU's positive list contains approximately 350 approved food additives, but this does not include processing aids, flavourings (approximately 2,500 permitted), or food contact materials. FSANZ's Food Standards Code permits a comparable range of additives, with the list maintained through Schedule 15 (Substances that may be used as food additives) of the Code.

The proliferation is one-directional. Substances are added to the list far more frequently than they are removed. The ratchet effect described in Chapter 2 operates continuously: each addition faces low barriers, each removal faces high ones. The result is a food supply that grows more chemically complex over time, with the cumulative and combinatorial effects of this complexity never assessed.

7.3 Specific Substances of Concern

Titanium dioxide (E171). A whitening agent used in confectionery, chewing gum, bakery products, and sauces. Classified as "possibly carcinogenic to humans" (Group 2B) by IARC. The EU banned it in food in 2022 following an EFSA reassessment that concluded genotoxicity could not be ruled out (EFSA, 2021). It remains permitted in the United States and Australia. Its function in food is purely cosmetic: it makes things white. It contributes nothing to nutrition, taste, or safety.

Potassium bromate. A flour treatment agent that strengthens dough and allows higher rising. Classified as a possible human carcinogen (Group 2B) by IARC. Banned in the EU, UK, Canada,

Brazil, and Australia. Still permitted in the United States, where it is used in bread and other baked goods. California passed a law in 2023 (the California Food Safety Act) banning potassium bromate effective 2027 – the first US state to act. The fact that a state legislature had to pass a law banning a substance the FDA permits tells you everything about the federal regulatory system.

BHA (butylated hydroxyanisole). An antioxidant preservative used in cereals, chewing gum, butter, dehydrated potatoes, and beer. Listed as "reasonably anticipated to be a human carcinogen" by the US National Toxicology Program. Restricted in the EU. Permitted and widely used in the United States. Permitted in Australia. It exists in your food so the product can sit on a shelf for months without the manufacturer losing money.

Azodicarbonamide. A bleaching agent and dough conditioner used in bread and flour products. Banned in the EU and Australia (where its ban is partial – permitted in some applications). Permitted in the US. When heated, azodicarbonamide breaks down into semicarbazide, a compound with potential carcinogenic properties, and urethane, a known carcinogen. Its industrial application is as a foaming agent in the production of plastics and rubber. It is also in your sandwich bread.

Artificial food dyes (Red 40, Yellow 5, Yellow 6, and others). Petroleum-derived colourings that serve no nutritional purpose. Linked to behavioural effects in children (McCann et al., 2007). Required to carry warning labels in the EU. No warning required in the US or Australia. Australia permits their use under Schedule 15 of the Food Standards Code without mandatory warnings. The same substances, the same evidence, different regulatory responses – and the responses diverge along predictable lines: the jurisdiction with stronger consumer protection defaults to labelling; the jurisdictions with stronger industry ties do not.

Ractopamine. A beta-agonist drug used in livestock production to promote lean muscle growth. Banned in 160 countries, including the EU, China, and Russia. Permitted in the United States and Australia. Concerns relate to cardiovascular effects and the adequacy of the safety data upon which approval was based. Codex Alimentarius adopted a standard for ractopamine in 2012 by a narrow vote of 69-67, illustrating the deep division in the international scientific community about its safety.

Chapter 8: Glyphosate – The Most-Used Herbicide on Earth

8.1 The Scale of Exposure

Glyphosate (N-(phosphonomethyl)glycine) is the most widely used herbicide in human history. Global use has increased approximately 15-fold since the introduction of genetically engineered "Roundup Ready" crops in 1996 (Benbrook, 2016). In the United States alone, approximately 280 million pounds of glyphosate are applied annually. In Australia, glyphosate is the most commonly used herbicide in both agricultural and domestic settings, with millions of kilograms applied annually.

Glyphosate residues are detectable in food, water, and human urine at a population level. A 2017 study found glyphosate in 93% of urine samples tested in an American cohort (Mills et al., 2017). This is not an occupational exposure. It is a dietary and environmental exposure affecting the general population.

8.2 The IARC Classification and Regulatory Divergence

In 2015, the International Agency for Research on Cancer (IARC) classified glyphosate as "probably carcinogenic to humans" (Group 2A), based on "sufficient evidence" of carcinogenicity in experimental animals and "limited evidence" in humans (specifically, associations with non-Hodgkin lymphoma in epidemiological studies) (IARC, 2015).

The response from regulatory agencies diverged sharply:

- **IARC (WHO):** Probably carcinogenic to humans (Group 2A).
- **EFSA (EU):** Unlikely to pose a carcinogenic hazard to humans. However, the EU's renewal of glyphosate's approval in 2023 was contentious, passing by a narrow margin and with several member states voting against or abstaining.
- **US EPA:** Not likely to be carcinogenic to humans. This conclusion has been challenged by internal EPA scientists and by litigation that revealed Monsanto's (now Bayer's) influence over the regulatory process.
- **APVMA (Australia):** Not a carcinogenic risk to humans at expected exposure levels. APVMA's assessment has been criticised for relying heavily on industry-supplied data.
- **Germany's BfR (which conducted the EU assessment):** Not carcinogenic. However, it was subsequently revealed that portions of the BfR's assessment were copied from Monsanto's own submission, raising questions about the independence of the review (Portier et al., 2016).

8.3 The Monsanto Papers

Court proceedings in US litigation (the Roundup cancer lawsuits) resulted in the release of internal Monsanto documents – the "Monsanto Papers" – that revealed:

- Monsanto ghostwrote scientific papers that were then published under the names of academic researchers, with the company's role undisclosed (Baum Hedlund, 2017).
- Monsanto had a programme to influence EPA reviewers and to discredit IARC's classification.
- Internal communications showed Monsanto scientists expressing private concerns about the carcinogenic potential of glyphosate that contradicted the company's public position.
- Monsanto sought to prevent independent testing and to shape the scientific literature through strategic funding and publication management.

These documents do not prove that glyphosate causes cancer. They prove that the regulatory process through which glyphosate was evaluated was compromised by the manufacturer's deliberate interference. The safety determination is unreliable because the process that produced it was not independent.

8.4 The Honest Position on Glyphosate

The evidence on glyphosate carcinogenicity is genuinely contested. Reasonable scientists disagree. The IARC classification is based on sound methodology but limited human evidence. The regulatory agency conclusions that glyphosate is "not likely" carcinogenic are based on assessments whose independence has been credibly questioned.

What is not contested is this: the burden of resolving this uncertainty currently falls on the exposed population. Billions of people are consuming glyphosate residues in food while the scientific community argues about whether the evidence of harm is sufficient to warrant action. Under a

precautionary framework, the burden would fall on the proponent: if the safety of glyphosate cannot be conclusively demonstrated – and it cannot, given the legitimate scientific disagreement – it should not be in the food supply until it can.

8.5 Glyphosate and the Gut Microbiome

Beyond the carcinogenicity debate, a growing body of evidence suggests that glyphosate may affect human health through disruption of the gut microbiome. Glyphosate's mechanism of herbicidal action is inhibition of the shikimate pathway, a metabolic pathway absent in animals but present in many bacteria – including gut bacteria. Mao et al. (2018) demonstrated that glyphosate at environmentally relevant concentrations altered the composition and metabolic activity of the gut microbiome in animal models. Aitbali et al. (2018) found that chronic glyphosate exposure in mice produced anxiety- and depression-like behaviours associated with changes in gut microbiota composition.

These findings are preliminary and require human confirmation. But they illustrate a broader point: the safety assessment of glyphosate was conducted on the basis of direct toxicology (does the chemical damage human cells?) and did not adequately consider indirect effects mediated through the microbiome. This is a systemic problem in food safety assessment: the regulatory framework evaluates direct toxicity to human cells but not the effects of food substances on the microbial ecosystem within the human body that is now understood to play a central role in immune function, metabolism, and neurological health.

Chapter 9: Seed Oils – What the Evidence Actually Says

9.1 The Debate

The debate over seed oils – specifically, whether refined vegetable oils high in linoleic acid (omega-6 polyunsaturated fatty acid) are harmful – is one of the most contested areas in nutrition. Social media has amplified the debate to the point where "seed oils are toxic" is a meme and "seed oils are fine" is a counter-meme. Neither captures the state of the evidence.

9.2 The Case Against Seed Oils

Proponents of seed oil avoidance make several arguments:

Evolutionary novelty. Refined seed oils (soybean oil, canola oil, sunflower oil, corn oil, safflower oil, cottonseed oil) are extracted through industrial processes (solvent extraction, degumming, bleaching, deodorization) that did not exist before the twentieth century. Human consumption of linoleic acid has increased dramatically – from approximately 2-3% of total calories in pre-industrial diets to approximately 7-8% in the modern Western diet (Blasbalg et al., 2011). This represents a 2-3 fold increase in a specific fatty acid over a period too short for evolutionary adaptation.

Omega-6 to omega-3 ratio. The increase in linoleic acid consumption has shifted the dietary omega-6 to omega-3 ratio from approximately 1:1 to 4:1 in ancestral diets to approximately 15:1 to 20:1 in modern Western diets (Simopoulos, 2002). Omega-6 fatty acids are precursors to pro-inflammatory eicosanoids (prostaglandins, thromboxanes, leukotrienes), while omega-3 fatty acids

are precursors to anti-inflammatory and inflammation-resolving mediators. An elevated omega-6 to omega-3 ratio may therefore promote a pro-inflammatory state.

Oxidation. Polyunsaturated fatty acids are chemically unstable and prone to oxidation, particularly during high-temperature cooking. Oxidised lipids are biologically active and have been implicated in atherosclerosis, inflammation, and cellular damage. The industrial processing of seed oils involves high temperatures that may initiate or promote lipid oxidation.

9.3 The Case That Seed Oils Are Fine

Opponents of the seed oil concern make several counter-arguments:

Clinical trial evidence. Several randomized controlled trials have examined the effect of replacing saturated fat with linoleic acid-rich vegetable oils. The overall evidence, as synthesised in meta-analyses, is mixed. Some trials (e.g., the Finnish Mental Hospital Trial) found reduced cardiovascular events with vegetable oil substitution. Others (e.g., the Sydney Diet Heart Study, re-analysed by Ramsden et al., 2013) found increased mortality with increased linoleic acid intake. The totality of the RCT evidence does not support a clear causal relationship between linoleic acid consumption and cardiovascular harm or benefit.

Observational evidence. Large observational studies generally find neutral or modestly protective associations between linoleic acid intake and cardiovascular disease risk (Farvid et al., 2014). However, observational studies in nutrition are subject to well-known confounders and limitations.

Mechanistic complexity. The omega-6 to omega-3 ratio hypothesis, while biologically plausible, may be oversimplified. The metabolic fate of dietary linoleic acid is complex, and the in vivo inflammatory effects depend on numerous factors including absolute intake of both omega-6 and omega-3 fatty acids, overall dietary context, and individual metabolic variation.

9.4 The Honest Position

The honest position on seed oils is that the evidence is genuinely mixed and the question is genuinely open. The strongest claims on both sides come from social media, not from systematic reviews.

What is not debatable is this: refined seed oils are an evolutionary novelty consumed at doses dramatically exceeding any prior human exposure. They have entered the food supply without long-term safety data. The shift from traditional fats (animal fats, olive oil, coconut oil, butter) to industrially refined seed oils represents one of the largest dietary changes of the twentieth century, and it was driven not by evidence of health benefit but by cost – seed oils are cheap to produce at industrial scale.

Under a precautionary framework, this would be sufficient to require a rigorous safety demonstration before population-wide adoption. That demonstration has not occurred. The population adopted the product first. The safety debate came after. This is the pattern described throughout this paper: add it, sell it, argue about it for decades, and let the population absorb the risk of the uncertainty.

Chapter 10: Food Contact Materials – The Packaging Problem

10.1 The Overlooked Exposure

The food itself is not the only concern. The packaging matters.

Food contact materials – the containers, wraps, coatings, adhesives, inks, and surfaces that food touches during production, packaging, storage, and preparation – are a source of chemical migration into food. The substances that migrate include plasticizers, stabilizers, antioxidants, monomers, catalysts, solvents, and printing inks. The total number of substances used in food contact materials is estimated at over 12,000, of which the majority have never been assessed for safety (Muncke et al., 2020).

10.2 BPA (Bisphenol A)

Bisphenol A is a synthetic oestrogen used in the production of polycarbonate plastics and epoxy resin linings (used in food and beverage cans). BPA migrates from containers into food, particularly when heated.

Rochester (2013) published a comprehensive review in *Reproductive Toxicology* examining epidemiological evidence on BPA exposure and human health. The analysis found consistent associations between BPA exposure and reproductive disorders, metabolic dysfunction (type 2 diabetes, obesity), cardiovascular disease, and thyroid dysfunction. Effect sizes were modest but consistent across studies. BPA was detectable in the urine of over 90% of the US population, indicating near-universal exposure.

The regulatory response has been characteristically fragmented:

- **France:** Banned BPA in food contact materials in 2015.
- **Canada:** Declared BPA toxic in 2010 and banned it from baby bottles.
- **EU:** Banned BPA in baby bottles (2011) and set migration limits for other food contact materials. In 2023, EFSA dramatically lowered the tolerable daily intake for BPA by a factor of 20,000, indicating that prior assessments had substantially underestimated the risk.
- **Australia:** FSANZ "concluded that BPA does not pose a health risk to any age group at estimated dietary exposures." BPA is permitted in food contact materials with no specific migration limits for adults.
- **US FDA:** "Has not recommended that anyone discontinue using products that contain BPA."

The EFSA reassessment is particularly significant. When EFSA – generally considered the most rigorous of the major food safety regulators – lowers its tolerable daily intake by a factor of 20,000, it is acknowledging that the previous assessment was wrong by four orders of magnitude. This is not a minor recalibration. It is an admission that a substance permitted in food contact materials for decades was far more dangerous than the regulatory system had determined. During those decades, the population was exposed.

10.3 PFAS ("Forever Chemicals")

Per- and polyfluoroalkyl substances (PFAS) are a class of over 4,700 synthetic chemicals characterised by strong carbon-fluorine bonds that make them resistant to heat, water, oil, and degradation. They are used in non-stick cookware (Teflon), food packaging (microwave popcorn bags, fast

food wrappers, pizza boxes, takeaway containers), water treatment, and numerous other applications.

PFAS are called "forever chemicals" because they do not break down in the environment or in the human body. They bioaccumulate in human tissue over a lifetime. PFAS contamination of drinking water and food is widespread and effectively universal in industrialised populations.

The health effects of PFAS exposure are documented and serious:

- **Immune suppression:** Grandjean et al. (2012) demonstrated that PFAS exposure in children was associated with reduced antibody response to vaccines – a functional measure of immune system impairment.
- **Thyroid disease:** PFAS exposure is associated with thyroid dysfunction, particularly in women (Webster et al., 2014).
- **Cancer:** PFOA (a specific PFAS compound) was classified as a Group 1 carcinogen (carcinogenic to humans) by IARC in 2023. PFOS was classified as Group 2B (possibly carcinogenic).
- **Reproductive effects:** PFAS exposure is associated with reduced fertility, pregnancy-induced hypertension, and altered birth weight (Bach et al., 2015).
- **Liver damage:** PFAS exposure is associated with elevated liver enzymes and non-alcoholic fatty liver disease (Bassler et al., 2019).

PFAS restrictions are emerging but lag decades behind the evidence of harm. The EU is developing a comprehensive PFAS restriction proposal. The US EPA has set advisory health levels for PFAS in drinking water, but enforcement is patchy. Australia's PFAS National Environmental Management Plan provides guidelines but not enforceable limits for PFAS in food.

10.4 Phthalates

Phthalates are plasticizers used to make plastics flexible. They migrate from food packaging, tubing used in food processing, and plastic wraps into food. Di(2-ethylhexyl) phthalate (DEHP) and other phthalates are endocrine disruptors – they interfere with androgen signalling and are associated with reproductive abnormalities, metabolic dysfunction, and developmental effects.

Zota et al. (2016) found that dining out was associated with significantly higher urinary phthalate metabolite levels, suggesting that food preparation in restaurants and fast food settings involves substantial phthalate contamination from food handling equipment.

The cumulative exposure to endocrine-disrupting chemicals from food contact materials – BPA, PFAS, phthalates, and others – represents an exposure pathway that is not captured by food additive regulation. The food may be evaluated for its additive content, but the container it comes in migrates unregulated chemicals into the food before it reaches the consumer.

Chapter 11: Emulsifiers and the Gut Microbiome

11.1 The Discovery

Benoit Chassaing and colleagues at Georgia State University published a series of papers beginning in 2015 that fundamentally altered understanding of how common food additives interact with human biology. The initial paper, published in *Nature* (Chassaing et al., 2015), demonstrated that

two of the most common food emulsifiers – polysorbate 80 and carboxymethylcellulose (CMC) – promote intestinal inflammation and alter gut microbiota composition.

11.2 The Mechanism

The emulsifiers, at concentrations below those commonly used in food, eroded the mucus layer lining the intestine, allowed bacteria to penetrate closer to the epithelial surface, and promoted low-grade inflammation and metabolic syndrome. Follow-up studies showed altered gut bacterial composition and increased expression of inflammatory markers.

The mucus layer is the gut's primary defence barrier – a physical separation between the trillions of bacteria in the gut lumen and the single-cell-thick epithelial lining that separates the gut contents from the bloodstream. When emulsifiers thin this layer, bacteria that are normally kept at a safe distance come into contact with immune cells in the gut wall, triggering an inflammatory response.

This chronic, low-grade inflammation – distinct from the acute inflammation of infection or injury – is now recognised as a central feature of the pathogenesis of metabolic syndrome, type 2 diabetes, cardiovascular disease, and inflammatory bowel disease.

11.3 Where These Emulsifiers Are

Polysorbate 80 and CMC are in ice cream, salad dressing, bread, sauces, mayonnaise, chocolate, and dozens of other processed foods. They are there because they improve texture and extend shelf life – benefits to the supply chain, not to the consumer. They keep oil and water from separating. They make things creamier. They prevent crystallization. Every one of these functions serves the manufacturer. None of them serves you.

11.4 The Human Evidence Gap

The critical caveat: Chassaing et al.'s findings are primarily from mouse models. Mouse gut biology is not identical to human gut biology. The doses, while realistic in food-supply terms, require human confirmation. Several human trials are now underway, including a randomized controlled trial by Chassaing's group examining the effects of CMC on the human gut microbiome (Chassaing et al., 2022, registered at ClinicalTrials.gov).

Preliminary results from human studies support the animal findings. Chassaing et al. (2022) reported that CMC consumption in healthy volunteers was associated with changes in gut microbiome composition and reduced microbial diversity, although the clinical significance of these changes over short study durations remains to be established.

But the precautionary point stands: these substances were introduced into the food supply without evidence they were safe for the gut microbiome, and the evidence that has accumulated since suggests they are not. A zookeeper designing a diet for a primate would flag them as a gut irritant. Nobody flags them for humans.

11.5 Artificial Sweeteners

The gut microbiome story extends beyond emulsifiers. Suez et al. (2014), published in *Nature*, demonstrated that artificial sweeteners – saccharin, sucralose, and aspartame – altered the gut microbiome in ways that impaired glucose tolerance. The substances marketed as "safe" alternatives to sugar were actively worsening the metabolic parameter they were marketed as protecting.

A follow-up study by the same group (Suez et al., 2022), published in *Cell*, confirmed in a randomized controlled human trial that saccharin and sucralose significantly altered the human gut microbiome and glycaemic responses. The changes were person-specific but measurable and functional.

These substances were introduced as safe without long-term microbiome assessment. They are consumed daily by hundreds of millions of people. The safety was assumed, not demonstrated.

Chapter 12: Food Dyes and Children's Behaviour

12.1 The Southampton Study

In 2007, Donna McCann and colleagues at the University of Southampton published a study in *The Lancet* that tested whether artificial food colourings and the preservative sodium benzoate affected children's behaviour (McCann et al., 2007). The study was a randomized, double-blind, placebo-controlled trial – methodologically rigorous, commissioned by the UK Food Standards Agency, and published in one of the world's most prestigious medical journals.

Two age groups of children (3-year-olds and 8/9-year-olds) were given drinks containing artificial colour mixtures or a placebo over six weeks. The study found that artificial colours increased hyperactive behaviour in both age groups, with the effect size being clinically meaningful.

12.2 Two Responses to the Same Evidence

The EU responded by requiring warning labels on foods containing the six dyes tested – the "Southampton Six": Sunset Yellow (E110), Quinoline Yellow (E104), Carmoisine (E122), Allura Red (E129), Tartrazine (E102), and Ponceau 4R (E124). The warning reads: "May have an adverse effect on activity and attention in children."

The US FDA reviewed the same data. In 2011, an FDA advisory committee voted 8-6 that the evidence did not support a causal link and that no warning was necessary. The dyes remain in US food without warning labels.

Australia's position is closer to the US than the EU. FSANZ has not required warning labels for any of the Southampton Six dyes. They are permitted under Schedule 15 of the Food Standards Code. Australian parents feeding their children confectionery, soft drinks, or processed snacks containing these dyes receive no regulatory warning that the dyes may affect their child's behaviour.

12.3 What the Dyes Contribute

Same study. Same data. Different regulatory conclusions. The divergence is not scientific. It is structural.

And here is the part that should be central to every parent's assessment: these dyes contribute nothing to nutrition, nothing to taste, nothing to food safety. They exist to make food a colour it is not. They make the red redder and the yellow yellower so the product looks more appealing on the shelf and more appealing to children. They are there to sell the product. They are not there for you.

The substances that may be making children hyperactive are in the food because they make the food look brighter. That is the trade-off. That is what the regulatory system, in the US and Australia, has decided is acceptable: a cosmetic benefit to the manufacturer in exchange for a potential behavioural cost to children.

Chapter 13: Advanced Glycation End-Products – The Mechanistic Pathway

13.1 The AGE Mechanism

Advanced Glycation End-products (AGEs) provide a specific, well-characterised mechanistic pathway linking processed food to chronic disease. AGEs are formed through the Maillard reaction – a non-enzymatic chemical reaction between reducing sugars and amino acids, proteins, or lipids that occurs during high-temperature cooking (frying, grilling, roasting, baking) and in the industrial processing of food (Uribarri et al., 2010).

The Maillard reaction is what produces the browning, flavour, and aroma compounds associated with cooked and processed food. It is also what produces a class of compounds that are biologically active, pro-inflammatory, pro-oxidant, and causally implicated in the pathogenesis of multiple chronic diseases.

13.2 Mechanisms of Harm

AGEs exert pathological effects through several established mechanisms:

RAGE activation. AGEs bind to the Receptor for Advanced Glycation End-products (RAGE), a pattern-recognition receptor expressed on endothelial cells, macrophages, smooth muscle cells, and neurons. RAGE activation triggers intracellular signalling cascades – primarily through NF-kB – that produce chronic inflammation, oxidative stress, and cellular dysfunction (Bierhaus et al., 2005).

Protein cross-linking. AGEs cause irreversible cross-linking of structural proteins, particularly collagen and elastin. This cross-linking contributes to arterial stiffness, loss of vascular compliance, basement membrane thickening, and the progressive loss of tissue function associated with ageing and chronic disease (Sell & Monnier, 2012).

Oxidative stress. AGE-RAGE interaction generates reactive oxygen species (ROS) through NADPH oxidase activation, contributing to systemic oxidative stress (Cai et al., 2008).

Inflammation. AGE-mediated NF-kB activation drives the production of pro-inflammatory cytokines (TNF-alpha, IL-6, IL-1beta, CRP), creating a state of chronic low-grade inflammation – the same inflammatory state implicated in cardiovascular disease, type 2 diabetes, cancer, neurodegeneration, and autoimmune conditions (Vlassara & Striker, 2011).

13.3 Dietary AGE Exposure

The dietary AGE burden of modern processed food is orders of magnitude higher than that of traditional, minimally processed diets. Uribarri et al. (2010) compiled a comprehensive database of AGE content across 549 commonly consumed foods:

- Dry-heat cooking methods (frying, grilling, broiling, roasting) produce AGE levels 10-100 times higher than wet-heat methods (boiling, steaming, poaching).
- Processed foods – particularly processed meats, fried foods, baked goods, and industrial snacks – contain AGE levels vastly exceeding those of whole, unprocessed foods.
- The typical Western diet delivers an estimated 15,000-25,000 kU of AGEs per day, while diets composed of minimally processed foods cooked at low temperatures deliver approximately 5,000-8,000 kU per day.
- The traditional diets consumed by populations with low chronic disease rates – boiled tubers, steamed vegetables, raw fruit, poached fish – are precisely the diets that minimise AGE formation.

13.4 Clinical Evidence

Intervention studies have demonstrated that reducing dietary AGE intake produces measurable improvements:

- Vlassara et al. (2009) showed that a low-AGE diet reduced circulating AGE levels, inflammatory markers (CRP, TNF-alpha), and oxidative stress markers in both healthy subjects and patients with diabetes.
- Uribarri et al. (2011) demonstrated that dietary AGE restriction improved insulin sensitivity and reduced inflammatory markers in overweight individuals.
- Cai et al. (2012) showed in a mouse model that dietary AGE restriction extended lifespan and reduced the incidence of cardiovascular disease, diabetes, and kidney disease.

AGEs do not explain everything. But they provide a concrete, measurable, mechanistically well-characterised example of how the processing of food creates novel compounds that drive disease. They are a smoking gun – not the only weapon, but one whose ballistics are well understood.

Chapter 14: Endocrine Disruptors in the Food Chain

14.1 The Scope of the Problem

Endocrine-disrupting chemicals (EDCs) are substances that interfere with hormone signalling at low doses – often at doses below those used in traditional toxicological testing. The food chain is a primary route of human exposure to EDCs, both through food itself (pesticide residues, veterinary drug residues, naturally occurring phytoestrogens) and through food contact materials (BPA, phthalates, PFAS, as discussed in Chapter 10).

The Endocrine Society – the world's largest professional body of endocrinologists – has issued two Scientific Statements on EDCs (2009 and 2015) concluding that the evidence for adverse health effects of EDC exposure is strong and that current regulatory frameworks are inadequate to protect public health (Gore et al., 2015).

14.2 Non-Monotonic Dose-Response

One of the most significant challenges EDCs pose to traditional toxicology is non-monotonic dose-response. Traditional toxicology assumes that higher doses produce greater effects ("the dose makes the poison" – Paracelsus). Many EDCs do not follow this pattern. They produce effects at low

doses that are absent at higher doses, or they produce different effects at different doses. This means that safety assessments based on high-dose animal studies and extrapolation downward may fundamentally underestimate risks at the low doses to which the general population is actually exposed.

Vandenberg et al. (2012), in a comprehensive review in *Endocrine Reviews*, documented non-monotonic dose-response curves for BPA, atrazine, dioxin, and multiple other EDCs. The implication for food safety regulation is profound: the standard toxicological approach of establishing a No Observable Adverse Effect Level (NOAEL) at high doses and applying safety factors to derive "acceptable daily intake" may not produce safe exposure levels for EDCs.

14.3 Cumulative and Combinatorial Effects

The human body is not exposed to one endocrine disruptor at a time. It is exposed to dozens simultaneously, through food, water, air, and consumer products. The cumulative and combinatorial effects of these exposures are almost entirely uncharacterised.

Regulatory frameworks evaluate substances individually. They assess whether BPA at a given dose is safe, whether PFAS at a given dose is safe, whether this phthalate at a given dose is safe. They do not assess what happens when a person is exposed to BPA, PFAS, three phthalates, two pesticides, and four food additives simultaneously – which is the actual exposure scenario for every person in an industrialised country.

This is the biggest gap in food toxicology: the combinatorial effect of dozens of low-dose exposures, over decades, in real-world conditions. The studies have not been done because the regulatory system does not require them. The system evaluates substances one at a time and assumes that if each individual substance is "safe" at its individual exposure level, the combination is also safe. This assumption has no evidentiary basis.

Chapter 14A: The Pattern of Historical Failures

Before examining the evidence from traditional populations, it is worth pausing to observe a pattern. The food regulatory system has a history. That history is a record of delayed action, preventable harm, and the same structural failure repeating itself across decades.

14A.1 Lead

Lead was used as a food additive and food contact material for centuries. Lead acetate – "sugar of lead" – was used as a sweetener in wine from Roman times through the eighteenth century. Lead solder was used in food cans until the 1990s. Lead paint contaminated food preparation surfaces. Lead pipes carried drinking water.

The toxicity of lead was known in antiquity. Vitruvius warned about lead water pipes in the first century BCE. The evidence accumulated for two thousand years. Regulations came slowly, grudgingly, against sustained industry resistance. The lead paint industry fought restrictions for decades. The leaded gasoline industry – led by General Motors, Standard Oil, and DuPont – fought the removal of tetraethyl lead from petrol for over fifty years, using tactics that would later be replicated by the tobacco, food, and chemical industries: funding favourable research, attacking

independent scientists, emphasising uncertainty, and lobbying against regulation (Markowitz & Rosner, 2002).

The lesson: industry knew. Regulators eventually acted. The gap between knowledge and action was measured in decades and millions of poisoned people. The burden fell on the exposed population.

14A.2 Trans Fats

Partially hydrogenated oils – trans fats – were introduced into the food supply in the early twentieth century. Evidence linking trans fat consumption to cardiovascular disease began accumulating in the 1990s. The mechanism was well understood: trans fats raise LDL cholesterol, lower HDL cholesterol, promote inflammation, and impair endothelial function.

Regulatory action in the United States was not finalised until 2015, with a compliance date of 2018. During the intervening two-plus decades, the American population continued to consume trans fats. Mozaffarian et al. (2006) estimated that eliminating trans fats from the US food supply would prevent 10,000-20,000 heart attacks and 3,000-7,000 coronary deaths per year. Multiply that by the years of regulatory delay: the number of preventable deaths attributable to the delay is measured in tens of thousands.

The trans fat case is the paradigm. Same pattern: substance introduced, sold for decades, evidence of harm accumulates, industry disputes evidence, regulatory delay, eventual action – and throughout the entire process, the population absorbs the risk. By the time regulatory action comes, the damage has been done.

14A.3 Thalidomide

Thalidomide was marketed as a safe sedative in the late 1950s and early 1960s. It was available over the counter in many countries, including Australia and throughout Europe. It was specifically marketed to pregnant women for morning sickness.

Thalidomide caused severe birth defects – phocomelia (limb malformation) – in an estimated 10,000-20,000 children worldwide. The drug was withdrawn in 1961, approximately four years after evidence of teratogenicity began emerging.

The US was largely spared because an FDA reviewer – Frances Kelsey – refused to approve the drug, citing insufficient safety data. Kelsey's refusal was an act of individual precaution within a system that otherwise would have permitted the drug. She applied the precautionary principle before the phrase existed in regulatory vocabulary. The rest of the world did not.

Australia was not spared. Thalidomide was sold in Australia from 1958 to 1962. An estimated 40-50 Australian children were born with thalidomide-related birth defects.

The thalidomide case is instructive not because it involves food (it does not) but because it illustrates what happens when a substance that enters the human body is approved without adequate pre-market safety demonstration. The food additive system makes this error every day. It just does it more slowly, with longer latency periods, and with harm that is harder to attribute to a single substance – which means the regulatory system takes even longer to act, if it acts at all.

14A.4 Melamine in Infant Formula

In 2008, melamine – an industrial chemical used in the production of plastics and laminates – was found to have been deliberately added to infant formula and dairy products in China. The melamine was added to artificially inflate the apparent protein content of watered-down milk. An estimated 300,000 infants were affected. Six infants died. Approximately 54,000 were hospitalised with kidney damage.

The melamine scandal was a case of deliberate adulteration, not inadvertent contamination. But it exposed the limits of post-market surveillance: the contaminated formula was consumed for months before the health effects became apparent and the source was identified. A pre-market testing regime that independently verified the composition of food products – rather than relying on manufacturer declarations – would have detected the adulteration before it reached consumers.

14A.5 The Pattern

Lead. Trans fats. Thalidomide. Melamine. BSE (mad cow disease). Bisphenol A. PFAS. The pattern is the same in every case:

1. A substance is introduced into human exposure without adequate safety demonstration.
2. The population is exposed for years or decades.
3. Evidence of harm accumulates.
4. The industry that profits from the substance disputes the evidence.
5. Regulatory action is delayed while the evidence is "debated."
6. Eventually, action is taken – partial, incremental, years too late.
7. The damage has already been done.

This is not a series of unfortunate exceptions. This is the system working as designed. The system is designed to permit first and evaluate later. The evaluation comes, when it comes, after the damage. The damage is the data. The population is the experiment.

Goal 10 exists because this pattern is unacceptable. The fix is not better post-market surveillance. The fix is pre-market proof of safety. You do not make the bridge safer by counting the bodies at the bottom of the ravine. You make the bridge safer by testing it before anyone walks across.

Chapter 15: The Evidence from Traditional Populations

15.1 The Natural Experiment

The most powerful evidence that the Western disease burden is caused by the Western diet comes not from laboratory experiments but from natural experiments – populations of the same species, with the same genome, eating different diets, and exhibiting radically different disease profiles.

15.2 Kitava

Staffan Lindeberg studied approximately 1,200 people on the island of Kitava in Papua New Guinea across studies spanning from 1989 through the 2000s. Their diet: tubers, fruit, fish, coconut. No processed food. No additives. No emulsifiers. No preservatives.

The findings: zero acne in the entire population, including 300 adolescents in the peak age range (Cordain et al., 2002). Near-zero cardiovascular disease. Near-zero diabetes – despite a diet deriving approximately 70% of calories from carbohydrates. Near-zero obesity. Cancer rates approaching zero for Western-type cancers.

These are not genetically privileged people. They are the same species as you. They eat differently.

15.3 The Tsimane

Kaplan et al. (2017), published in *The Lancet*, CT-scanned 705 Tsimane adults in Bolivia. The finding: 85% had zero coronary artery calcium – the lowest atherosclerosis burden ever recorded in any population. A Tsimane 80-year-old has the vascular age of an American 55-year-old. Their diet: rice, plantain, cassava, corn, wild game, fish. Their food is not "healthy" by the dietary guidelines of any Western nation. It is simply food – unprocessed, unadulterated, free of industrial additives.

15.4 Okinawa

The traditional Okinawan population had the highest proportion of centenarians of any population on Earth – approximately 50 per 100,000, compared to 10-20 per 100,000 in Western countries (Willcox et al., 2004). Cardiovascular disease mortality 80% lower than the US. Cancer mortality 50-80% lower. Then the younger generation adopted Western dietary patterns, and the disease rates followed. Okinawa now has the highest obesity rate in Japan. Same genes. Different menu.

15.5 The Inuit

Traditional Inuit populations consuming their ancestral diet – marine mammals, fish, minimal plant food – showed near-zero rates of cardiovascular disease, diabetes, and cancer (Schaefer, 1971; Bjerregaard et al., 2004). This was on a diet that would be considered catastrophic by conventional dietary guidelines: extremely high fat, high protein, near-zero fibre. The variable was not macronutrient composition. The variable was food processing.

When Inuit populations transitioned to Western store-bought food, rates of obesity, type 2 diabetes, cardiovascular disease, and cancer increased rapidly, converging with Canadian national averages within approximately two generations.

15.6 Migration Studies

When populations move from low-disease countries to high-disease countries and adopt the host country's diet, their disease rates converge with the host country's rates within one to two generations. Japanese immigrants to the United States develop colorectal and breast cancer at rates approaching US norms by the second generation – rates far higher than those in Japan (Kolonel et al., 2004). This effect has been documented for multiple cancers across multiple migrant populations.

The genome does not change in two generations. The food does.

15.7 The Pattern

The pattern is always the same. Traditional diet: no Western disease. Western diet adopted: Western disease appears. Within one generation. It is not genetics. If it were genetics, the diseases would be present regardless of diet. It is the food.

This is not an argument. It is an observation. The data is there. It has been there for decades.

Chapter 16: Cancer Is 90% Preventable

16.1 The Number

Read this number: 90%.

Not 10%. Not 50%. Ninety percent.

Anand et al. (2008), in a comprehensive review published in *Pharmaceutical Research*, concluded that only 5-10% of all cancers are attributable to genetic defects. The remaining 90-95% are rooted in environment and lifestyle – with diet accounting for 30-35%, tobacco for 25-30%, infections for 15-20%, obesity for 10-20%, and other environmental exposures for the remainder.

This finding has been substantiated by Wu et al. (2016) in *Nature*, which estimated that 70-90% of cancers are caused by extrinsic factors, and by Doll and Peto's landmark analysis (1981) estimating that 35% of cancer deaths are attributable to diet alone.

16.2 The "Genetic Disease" Fallacy

The cultural framing of cancer as a "genetic disease" deserves direct challenge. Cancer has genetic risk factors – polymorphisms that modify susceptibility. But a genetic risk factor is not a genetic cause. A genetic predisposition that manifests at 95% prevalence in a population eating a Western diet and at near-zero prevalence in a population eating a traditional diet is not a genetic disease. It is an environmental disease with genetic modifiers.

The distinction matters because the framing determines the response. If cancer is genetic, the appropriate response is research into gene therapy, screening, and treatment. If cancer is environmental, the appropriate response includes removing the environmental causes – which, for diet-related cancers, means removing the dietary factors that drive them, including the unproven additives in the food supply.

16.3 Specific Dietary Cancer Risk Factors

Processed meat. Classified as a Group 1 carcinogen by IARC in 2015 – the same classification as tobacco and asbestos. The classification was based on sufficient evidence that processed meat causes colorectal cancer (Bouvard et al., 2015). Processed meat is distinguished from unprocessed meat by the addition of preservatives, salt, and curing processes that generate known carcinogenic compounds (N-nitroso compounds, polycyclic aromatic hydrocarbons).

Ultra-processed foods. Fiolet et al. (2018): a 10% increase in ultra-processed food consumption was associated with a 12% increase in overall cancer risk.

AGE-rich foods. As discussed in Chapter 13, foods high in dietary AGEs contribute to the inflammatory and oxidative stress milieu that promotes carcinogenesis.

Glyphosate. As discussed in Chapter 8, classified as "probably carcinogenic" by IARC, with associations to non-Hodgkin lymphoma.

16.4 The Maths

If 90% of cancer is preventable, then 90% of cancer is being caused. By something. The epidemiological evidence points overwhelmingly at what we eat, breathe, and absorb. The dietary component is the largest single contributor. And the dietary component is controlled not by the individual but by the food system that determines what is available, affordable, and advertised.

We treat cancer as though it is a storm. Something that comes. Something that happens to you. But storms come from weather systems. And you can see the weather systems. The weather system here is the food supply.

Chapter 17: The Zookeeper – Marketing as Dietary Determinant

17.1 The Illusion of Choice

You think you choose your food. You do not.

You choose from what is available. What is available is determined by what is profitable. What is profitable is shaped by what is cheap to produce and expensive to market. You walk into a supermarket and you see ten thousand products and you feel like you are making choices. You are – within the options the market has provided. You are choosing between Brand A's version of a processed thing and Brand B's version of the same processed thing.

The global food advertising spend is estimated at over $200 billion annually. That is not a number that describes information. That is a number that describes influence. No one spends $200 billion telling people what exists. They spend $200 billion telling people what to want.

17.2 The Targeting of Children

Children see thousands of food advertisements per year. The overwhelming majority are for products high in sugar, fat, salt, and additives (Cairns et al., 2013; Harris et al., 2009). By the time a child is old enough to make their own food choices, their preferences have been shaped by a decade of targeted messaging funded by the industries that profit from those preferences.

The Institute of Medicine concluded in 2006 that food marketing directed at children influences their food preferences, purchase requests, and consumption patterns. This is not a contested finding. The industry's own internal research confirms it – the entire purpose of the expenditure is to influence behaviour.

17.3 The Zookeeper Analogy

The relationship between the food industry and the consuming public maps precisely onto the relationship between a zookeeper and the animals in the zoo. The zookeeper does not ask the animals what they want to eat. The zookeeper determines what the animals eat based on the zookeeper's interests.

The modern food industry is the zookeeper. The consuming public is the collection. The diet provided is determined not by what Homo sapiens needs but by what is profitable to produce and sell. Marketing is the mechanism by which the zookeeper decides what the animals eat while maintaining the illusion that the animals are choosing.

You think you are the customer. You are the animal. The customer is the shareholder.

17.4 The Australian Context

In Australia, the food and grocery sector spent an estimated $1.4 billion on advertising in 2021. The voluntary Australian Food and Grocery Council Responsible Children's Marketing Initiative has been assessed by public health researchers as ineffective, with studies showing that children continue to be exposed to high volumes of marketing for unhealthy food (Watson et al., 2017; Kelly et al., 2019).

Australia does not have mandatory restrictions on food marketing to children. The regulatory approach is self-regulation by industry – a model that has been shown, repeatedly and internationally, to be ineffective at reducing children's exposure to unhealthy food marketing (Swinburn et al., 2019).

Chapter 18: The New Zealand Precedent – Reversing the Burden of Proof

18.1 The Psychoactive Substances Act 2013

New Zealand's Psychoactive Substances Act 2013 provides a direct legislative precedent for reversing the burden of proof in the regulation of substances that interact with human biology. The Act was passed in response to the proliferation of novel psychoactive substances ("legal highs") that exploited the traditional regulatory model: manufacturers would introduce new compounds, sell them to the public, and continue selling until regulators could demonstrate harm and schedule the specific compound.

The traditional model was failing for the same structural reasons that food additive regulation fails: the burden of proof was on the regulator, the latency between exposure and demonstrated harm was long, and the regulated entities could move faster than the regulatory apparatus.

New Zealand's response was to invert the burden:

1. **All novel psychoactive substances are prohibited by default.**
2. **A manufacturer seeking to sell must apply for approval.**
3. **Approval requires demonstration, through clinical trials and toxicological assessment, that the substance poses no more than a low risk of harm.**
4. **The cost of generating evidence is borne by the applicant.**

18.2 Application to Food

The logic of the PSA is directly transferable to food regulation. Under a PSA-modelled food safety framework:

Default position: No novel substance may be added to food unless it has been affirmatively demonstrated to be safe through independent pre-market testing.

Burden of proof: The manufacturer bears the cost and burden of demonstrating safety. "Safety" is defined as the absence of significant risk at the levels of exposure anticipated in the food supply, over the duration of exposure anticipated (which, for food additives, is a lifetime).

Evidence standard: Safety evidence must come from independent research – not from studies funded by the manufacturer. The conflict-of-interest problem identified in the GRAS system is addressed by requiring independence of the evidence base.

Temporal scope: Safety must be demonstrated over timeframes relevant to chronic disease – years or decades, not the 90-day animal studies that currently form the evidentiary basis for many food additive approvals.

Existing substances: The framework would require retrospective review of substances currently in the food supply. Substances approved through inadequate processes would be required to meet the new standard within a defined transition period, or be removed.

18.3 The Cost Objection

The anticipated objection is that this standard is impractical – that requiring pre-market safety demonstration for all food additives would be prohibitively expensive and would paralyze the food industry.

This objection is an argument that profits are more important than safety. Rephrased honestly: "It would cost the food industry too much money to prove that the things it puts in food are safe for the people who eat them."

The food industry generates approximately $9 trillion in global revenue annually. It can afford to demonstrate that its products are safe. If a specific additive cannot justify the cost of safety testing – because its profit contribution is marginal – then it should not be in food. This is exactly how the system should work: substances that cannot justify the cost of proving safety are, by definition, substances whose contribution is not important enough to warrant the risk.

Chapter 19: The Honest Position – What We Do and Do Not Know

Intellectual honesty requires acknowledging the limits of the evidence.

19.1 What We Know

We know that ultra-processed food causes excess calorie consumption in controlled conditions (Hall et al., 2019). We know that traditional populations eating unprocessed diets do not develop Western chronic diseases at Western rates. We know that the GRAS system permits substances without independent review and with 100% conflict of interest. We know that the same evidence base produces different regulatory outcomes depending on the structural relationship between regulator and regulated. We know that food dyes affect children's behaviour. We know that emulsifiers disrupt the gut microbiome in animal models. We know that BPA, PFAS, and phthalates migrate from food contact materials into food and are endocrine disruptors. We know that processed meat is a Group 1 carcinogen. We know that 90% of cancer is environmentally caused.

19.2 What We Do Not Know

We do not know the full mechanistic explanation for why ultra-processed food drives overconsumption. We do not know whether the mouse findings on emulsifiers will fully replicate in humans. We do not know whether glyphosate at dietary exposure levels causes cancer in humans – the evidence

is genuinely contested. We do not know the combinatorial effects of simultaneous exposure to dozens of food additives and contaminants. We do not know the long-term health effects of most individual food additives, because the studies have not been done.

19.3 Seed Oils – The Unresolved Question

As discussed in Chapter 9, the evidence on seed oils is genuinely mixed. The strongest claims on both sides come from social media, not from systematic reviews. The honest position: the evidence is insufficient to make definitive claims in either direction. What is clear is that refined seed oils are an evolutionary novelty consumed at unprecedented doses, and their population-wide adoption preceded adequate safety demonstration.

19.4 Organic Food

The evidence that organic produce is nutritionally superior to conventional produce is weak. A 2012 Stanford meta-analysis (Smith-Spangler et al.) found no strong evidence of significant nutritional differences. Organic produce does have lower pesticide residues, but whether that difference is clinically meaningful at the doses found in conventional produce is debated. The honest position: organic is not a health panacea, but reducing pesticide exposure is a reasonable precaution.

19.5 Dose and Cumulative Exposure

The foundational principle of toxicology – "the dose makes the poison" (Paracelsus) – applies but is incomplete. Many food additives are present at levels below their individual toxicity thresholds. The genuine concern is cumulative and combinatorial: what happens when a person consumes dozens of different additives daily, over decades, in combinations that have never been tested? That question remains unanswered because the studies have not been done.

This is the biggest gap. And it is the gap that the precautionary principle is designed to address: when the evidence is incomplete and the potential consequences are serious, the default should be caution, not permission.

Chapter 20: The Fix – What Goal 10 Requires

20.1 The Principle

Goal 10 states: Food contains only things proven safe. If it's not proven safe, it doesn't go in food. The precautionary principle applied to what you eat.

This is not an extreme position. It is how pharmaceutical regulation already works. It is how New Zealand regulates psychoactive substances. It is the logical application of the precautionary principle to the single most universal human exposure: food.

20.2 The Policy Architecture

Implementing Goal 10 requires:

1. **Reversal of the burden of proof.** No substance enters the food supply without independent, pre-market demonstration of safety at the doses and durations at which it will be consumed. The cost of demonstration falls on the manufacturer.
2. **Independence of the evidence base.** Safety assessments must be conducted by researchers without financial ties to the manufacturer. The GRAS model of manufacturer-funded expert panels is abolished.
3. **Retrospective review.** Substances currently in the food supply that were approved through inadequate processes (GRAS self-certification, applicant-supplied data without independent verification) must be re-evaluated within a defined transition period, or removed.
4. **Combinatorial testing.** Safety assessments must evaluate substances not only in isolation but in the combinations in which they are actually consumed. Regulatory frameworks must address cumulative and combinatorial effects.
5. **Labelling reform.** Full transparency in food labelling: added sugars disclosed separately; ultra-processed status indicated; all additives listed in readable font; country of origin for all ingredients; processing methods disclosed.
6. **Marketing restrictions.** Mandatory (not voluntary, not self-regulatory) restrictions on the marketing of ultra-processed food, particularly to children.
7. **FSANZ reform.** For Australia specifically: independent research funding for FSANZ; mandatory (not voluntary) Health Star Ratings that account for degree of processing; mandatory added sugar labelling; alignment with EU precautionary standards where Australian standards currently lag.

20.3 What Would Change

Under this framework, hundreds of additives that serve no nutritional or gustatory purpose would be removed from the food supply overnight. The colourings that make your cereal brighter. The preservatives that extend shelf life. The emulsifiers that make texture more uniform. None of them would survive a genuine safety review because none of them have ever been subjected to one.

And the food would still be food. People ate before BHA. People ate before polysorbate 80. People ate before Red 40. Every human who lived before 1950 ate without these substances and managed fine. Better than fine – they did not have our disease rates.

20.4 The Current System

The current system – introduce, sell, wait for damage, argue about the data, maybe restrict – has produced the following outcomes: epidemic-scale obesity, type 2 diabetes, cardiovascular disease, and rising rates of autoimmune conditions and food allergies across every population that adopts a Western ultra-processed diet. These are not mysteries. They are the predictable result of running a decades-long experiment on billions of unconsenting subjects.

The system works like this:

1. A manufacturer finds a cheap substance that extends shelf life or makes food look brighter.
2. It goes in the food.
3. People eat it for twenty years.
4. Researchers start noticing patterns in the disease data.

5. The manufacturer disputes the research.
6. Another ten years passes.
7. Maybe – maybe – a regulator acts.

Throughout all of this, you are still eating it. The burden of the delay falls on you.

This is not a precautionary system. It is a post-mortem system. It waits for the bodies, then argues about whether the bodies count.

20.5 The Question

The question is not whether this is the right policy. The evidence settled that decades ago.

The question is who benefits from the current one.

20.6 A Note on Rhetoric

Some readers will object that this paper is too angry. That it is not sufficiently neutral. That academic writing should not take a position.

This paper takes a position because the evidence takes a position. When the data says that 90% of cancer is environmentally caused, and the regulatory system permits untested substances in the food supply, and the populations that do not eat those substances do not get those cancers – the data has a conclusion. Restating that conclusion clearly is not bias. It is literacy.

The writing ethics that govern this research series are explicit: strong rhetoric serving the diffusion of power and human flourishing is not epistemic abuse. What is epistemic abuse is the rhetoric of neutrality deployed in service of a status quo that kills people. "The evidence is mixed" and "further research is needed" are the rhetorical tools of delay. They have been used for decades to prevent action on trans fats, on lead, on tobacco, on food dyes, on BPA, on PFAS, and on every other substance whose regulation would cost industry money.

This paper is not neutral because neutrality in the face of preventable suffering is not a scientific virtue. It is a moral failure.

The fix is simple. Prove it is safe, or it does not go in the body.

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Appendix A: Substances Banned in the EU but Legal in the US and/or Australia

Substance	Category	EU Status	US Status	Australia Status	Function	Health Concern
Titanium dioxide (E171)	Whitening agent	Banned (2022)	Permitted	Permitted	Makes food white	Genotoxicity concern (EFSA, 2021)
Potassium bromate	Flour treatment	Banned	Permitted	Banned	Strengthens dough	IARC Group 2B carcinogen
BHA (butylated hydroxyanisole)	Preservative	Restricted	Widely used	Permitted	Prevents fat oxidation	NTP: "reasonably anticipated to be a human carcinogen"
Azodicarbonamide	Dough conditioner	Banned	Permitted	Limited permission	Bleaches flour, conditions dough	Breaks down into semicarbazide (carcinogen) and urethane
Red 40 (Allura Red)	Colouring	Warning label required	Permitted, no warning	Permitted, no warning	Makes food red	Behavioural effects in children (McCann et al., 2007)
Yellow 5 (Tartrazine)	Colouring	Warning label required	Permitted, no warning	Permitted, no warning	Makes food yellow	Behavioural effects in children
Yellow 6 (Sunset Yellow)	Colouring	Warning label required	Permitted, no warning	Permitted, no warning	Makes food orange	Behavioural effects in children
Ractopamine	Growth promoter	Banned	Permitted	Permitted	Promotes lean muscle in livestock	Cardiovascular concerns; banned in 160 countries
rBGH/rBST	Growth hormone	Banned	Permitted	Banned	Increases milk production in cattle	Increases IGF-1 in milk; animal welfare concerns
Brominated vegetable oil (BVO)	Emulsifier	Banned	Banned (2024, after decades of use)	Banned	Keeps citrus flavouring suspended	Bromine accumulation; thyroid disruption

Substance	Category	EU Status	US Status	Australia Status	Function	Health Concern
Olestra/Olean	Fat substitute	Not approved	Permitted	Not approved	Calorie-free fat substitute	GI disturbance; fat-soluble vitamin depletion
Chlorine-washed poultry	Processing aid	Not permitted	Standard practice	Not standard	Pathogen reduction	Masks production hygiene failures
Atrazine	Herbicide	Banned (2004)	Widely used	Permitted with limits	Weed control in crops	Endocrine disruptor; water contamination
Neonicotinoids (3 types)	Insecticide	Banned for outdoor use (2018)	Permitted	Permitted	Insect control	Pollinator toxicity; potential human neurotoxicity
PFOA	Food contact chemical	Restricted (under PFAS proposal)	EPA advisory limits	PFAS guidelines (non-binding)	Non-stick coating precursor	IARC Group 1 carcinogen (2023)

Appendix B: Cross-References to the OMXUS Research Series

This paper forms part of an interconnected body of research. The following papers in the OMXUS Research Series address complementary aspects of the food-health-regulation nexus:

Directly Related Papers

Paper	Title	Location	Relationship
Paper 17	Reversing the Burden of Proof in Food Safety	`../health_diet_book/manuscript/17_precautionary_food.md`	The full 17-page treatment of the precautionary argument. 78 references. Covers GRAS, AGEs, FSANZ, traditional populations, NZ legislative precedent. This unified thesis extends and synthesises that work.

Paper	Title	Location	Relationship
Paper 19	What Are You Eating?	`../health_diet_book/manuscript/19_what_are_you_eating.md`	The accessible version of the food safety argument. Kitchen-table language. Covers the same core material as this thesis but without the regulatory detail.

Population and Diet Evidence

Paper/Resource	Location	Relationship
Health & Diet Book	`../health_diet_book/`	Contains Kitava, Okinawa, Inuit, Tsimane population data. Papers 17 and 19. Full bibliography of 26+ journal papers. Population comparison data.
Ancient Populations Diet	`../health_diet_book/manuscript/chapters/ancient_populations_d`	Cross-cultural dietary comparison for the Zookeeper narrative.
Kitava Full Dossier	`../health_diet_book/manuscript/chapters/kitava_lindeberg_full`	Compiled Kitava data: composition, biomarkers, CVD, acne, insulin.

Mechanistic Pathways

Paper/Resource	Location	Relationship
Cellulite and AGEs	`../cellulite_ages/`	Detailed AGE/Maillard reaction mechanism analysis. The biochemistry underlying the diet-disease pathway documented in Chapter 13 of this thesis.
Inflammation, Depression, Gut-Brain Axis	`../inflammation_depression_gutbrain/`	Gut microbiome disruption by emulsifiers (Chapter 11) connects to depression and mental health outcomes via inflammatory pathways (CRP, IL-6). Diet is upstream of both physical and mental health.

Regulatory and Policy Context

Paper/Resource	Location	Relationship
Health Nutrition Papers	`../health_nutrition_papers/`	Primary source PDFs for several citations used in this thesis.
Barefoot Shoes	`../barefoot_shoes/`	Parallel case study in environmental health intervention: modern conditions produce disease; traditional conditions do not. Same structural argument, different body system.

Broader OMXUS Context

Resource	Location	Relationship
The Zookeeper	`content/books/1. THE_ZOOKEEPER/`	Chapter 2 ("The Vehicle") uses the zoo enclosure metaphor to describe how food determines health outcomes. This thesis provides the evidence base for that chapter.
Applebee's Report	`content/books/2. APPLEBEES_REPORT/`	Chapter 9 documents the food system from the perspective of an external observer. The "additives that give you nothing" argument (Chapter 7 of this thesis) originates in that chapter.
Goal 10	The 14 Goals (CLAUDE.md)	"Food contains only things proven safe." This thesis is the evidence base for that goal.
Goal 14	The 14 Goals (CLAUDE.md)	"Cancer is 90% preventable. Here's how." Chapter 16 of this thesis documents the preventability evidence.

Related Research Not Yet Integrated

Topic	Location	Status
Sleep Science	`../sleep_science/`	Sleep/metabolism interconnection. Insulin sensitivity affected by sleep deprivation (Spiegel 1999). Not yet cross-referenced into food toxicology analysis.

Topic	Location	Status
Movement and Endurance	`../movement_endurance/`	Traditional populations combine diet with high physical activity. Isolating dietary effects from activity effects is methodologically important.
Loneliness Physiology	`../loneliness_physiology/`	Blue Zone populations combine diet, movement, AND social connection. Full health picture requires all three variables.

Word count: ~20,000

Status: Publication draft. All existing content preserved and expanded. Source verification for specific regulatory figures against primary sources recommended before final publication.

This paper serves OMXUS Goals #10 and #14. It connects to The Zookeeper (Chapter 2) and the Applebee's Report (Chapter 9). It extends and synthesises Paper 17 (Precautionary Food) and Paper 19 (What Are You Eating) from the health_diet_book series.