

THE METABOLIC AMBUSH

Constructed Food Reality, Biological Preconditioning, Pharmacological Individualisation, and the Transfer of Metabolic Costs to the Subject

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The Metabolic Ambush lies not only in what a person eats, but in what they can access; in how the conditions that make them desire it again are produced; and in the statistical judgements through which their embodied reality is made visible or invisible.	32
Systems may produce or intensify the condition; the subject performs the act; the body bears the consequence; pharmacology intervenes at the biological node. Metabolic justice begins by treating disease without ceasing to ask why its costs are repeatedly transferred to the subject's body.	32
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Abstract

This article challenges accounts of metabolic disease and changes in body weight that begin and end with clinical outcomes, individual choice, or behavioural failure. It introduces the **Metabolic Ambush** as a condition in which much of the biological, material, and social history producing an outcome has already taken effect by the time that outcome becomes clinically visible. Insulin resistance is treated as a multi-organ metabolic node without being elevated into a universal cause. Obesity is recognised as a real phenotype and a possible causal mediator, but not as an adequate explanation of its own production.

Food is assigned analytical priority not because it has been shown to possess the largest causal effect in every disease, population, and individual, but because it is a principal material route through which economic, agricultural, and industrial conditions enter the body as compulsory and repeated exposure. A dietary pattern is therefore reconceptualised not as a fixed attribute of the subject, but as a realised record of consumption produced under conditions of income, price, production, distribution, time, and access. The analysis distinguishes sucrose from high-fructose corn syrup without converting either similarity into harmlessness or difference into a claim of unique toxicity. Molecule, dose, energy context, food matrix, repetition, and architecture of access must be examined together.

The clinical efficacy of GLP-1-based pharmacotherapies is explicitly acknowledged. Their effects demonstrate that hunger, satiety, reward, and eating behaviour are not products of will alone. Yet pharmacological intervention does not, by itself, alter food prices, product formulation, unequal access, or the social sources of chronic stress. The central sequence is therefore as follows: **conditions may be produced within systems; action is performed by the subject; consequences become visible in the body; responsibility and treatment costs are then transferred back to that subject.** Agency is not denied. It is distinguished from authorship of the world in which action takes place.

Keywords: Metabolic Ambush; Reality Paradigm; insulin resistance; constructed food reality; biological preconditioning; food access; GLP-1; semaglutide; tirzepatide; pharmacological individualisation; metabolic cost transfer; Homovictimus.

Method and Evidential Status

This article is neither a systematic review nor a meta-analysis, and it is not a clinical guideline. It combines a sociological manifesto with a critical synthesis of selected primary human studies, prospective cohorts, randomised controlled trials, and official health assessments.

The sources do not occupy a single evidential plane:

- A randomised intervention may establish the effect of a specified intervention under specified conditions; it does not establish an entire structural chain of causation.
- A prospective association may establish temporal order and predictive value; it does not, by itself, establish causation.
- A proxy measure is not interchangeable with a direct measurement, although it may permit population-level comparison within properly stated limits.
- Detection of a chemical or environmental exposure does not establish clinical harm at every dose.
- The efficacy of a drug does not mean that the material and social conditions generating the need for treatment have been resolved.

- Industry funding does not automatically invalidate a finding, but it makes the formulation of the research question, comparator selection, analytical control, data access, and publication control matters for scrutiny.

The evidential discipline of this article turns on a basic distinction:

To precede, to predict, to be associated with, and to cause are not the same claim.

These distinctions are not concessions intended to weaken the theory. They are the conditions under which it becomes testable, portable, and capable of surviving criticism.

This article does not provide individual diagnosis or treatment advice. Clinical decisions require an assessment of the individual patient, the balance of benefits and harms, and professional medical judgement.

MANIFESTO

The point of departure is not the statistic.

It is the **human body**.

Statistics do not create what occurs within the body. They render measurable parts of it visible. A country may not measure fasting insulin, may lack longitudinal HOMA-IR series, or may restrict metabolic surveillance to glucose and HbA1c. Such omissions do not abolish the unmeasured physiology.

But where data do not exist, numbers cannot honestly be manufactured.

The epistemic order of the manifesto is therefore explicit:

**first, the body;
then, mechanism;
then, temporality;
then, population scale.**

A statistic is not a cause.

It is a measure of scale.

And a measure has no authority to declare nonexistent what it was not designed to register.

1. Statistics Do Not Create Reality, but They Can Capture the Authority to Judge It

The human body exists before the statistical sentence pronounced about it.

A person may maintain normal glucose at the cost of elevated insulin secretion. Hepatic fat may be accumulating. Satiety, reward, and repeated consumption may already be changing. Yet the diagnostic threshold for diabetes may not have been crossed.

The bodily process is real before it is classified.

Only afterwards does the institutional sequence appear:

normal glucose → no diabetes → no diagnostic threshold crossed → no metabolically visible disorder.

Statistics cannot retrospectively remove an event from the body. They can, however, alter its **institutional visibility and binding force** by determining which variables are measured, which thresholds are adopted, and which results are designated normal, risky, or pathological.

The distinction is decisive:

Statistics cannot change the occurrence; they can change its visibility, name, category, and force as a judgement.

This manifesto is not hostile to statistics. It is hostile to the substitution of a statistical representation for the body it incompletely represents.

Lived metabolic reality cannot be manipulated out of existence. Yet selected indicators, thresholds, and categories can reconstruct its public and clinical status. This is the first movement of the Metabolic Ambush:

The body may already be paying the cost while the official judgement remains 'normal'.

2. The Metabolic Ambush

The term ambush is deliberate. It does not allege a secret conspiracy.

The Metabolic Ambush describes a temporal structure:

By the time a clinical outcome becomes visible, much of the biological, material, and social history that produced it has already occurred.

Metabolic change does not begin on the day diabetes is diagnosed. The history of hepatic steatosis does not begin when it is first detected. Renal dysfunction does not originate on the morning albumin first appears in the urine. A coronary event is not the first moment at which the vascular system has been affected.

Diagnosis is not the beginning.

It is a threshold at which an earlier process becomes institutionally visible.

The ambush lies precisely here:

The outcome is visible now; the conditions that prepared it were operating before now.

Disease must therefore be investigated not only forward from diagnosis, but backwards from the body into the history that made the diagnosis possible.

3. Normal Glucose Is Not Metabolic Normality

Two people may each have a fasting glucose concentration of 90 mg/dL.

One body may maintain that value with relatively little insulin. The other may require substantially greater insulin secretion to achieve the same result.

On paper:

90 = 90.

In biology:

the two states need not be equivalent.

The METSIM study found substantial variation in insulin sensitivity and secretion while glucose remained within the normal range. In the RISC study, insulin sensitivity was measured directly by hyperinsulinaemic-euglycaemic clamp, and lower baseline sensitivity was associated with later deterioration in glucose tolerance (Stancáková et al., 2009; Ferrannini et al., 2011).

The implication is straightforward:

Normoglycaemia is an outcome. The hormonal cost at which it is maintained is a separate question.

Diabetes is not the totality of metabolic dysfunction. It is one possible late manifestation.

HOMA-IR is not a direct measurement of insulin resistance. It is a proxy calculated from fasting glucose and insulin. Nor is there a universally agreed clinical threshold. These limitations are real. Yet the imperfection of a measure does not invalidate convergent deterioration across multiple metabolic indicators at population scale.

The absence of a universal threshold does not abolish a metabolic problem expanding across the world.

That expansion is not inferred from a single HOMA-IR series. NCD Risk Factor Collaboration analyses covering 200 countries and territories documented widespread increases in obesity and diabetes between 1990 and 2022. Age-standardised diabetes prevalence increased in 131 countries for women and 155 for men, while the global number of adults living with diabetes reached approximately 828 million in 2022 (NCD Risk Factor Collaboration [NCD-RisC], 2024a; NCD-RisC, 2024b).

These findings do not demonstrate an identical mechanism or rate of change in every country. They do show that the problem cannot be dismissed as a local artefact of measurement.

4. A Wider Metabolic Population May Change Before Diabetes Appears

An analysis of 17,310 US adults without diabetes in NHANES 1999-2018 reported marked increases in hyperinsulinaemia and HOMA-IR-defined insulin resistance under fixed research definitions. Age-standardised hyperinsulinaemia prevalence rose from 28.2 to 41.4 per cent, and HOMA-IR-defined insulin resistance from 24.8 to 38.4 per cent (Wu, Ke, & Nianogo, 2025).

These percentages are not universal diagnostic thresholds. They are population estimates contingent upon operational definitions.

The critical point nevertheless remains:

people with diabetes were excluded from the sample.

It is therefore untenable to assume that:

the population with diabetes = the entire metabolically altered population.

A larger metabolic population may have changed before clinical disease is recorded. The task of statistics is not to close this field prematurely, but to state clearly what a threshold reveals and what it leaves outside the frame.

5. Insulin Resistance Is Not Merely Diabetes in Waiting

Insulin resistance is commonly narrated as a single line:

insulin resistance → prediabetes → type 2 diabetes.

That line is real, but incomplete.

In human studies, insulin resistance and hyperinsulinaemia have been measured before, or prospectively associated with, the development of fatty liver, certain coronary outcomes, albuminuria and renal impairment, hyperuricaemia, and some patterns of cognitive decline. Markers of insulin secretion such as C-peptide have also been associated with the later incidence of some cancers, although evidence in this field is more heterogeneous and predominantly observational (Rhee et al., 2011; Hedblad et al., 2002; Jang et al., 2015; Huh et al., 2017; Nakamura et al., 2014; Ekblad et al., 2017; Luchsinger et al., 2004; Cust et al., 2007; Ma et al., 2004).

Four evidential statuses must not be collapsed:

- being measured before disease;
- predicting disease;
- being associated with disease;
- causing disease.

Even a prospective association does not, by itself, establish causation. Insulin resistance may be a consequence, mediator, or proxy of visceral adiposity, hepatic fat, physical inactivity, inflammation, or other processes.

In the IRIS randomised trial, pioglitazone reduced the composite outcome of recurrent stroke or myocardial infarction in insulin-resistant patients without diabetes who had recently experienced an ischaemic stroke or transient ischaemic attack. The intervention strengthens the case for the clinical relevance of insulin resistance, but the multiple actions of pioglitazone do not permit insulin to be declared the single independent cause (Kernan et al., 2016).

The argument therefore does not require the claim that insulin causes every disease.

Its more limited claim is also more defensible:

Insulin resistance is an important node in multi-organ metabolic dysfunction that may later become visible under several different clinical names.

It is not the only node. But it is too consequential to be confined to the category of diabetes alone.

6. Not a Single Cause, but Interacting Metabolic Nodes

Human metabolism is not a straight line.

Insulin resistance interacts reciprocally with:

- visceral and ectopic adiposity;
- hepatic and skeletal-muscle metabolism;

- lipid regulation;
- inflammation;
- sleep and circadian rhythm;
- physical activity and energy balance;
- stress hormones;
- medicines;
- environmental exposures.

One process may intensify another, which may then feed back into the first.

The model is not:

insulin resistance → everything.

It is:

interacting metabolic nodes connected through feedback.

Insulin resistance is one important node in that network. It is not the network in its entirety.

7. Obesity Is a Visible Outcome, Not the History of the Body

This manifesto does not deny the existence or clinical significance of obesity. Excess adiposity can be assessed through BMI, waist circumference, fat mass, and fat distribution.

What it rejects is a different operation:

the conversion of obesity into the beginning of metabolic disorder, an explanation of itself, and the name of the body's entire history.

Obesity is a phenotype. It is not the whole metabolic story.

Two people with the same BMI need not have the same insulin sensitivity, visceral fat, hepatic fat, fasting insulin, triglycerides, or muscle mass. Conversely, a person with a BMI in the conventional normal range may be insulin-resistant, hyperinsulinaemic, or have fatty liver.

Body size is not a perfect proxy for metabolic state.

Adiposity, particularly visceral and ectopic adiposity, may become a causal mediator of later metabolic deterioration. That does not make obesity an explanation of its own production. An outcome may become a mediator in a subsequent causal chain without becoming the origin of that chain.

Analyses using genetic instrumental variables support causal effects of overall and abdominal adiposity on insulin resistance and risk of type 2 diabetes. A controlled human study comparing equivalent weight loss achieved by diet or gastric bypass found broadly similar improvements in hepatic and skeletal-muscle insulin sensitivity in both groups (Xu et al., 2020; Yoshino et al., 2020).

The more precise judgement is therefore:

Obesity may be a visible phenotype and a causal mediator of some diseases; it does not explain its own production and cannot stand in for the entire metabolic causal chain.

The statement ‘this person became ill because they were obese’ often appears explanatory. Yet it leaves the central question unanswered:

Why did this body arrive at this form of fat storage, this insulin response, and this metabolic state?

When obesity replaces that question, the visible outcome begins to masquerade as the originating cause.

The appearance of the body is not the history of the body.

8. Reasoning Backwards from the Body

Clinical medicine encounters glucose, insulin, liver, kidney, vasculature, adipose tissue, and diagnosed disease.

But the history of what entered the body did not begin in the consulting room.

Food has a history. Following it backwards takes analysis from:

body → table → access → production → agricultural inputs → soil.

This is not, by itself, a programme of solutions. It is an attempt to restore visibility to a causal sequence that disappears when inquiry begins only at diagnosis.

9. Why Food Comes First

Food is not the sole cause of metabolic dysfunction. Nor is it an ordinary environmental variable.

It is:

the principal material flow that enters metabolism compulsorily and repeatedly.

The body breaks it down, absorbs it, transforms it, stores it, oxidises it, and responds hormonally.

To say that food comes first is not to claim in advance that it has the largest measured causal effect in every disease, population, or person. Its priority is **analytical and material**: food is a primary point of entry.

The stronger reason is structural:

Food is a principal material passage through which economic and agricultural inequality becomes biological exposure.

Income inequality does not cross the cell membrane directly. Agricultural policy does not circulate in the bloodstream. Price, distribution, working time, and access are not themselves edible substances.

They nevertheless determine:

- which products are produced;
- which are made inexpensive;
- which are consistently available;
- which reach the table;

- how frequently they are consumed.

They therefore reach the body **in the form of food**.

Food comes first because it is a principal material carrier through which an already organised economic, agricultural, and industrial reality is translated into metabolic reality.

Food is not alone. But neither is it a secondary footnote in the history of metabolic disease.

10. Constructed Food Reality

To speak of a 'constructed reality' is not to suggest that reality is unreal. The construction is materially concrete.

By the time a person reaches a market shelf, a shop, or a dining table, the product may already have been:

- cultivated and harvested;
- refined or otherwise processed;
- formulated;
- combined with permitted additives;
- packaged;
- priced;
- distributed;
- made visible and accessible in some places rather than others.

Only then does the subject arrive.

The Reality Paradigm therefore asks a question prior to 'What did the person choose?':

What had already been constructed when the person arrived to choose?

Choice exists.

But the available set of objects has a history too.

11. Compulsory Inclusion

Human beings may refuse a product, a cuisine, or a particular diet. They cannot permanently withdraw from the material requirement to eat.

This is **compulsory inclusion**.

It does not mean that everyone consumes the same food or that every exposure is unavoidable. It means that no living person can occupy a position wholly outside the field of nutrition. The subject must repeatedly return to a food environment that they did not create alone.

The choice is therefore rarely between:

food and no food.

It is usually a choice amongst available foods, within limits of price, time, location, preparation, storage, culture, work, and bodily condition.

This distinction matters because a compulsory field cannot be analysed as though entry into it were itself an elective act. A person may exercise tactical agency within the field, but they did not author the biological necessity that repeatedly returns them to it.

Nutrition is not an optional domain into which the subject freely decides to enter. It is a material field in which the subject must act.

12. 'Dietary Pattern' Is a Record of Realised Consumption, Not an Attribute of Character

Dietary-pattern research can describe recurring combinations of foods. It becomes misleading when the pattern observed after consumption is treated as though it were an internally authored and stable property of the person.

The person did consume bread, meat, milk, fish, pulses, vegetables, fruit, or packaged products. The record is not fictitious. The error arises when the realised record is detached from the field that made some foods routine, others exceptional, and still others unavailable.

A household compelled to organise most meals around inexpensive bread and refined starches does not inhabit the same material food environment as one able to obtain meat, fish, milk, eggs, pulses, vegetables, and fruit in varied combinations. This does not prove that every consumer of animal-source foods will be healthier, nor that each such food is beneficial in every amount and context. It establishes a more basic point:

Different fields of access produce materially different diets and materially different ranges of possible outcome.

The truth of the statement 'this person ate more bread' does not settle why bread occupied that position. High bread consumption may reflect preference, but it may also reflect price, satiety per unit of expenditure, availability, storage, household size, working hours, and the absence of realistic substitutes.

A dietary pattern is therefore better understood as:

the statistical appearance of consumption realised within a structured field of access.

This does not remove conduct from the subject. It prevents the analyst from converting the conditions of conduct into a personality trait.

13. Choice Exists, but the Chooser Does Not Necessarily Author the Object or the Choice Set

The critique of constructed food reality does not require the denial of choice.

People select, refuse, compare, postpone, substitute, and consume. These are genuine acts. The question is not whether the act occurred, but whether the actor also created the objects, prices, information, locations, and bodily conditions amongst which the act occurred.

The capacity to choose and the authorship of the field of choice are not the same thing.

Price interventions demonstrate that purchasing is responsive to more than internal preference. In a randomised virtual-supermarket experiment, changes in food prices altered purchasing patterns (Waterlander et al., 2019). Across countries at different income levels, the affordability and availability of fruit and vegetables were associated with consumption, showing that recommendation and feasible acquisition cannot be treated as equivalent (Miller et al., 2016).

These findings do not prove that price determines every purchase. They disprove the stronger fiction that realised consumption can be read as an unmediated expression of desire.

The retrospective statistical sentence - 'this was the subject's dietary pattern' - may be descriptively correct and causally incomplete at the same time. When the realised outcome is recoded as the subject's original preference, structural conditions disappear into the grammar of personal choice.

The Reality Paradigm insists on preserving both propositions:

The choice is real. The reality in which it is made may also have been constructed.

14. Soil, Production, Access, Food, and the Body

The metabolic history of food crosses several domains of knowledge. No single profession contains the entire chain.

Agricultural science encounters the material beginning

Soil composition, water, crop selection, feed, cultivation, contamination, harvest, and storage affect the raw material entering the food system. This does not mean that 'soil causes insulin resistance'. It means that the material qualities and possible contaminants of food have histories before processing and consumption.

Food engineering encounters transformation

Refining, fractionation, extrusion, preservation, emulsification, sweetening, flavour design, texture, portioning, and packaging may alter the final product. The consumer often encounters neither an untouched crop nor an unchanged animal product, but a designed object whose sensory, physical, and commercial properties have been technically organised.

Economics and distribution encounter feasible access

The existence of a food does not make it attainable. Income, price, geography, retail infrastructure, working time, storage, cooking facilities, and distribution determine whether a product can enter a household with sufficient regularity to matter.

Clinical medicine encounters the embodied response

Medicine encounters glucose, insulin, liver, kidney, vasculature, adipose tissue, and diagnosed disease. Its object is indispensable, but it is located downstream of a material history.

None of these domains can substitute for the others. They converge in one sequence:

To read human health only from the threshold of the hospital is to read incompletely the history of the body that arrives there.

15. The Unseen Burden of Food

Food chains carry more than macro- and micronutrients. Soil, water, agriculture, processing, packaging, storage, and distribution may alter nutritional composition and physical structure while also providing routes for environmental and industrial exposures.

The ‘package’ of food should therefore be understood at three levels.

The material package

Packaging, food-contact surfaces, production environments, storage conditions, and possible contaminants.

The industrial package

Formulation, shelf life, flavour, texture, energy density, portioning, price, and marketing.

The historical package

Land use, agricultural policy, production technique, supply chain, distribution infrastructure, and economic access.

The person who consumes a product has not consciously chosen the whole of this history. Yet parts of that history may reach the body through the material properties of the final product.

The subject consumes not only food, but a history of production, processing, packaging, pricing, and access embedded within it.

This statement does not mean that all food is toxic.

Elements such as arsenic, lead, cadmium, and mercury may enter air, water, soil, and particular foods through both natural geological processes and human activity. Risk depends upon chemical form, dose, duration, age, and co-exposure. Detection of an element in a food and demonstration that customary intake causes disease are not the same evidential claim (U.S. Food and Drug Administration [U.S. FDA], 2024a; U.S. FDA, 2026a).

PFAS is likewise not a single substance, but a broad group comprising thousands of compounds with different properties. Environmental contamination, processing, and some food-contact materials can provide routes into food. Evidence concerning more extensively studied compounds such as PFOA and PFOS cannot simply be generalised to every member of the group. ‘PFAS exposure occurs’ is not equivalent to ‘every PFAS causes the same disease at every dose’ (U.S. FDA, 2026b).

The same discipline is required for microplastics and nanoplastics. Their detection in some foods and beverages establishes possible exposure. It does not settle dose, long-term human consequences, or clinical risk. Current methods of collection, identification, characterisation, and quantification remain uneven. The FDA states that current scientific evidence does not demonstrate that levels detected in foods pose a risk to human health; the FAO review likewise identifies major methodological and risk-assessment gaps while recognising dietary exposure as a legitimate field of investigation (Garrido Gamarro & Costanzo, 2022; U.S. FDA, 2024b).

The defensible conclusion is narrower than a rhetoric of universal toxicity and more demanding than a presumption of neutrality:

Food may carry environmental and industrial exposures; detecting exposure is not the same as demonstrating clinical harm.

This evidential boundary sharpens the structural question:

Does the food reaching the subject consist only of its declared nutrient composition, or does it also carry measurable burdens from its production and distribution history?

‘What did you eat?’ remains a valid question. It is not a complete one.

The fuller question is:

What did you eat; how was it produced, formulated, packaged, and made accessible; and what material burdens reached your body with it?

16. Food Is Not Passive Energy

Food is not merely a neutral quantity of calories awaiting voluntary allocation.

Its physical form, nutrient combination, energy density, palatability, rate of consumption, and degree of processing can affect intake and metabolic response. In a tightly controlled inpatient randomised trial, an ultra-processed diet produced greater ad libitum energy intake and weight gain than an unprocessed diet matched for presented calories, macronutrients, sugar, sodium, and fibre (Hall et al., 2019). The trial does not prove that every processed food has the same effect, nor does it identify one universal mechanism. It does demonstrate that formulation and food structure can alter realised consumption under controlled conditions.

Food can also participate in feedback. Repeated intake may change adiposity, insulin sensitivity, reward valuation, hunger, or satiety; those changes may then alter the conditions of later consumption. Experimental evidence that central insulin modulates food valuation, and that a short-term high-calorie diet can produce effects on brain insulin action beyond the intervention period, supports the plausibility of such biological feedback without reducing it to a single pathway (Tiedemann et al., 2017; Kullmann et al., 2025).

The relevant object is therefore not simply:

calories entering a passive body.

It is:

a materially organised food entering a responsive body whose response may alter the conditions of the next act.

17. Where Sucrose and Corn Syrup Meet - and Where They Do Not

The phrase corn syrup is frequently used as though it designated one substance. It does not.

Glucose syrup, commonly produced by hydrolysing maize starch, is composed predominantly of glucose and glucose-derived oligosaccharides.

High-fructose corn syrup (HFCS) is produced by enzymatically converting part of that glucose into fructose. Common formulations include HFCS-42, containing approximately 42 per cent fructose, and HFCS-55, containing approximately 55 per cent; most of the remaining sugar is glucose. Corn syrup, glucose syrup, and HFCS are therefore not interchangeable terms (U.S. FDA, 2018).

Sucrose is a disaccharide consisting of glucose and fructose joined by a glycosidic bond. Digestion cleaves that bond before absorption. In HFCS, glucose and fructose are already present as free monosaccharides.

This is a genuine chemical difference.

It does not justify the categorical claim that HFCS enters the blood or liver in a radically different manner because it is 'already unbound'. The sucrose bond is normally hydrolysed efficiently in the small intestine. Comparative human studies do not support converting that modest structural distinction into a universal claim that HFCS is inherently and substantially more harmful at all doses and under all conditions (Le et al., 2012; Stanhope et al., 2008).

Nor does chemical similarity establish harmlessness.

At high intakes - particularly in sugar-sweetened beverages and energy-surplus conditions - both sucrose and HFCS can deliver large, rapidly consumed loads of glucose and fructose. Controlled trials have reported increases in hepatic lipid and reductions in insulin sensitivity under sustained consumption of sucrose- or HFCS-sweetened beverages (Sigala et al., 2021). Trials comparing fructose and glucose also show that monosaccharide composition can matter under controlled high-dose conditions, including effects on visceral adiposity, lipids, and insulin sensitivity (Stanhope et al., 2009). Other controlled work demonstrates that energetic context, dose, and comparator substantially condition hepatic outcomes (Johnston et al., 2013).

The claim that fructose can be metabolised only by the liver is also too strong. The small intestine and kidneys participate in fructose clearance and metabolism, and the proportion converted into lipid varies with dose, energetic state, and metabolic context (Björkman & Felig, 1982).

The intersection between sucrose and HFCS is therefore not molecular identity. It is the capacity of both to become vehicles for repeated, inexpensive, liquid, and highly formulated free-sugar exposure.

Metabolic risk resides not only in the name of the sweetener, but in the dose, physical form, repetition, energy context, price, and architecture of access through which exposure is produced.

To demonise HFCS while absolving sucrose is not scientific. To emphasise their similarities while removing both from structural scrutiny is not scientific either.

18. Addiction Is Not Merely a Metaphor, but Its Object and Evidential Status Must Be Specified

The term addiction is not used here as rhetorical decoration.

In relation to some highly processed foods, people report and display:

- loss of control;
- intense craving;
- unsuccessful attempts to reduce consumption;
- continued consumption despite adverse consequences;
- tolerance- and withdrawal-like experiences.

The Yale Food Addiction Scale applies symptoms derived from substance-use-disorder criteria to the consumption of highly processed foods. A systematic review and meta-analysis of 272 studies reported a weighted mean prevalence of approximately 20 per cent for a YFAS-defined

addiction-like phenotype, although estimates varied widely by sampling frame, clinical status, and scale version; estimates in general adult populations were lower (Praxedes et al., 2022; Gearhardt et al., 2023).

This does not establish a separate clinical disorder. Food addiction is not an independent diagnostic category in the DSM. Because the scale is constructed from substance-use-disorder criteria, any resulting structural similarity must be interpreted with care.

The defensible claim nevertheless remains:

Addiction-like consumption of some highly processed foods is not merely figurative; it is a measurable behavioural and clinical phenotype.

Controlled research also suggests that foods combining fat and carbohydrate can acquire reward value beyond either macronutrient alone, strengthening the case for examining the architecture of the final product rather than treating every ingredient as an isolated exposure (DiFeliceantonio et al., 2018).

Two propositions must be separated:

‘Pure sugar is, by itself, a pharmacologically addictive substance.’

and:

‘Some intensively formulated foods, often combining refined carbohydrates, sugar, fat, flavour, and texture, are associated with human consumption patterns structurally resembling features of substance-use disorder.’

The strength of the manifesto does not depend upon proving the first proposition.

Nor does the sheer number of additives establish addiction. Preservatives, emulsifiers, flavourings, colourants, and texture modifiers do not share one function or one biological effect. Their diversity creates a duty of analysis, not a licence for indiscriminate accusation.

The final product must be assessed not only through isolated ingredients, but through their combinations, doses, physical structure, frequency of consumption, and behavioural effects.

If food has become a technical system, its evaluation cannot remain confined to single components.

19. Sugar and Food Research Cannot Be Presumed Spontaneously Neutral

Disagreement within nutrition science cannot always be neutralised by saying only that reasonable researchers reach different conclusions. The material production of evidence is itself open to investigation.

Internal documents showed that the Sugar Research Foundation funded a 1960s review of coronary heart disease, shaped the research objective, supplied material, and reviewed drafts while its role remained undisclosed in the published article. The resulting framework foregrounded saturated fat and cholesterol while minimising the possible role of sucrose (Kearns, Schmidt, & Glantz, 2016).

A separate analysis of systematic reviews on sugar-sweetened beverages and weight gain found that reviews whose authors reported financial conflicts of interest with the food industry were substantially more likely to conclude that no positive association existed (Bes-Rastrollo et al., 2013).

These findings do not justify rejecting every industry-aligned study. They impose a more exacting obligation:

Funding, question design, exposure definition, comparator selection, access to data, analytical flexibility, publication control, and conflicts of interest must be treated as part of the evidential status of a claim.

Funding does not automatically make a result false.

It does not automatically leave it neutral either.

The material conditions under which knowledge is produced cannot be excluded from the evaluation of that knowledge.

20. Psychosocial Stress, the Food Environment, and Biological Susceptibility

Consumption is shaped not only by economic access, but also by the biological and psychosocial condition in which a decision is made.

Human laboratory studies suggest that psychosocial stress can alter eating behaviour and preference for energy-dense foods in some individuals. In one study, women with higher cortisol responses following stress consumed more energy and more sweet foods, but the response was not uniform across participants (Epel et al., 2001). Another experiment found that acute stress could alter food choice, with effects varying according to pre-existing eating patterns (Oliver, Wardle, & Gibson, 2000).

Higher chronic stress has also been associated with differences in neural responses to high-calorie food cues. In a community sample, food cravings statistically and partially mediated the association between chronic stress and BMI, but the effect was small and observational mediation did not establish causation (Tryon et al., 2013; Chao et al., 2015).

The evidence does not warrant a deterministic formula:

stress → cortisol → processed food.

Stress responses involve the HPA axis, cortisol, sleep, appetite regulation, reward processing, emotional eating, learned habits, environmental cues, and substantial individual variation. Stress may increase intake in some people, reduce it in others, and alter food choice without changing total intake in still others.

The stronger theoretical conclusion is therefore probabilistic:

A stressed subject and a high-reward food environment are not independent variables.

When inexpensive, energy-dense, highly rewarding food is already widely available, and chronic stress has altered sleep, appetite, reward sensitivity, or behavioural control in a particular person, the question ‘Why did you choose this?’ can no longer be treated as a question about unconditioned will alone.

The sequence may be represented as follows:

production, formulation, price, and access → a high-reward food environment + psychosocial stress and individual biology → altered probability of consumption → repeated metabolic exposure → phenotype → retrospective attribution to personal choice.

This is biological preconditioning without biological determinism.

The capacity to choose is not the same as authorship of the material and biological field in which choice occurs.

21. The Metabolic Preconditioning of Action

A subject does not necessarily encounter food in a biologically neutral state.

Hunger, satiety, insulin sensitivity, sleep, stress, learned reward, food valuation, and prior exposure are already active when a decision is made. Some of these conditions may have been altered by earlier consumption. The body that acts today may therefore differ from the body that first entered the sequence.

The relevant movement is recursive:

food environment → repeated consumption → metabolic and behavioural response → altered conditions of later consumption → further exposure.

This does not mean that biology dictates a single act. It means that biology changes the probability, effort, and cost of possible acts.

The term **biological preconditioning** names this distinction:

The biological ground on which an act is performed may have been partly formed before the act itself.

The subject still eats, refuses, continues, or stops. But the intensity of hunger, craving, satiety, reward, and effort involved in those acts is not authored by conscious intention alone.

The subject's action remains real.

Its conditions are not exhausted by the subject's will.

22. A Correct Prescription Is Not Necessarily an Actionable Prescription

'Eat less sugar', 'choose fresh food', 'lose weight', and 'exercise more' may be medically defensible recommendations.

Their truth does not guarantee their feasibility.

A recommendation becomes actionable only when the subject has sufficient access, time, money, storage, preparation facilities, safety, bodily capacity, and behavioural latitude to carry it out. The same instruction can impose radically different practical burdens upon different people.

If the available foods remain inexpensive, energy-dense, and highly rewarding; if working time and household conditions restrict preparation; and if craving, reward response, insulin sensitivity, sleep, and stress have already changed, information alone does not dissolve the mechanism.

The correctness of a prescription does not guarantee the conditions of its implementation.

A harsher formulation is warranted:

To demand through moral command the reversal of an act already narrowed by material and biological conditions is to translate mechanism into character.

This is not an argument for declaring the subject innocent of every consequence. It is an argument against assigning the entire burden of explanation to the subject.

23. GLP-1-Based Treatment: A Real Intervention at the Biological Node

This manifesto does not deny the efficacy of GLP-1-based pharmacotherapy.

Semaglutide is a GLP-1 receptor agonist. Tirzepatide is a dual GIP and GLP-1 receptor agonist. They are neither the same molecule nor identical pharmacological classes, although both can exert powerful effects on appetite, energy intake, body weight, and metabolic regulation.

In the STEP 1 randomised trial, once-weekly 2.4 mg semaglutide combined with lifestyle intervention produced a mean body-weight reduction of approximately 14.9 per cent over 68 weeks in adults with overweight or obesity but without diabetes, compared with approximately 2.4 per cent with placebo (Wilding et al., 2021).

In SURMOUNT-1, tirzepatide at 5, 10, and 15 mg produced mean reductions of 15.0, 19.5, and 20.9 per cent respectively at 72 weeks, compared with 3.1 per cent with placebo. The most common adverse events were gastrointestinal and generally mild to moderate, occurring chiefly during dose escalation (Jastreboff et al., 2022).

Clinical benefit is not restricted to the scales. SELECT enrolled 17,604 adults with overweight or obesity and pre-existing cardiovascular disease but without diabetes. Semaglutide reduced the incidence of the composite of cardiovascular death, non-fatal myocardial infarction, or non-fatal stroke: 6.5 per cent versus 8.0 per cent with placebo, corresponding to a hazard ratio of 0.80. Adverse events leading to permanent discontinuation were nevertheless more frequent with semaglutide (Lincoff et al., 2023).

These trials support three propositions:

- 1 GLP-1-based treatments can produce real and clinically important effects.
- 2 Hunger, satiety, reward, and energy intake are biologically modifiable processes, not merely moral categories of will.
- 3 Clinical efficacy and structural explanation operate at different analytical levels.

GLP-1-based treatment does not refute biological preconditioning. It makes it experimentally visible.

If a medicine can alter hunger, satiety, and the probability of consumption, the conditions of eating cannot have been produced by will alone.

24. The Structural Limit of Treatment and the Reality after Withdrawal

GLP-1-based treatment acts upon a biological node.

By itself, it does not alter:

- food prices;
- product formulation;
- distribution systems;
- unequal access to nutritious food;
- working time;
- the social sources of chronic stress;
- conflicts of interest in food research.

This does not mean that the medicine merely ‘masks a symptom’. Weight loss, improvements in metabolic indicators, and cardiovascular event reduction in specified groups are real clinical outcomes.

Those outcomes cannot, however, substitute for the structural question:

How were the conditions that made biological intervention necessary produced, and why is treatment directed only at the subject’s body?

Withdrawal studies sharpen this distinction. In the STEP 1 extension, participants regained approximately two-thirds of their previous weight loss during the year after semaglutide and lifestyle intervention were withdrawn (Wilding et al., 2022). In the randomised-withdrawal STEP 4 trial, participants who continued semaglutide lost a further 7.9 per cent between weeks 20 and 68, while those switched to placebo gained 6.9 per cent (Rubino et al., 2021). SURMOUNT-4 similarly found that continued tirzepatide maintained and extended weight loss, whereas withdrawal led to substantial regain (Aronne et al., 2024).

These results do not, by themselves, support either of the following verdicts:

‘The patient lacks willpower.’

or:

‘The medicine has failed.’

The more defensible conclusion is:

The end of a pharmacological effect is not the end of the biological processes altered by the drug or of the environmental conditions the drug never changed.

Long-term treatment of chronic metabolic disease is not inherently exceptional. But the possibility of long-term treatment makes continuing scrutiny of safety, tolerability, nutrition, body composition, access, cost, and post-withdrawal outcomes indispensable under real-world conditions.

The precautionary principle need not become a technology of fear.

It requires only that demonstrated benefit not be mistaken for the closure of every long-term question.

25. Pharmacological Individualisation and the Transfer of Metabolic Costs

Pharmacological individualisation does not mean the mere provision of medicine to an individual. Clinical care necessarily acts upon particular bodies.

The term identifies a different operation:

the closure of explanation and intervention around the individual body while structural conditions producing or intensifying the metabolic outcome remain outside the inquiry.

The circuit has three stages.

1. Consumption

‘What you ate was your choice.’

2. Consequence

‘Your body is your responsibility.’

3. Intervention

‘The solution is continued treatment of your body.’

Each statement may contain a partial truth.

The subject did consume. The body did undergo consequences. Treatment may genuinely help.

Taken together, however, they can erase the constitutive responsibility of production and access. This is the transfer of metabolic costs:

Conditions are produced or intensified within a system; action is performed by the subject; consequences become visible in the subject’s body; the cost of treatment returns to the subject and the health system.

This is not an allegation of conspiracy. It is a diagnosis of decision architecture.

Pharmacological intervention may be necessary and effective. Yet if it substitutes for food policy, production oversight, price regulation, and equitable access, clinical success can conceal structural failure.

The judgement is therefore explicit:

Medicine may alter the biological node; it cannot, by itself, transform the constitutive material world.

Treatment must not be rejected, but neither may it replace investigation of the conditions that made treatment necessary.

26. The Visible Body and the Circuit of Responsibility

The subject gains weight.

The body becomes visible.

The instruction follows:

‘eat less’,

‘stop consuming sugar’,

‘lose weight’.

If the field of access, metabolic regulation, reward, stress, and working conditions have already altered the feasibility of those acts, diagnosis may redescribe the result without explaining it.

A circuit emerges:

constructed food environment → repeated exposure → metabolic phenotype → visible body → attribution of fault → prescription addressed to the same individual → unchanged environment.

The visible body becomes the site upon which an invisible history is judged.

This is why stigma is not merely an error of manners. It is an epistemic error: it mistakes the most visible bearer of a process for the sole author of that process.

Visibility concentrates blame where causation may be distributed.

27. Constitutive Responsibility and Tactical Responsibility

The manifesto distinguishes two forms of responsibility.

Constitutive responsibility

Responsibility for constructing the field in which action becomes possible, probable, inexpensive, difficult, or costly: production, formulation, pricing, distribution, marketing, regulation, employment conditions, and access.

Tactical responsibility

Responsibility for action taken by a subject within that already constituted field: purchasing, refusing, substituting, consuming, seeking treatment, or attempting change.

The distinction prevents two symmetrical errors.

The first is total individualism: the subject is treated as though they authored both the act and all of its conditions.

The second is total exculpation: the subject is treated as though they performed no act at all.

Neither is adequate.

To be an agent is not to be the sole author of the world in which one acts.

Agency persists. So does the architecture that shapes its range and cost.

This is the position of **Homovictimus**: a subject whose tactical act may be real, yet whose field of action was materially narrowed before that act was later recorded and judged as wholly free choice.

The appropriate allocation of responsibility must therefore follow the causal chain rather than the visibility of its final bearer.

28. The History of Human Health Does Not Begin in Hospital

Hospitals, clinicians, diagnostics, and medicines are indispensable. The argument is not against them.

It is against beginning the history of disease only where disease becomes clinically legible.

The metabolic history of a patient may include:

- soil and agricultural conditions;
- industrial formulation and food structure;
- pricing and retail geography;
- household income and working time;
- sleep and psychosocial stress;
- repeated exposure and biological adaptation;
- delayed detection;
- treatment access and cost.

By the time the body enters a clinic, this history may already have been condensed into glucose, hepatic fat, blood pressure, adiposity, vascular injury, craving, or fatigue.

Clinical medicine encounters the condensed result.

A complete account must reopen the history compressed within it.

The hospital is a decisive site of intervention. It is not the beginning of the causal world that made intervention necessary.

Human health therefore requires not only physicians, but agricultural scientists, food engineers, public-health researchers, economists, sociologists, regulators, and the people whose lives are being measured.

This is not a call to dissolve expertise. It is a refusal to allow downstream expertise to stand for the whole chain.

29. The Food System Is Not Automatically an Onopticon

Not every structured environment is an Onopticon. Production, classification, pricing, distribution, and behavioural influence are not sufficient by themselves to satisfy that concept.

Within the Reality Paradigm, the Onopticon names a more specific regime in which preconstructed reality is integrated across domains, options are substantively foreclosed, contestability is weak, and feedback reproduces or intensifies the architecture (Tan, 2026).

The food system may contain components relevant to such an inquiry:

- compulsory exposure to a structured field;
- categorisation of consumers and products;
- price and eligibility mechanisms;
- cross-domain composition of commercial and biological data;
- weak capacity to contest formulation or access;
- feedback between purchasing, targeting, and future availability.

But these features must be demonstrated rather than assumed.

The term Onopticon should not be used as a dramatic label for every instance of corporate influence, unequal access, or persuasive design. Where its threshold is not met, the analysis should stop.

The present argument therefore relies on a more limited set of concepts:

- constructed reality;
- compulsory inclusion;
- constitutive and tactical agency;
- biological depth;
- preconditioning of action;
- transfer of responsibility.

Conceptual discipline requires that a theory end where its evidence ends.

30. The Evidential Boundary of the Manifesto

This manifesto does **not** claim that:

- insulin causes every disease;
- every elevation in insulin is pathological;
- every insulin-resistant person will develop diabetes;
- HOMA-IR is a universal or direct clinical diagnosis;
- every person with obesity has the same metabolic state;
- adiposity has no causal role in subsequent disease;
- a person within the normal BMI range must be metabolically healthy;
- every processed food is harmful;
- every additive causes disease or addiction;
- every packaged product has the same metabolic effect;
- all food is toxic;
- detection of a contaminant proves disease at customary exposure;
- all PFAS compounds have identical properties or effects;
- current levels of microplastics and nanoplastics detected in food have been shown to cause clinical harm in humans;
- pure sugar has been established as an addictive pharmacological agent in isolation;
- corn syrup, glucose syrup, and HFCS are one substance;
- HFCS is more harmful than sucrose at every dose and under every condition;
- fructose is metabolised only by the liver or converted wholly and directly into fat;
- effects of free sugars are independent of dose, total energy, food matrix, and frequency;
- soil directly causes insulin resistance;
- psychosocial stress produces the same eating behaviour in everyone;
- stress causes processed-food consumption through a single cortisol pathway;
- semaglutide and tirzepatide are the same molecule or an identical drug class;
- GLP-1-based treatments are ineffective or intrinsically harmful;
- weight regain following withdrawal proves either patient failure or drug failure;
- structural criticism can replace clinically appropriate treatment;
- individual agency does not exist;
- food has the greatest measured causal share in every disease and every person;
- the modern food system in its entirety is an Onopticon.

It does make the following claims:

Diagnosis is not the beginning.

Normal glucose is not metabolic normality.

Insulin resistance is not merely diabetes in waiting.

Obesity does not explain the whole history of the body.

A dietary pattern cannot be detached from the field of access that produced it and then written back onto the subject as character.

Food is a principal material passage through which structural inequality becomes metabolic exposure.

Food may carry environmental and industrial exposures; detecting exposure is not the same as demonstrating clinical harm.

Sucrose and HFCS are not metabolically identical, but under comparable glucose-fructose loads their differences in most human studies do not displace the importance of dose, energetic context, and mode of consumption.

The free-sugar problem concerns not only molecular identity, but dose, repetition, liquid form, price, formulation, and architecture of access.

Addiction-like consumption of some highly processed foods is a measurable human phenotype.

Sugar and food research cannot be presumed neutral without examining documented industry influence at study level.

Human beings are compulsorily included in nutrition.

The food object and its field of access may be constructed before the subject arrives.

The biological consequences of consumption may alter the conditions of later consumption.

Psychosocial stress may alter the probability of selecting energy-dense, highly rewarding food in some people; the relation is not deterministic.

A medically correct prescription is not necessarily an actionable prescription.

Failure to perform an act is not automatically failure of will.

The clinical efficacy of GLP-1-based treatment demonstrates that hunger, satiety, and energy intake are pharmacologically modifiable biological processes.

Pharmacotherapy may alter a biological node; it cannot, by itself, alter food prices, formulation, unequal access, or the social sources of chronic stress.

Treatment must not be rejected, but it must not replace structural inquiry.

These propositions are not a final answer. They constitute a testable research programme.

Each arrow requires its own evidence:

production → composition composition → exposure exposure → metabolic response metabolic response → capacity for action field of access → realised consumption realised consumption → distribution of disease pharmacological intervention → clinical outcome treatment withdrawal → biological and environmental reassertion.

Gaps in that chain cannot be filled with rhetoric.

But measuring only the final link and treating all previous links as nonexistent is not science either.

FINAL MANIFESTO

Human beings must eat.

They cannot live outside the material world of nutrition.

Yet the necessity of eating does not mean that they created the food world placed before them.

Food has a past:

soil and production;

agricultural inputs;

processing;

refining;

formulation;

additives;

packaging;

pricing;

distribution;

access;

then the table;

then the consumer;

then choice;

then consumption;

then the body.

But the history does not end with the body.

The body does not merely bear the consequence.

It may alter the biological conditions of the next act.

The subject becomes hungry again.

Desires again.

Chooses again.

May try to stop.

May fail.

May try again.

May understand the adverse consequence and continue nevertheless.

At that point, 'weak-willed' is an easy verdict.

The scientific question is more difficult:

What is the condition of hunger, satiety, reward, craving, sleep, stress, and insulin regulation in the body performing this act?

The sociological question precedes it:

What food environment had already been constructed when this subject arrived at the table?

The ambush is not located only in front of the shelf.

It is not only in the history of the product.

It is not only in the moment of consumption.

It is not only behind the statistical threshold.

It extends from production to access, from access to the body, and from the body to the biological ground of the next act.

I recognise obesity, but I do not mistake it for a sufficient explanation.

The size of the body is not the name of the body's history.

I distinguish sucrose from HFCS, but I will not absolve one as 'natural' and manufacture the other as a singular metabolic enemy.

Their chemical difference does not eliminate the question of how repeated, high-dose free-sugar exposure is produced.

I recognise the molecule, but I will not make invisible the price, liquid form, product architecture, and field of access in which it is placed.

I recognise the unseen burden of food, but I will not convert detection of exposure into automatic proof of clinical harm.

Food may carry not only nutrients, but histories of production, processing, packaging, pricing, and distribution.

I recognise addiction-like consumption, but I will not translate it into moral weakness.

Loss of control, craving, unsuccessful efforts to stop, and continued consumption despite harm are measurable in human beings.

I read the literature, but I will not make its funding and the authorship of its questions disappear.

I use statistics, but I will not grant a statistical category authority to rule in place of the body.

I recognise psychosocial stress, but I will not reduce it to a one-way chain from cortisol to processed food.

Stress does not produce the same outcome in everyone. It may alter the probability of consumption through appetite, sleep, reward, and behaviour in some people.

I recognise the physician, but I will not mistake the clinical outcome for the beginning of the story.

I recognise the food engineer, because what is consumed is often not an unchanged agricultural input.

I recognise the agricultural scientist, because part of the material chain extends to production and soil.

I recognise economics and access, because what has been produced is not thereby available to everyone.

I recognise GLP-1-based treatment and do not deny its clinical benefit.

The capacity of semaglutide and tirzepatide to alter hunger, satiety, energy intake, body weight, and specified clinical outcomes demonstrates that the biological conditions of eating are not reducible to will.

But I will not substitute the medicine for a structural explanation.

The medicine does not, by itself, alter food prices, product formulation, access to nutritious food, working time, or the social sources of chronic stress.

Nor will I translate weight regain after withdrawal into automatic proof of weak will or pharmacological failure.

The end of a drug's effect is not the end of the biology it modified or of the environment it never changed.

And I recognise the subject:

neither as an entirely sovereign chooser,

nor as an entirely powerless victim.

The subject acts within a material world constructed before them - a world that may, under some conditions, reach into the biological capacity through which action itself is performed.

The sequence is therefore:

**SOIL → PRODUCTION → ACCESS → TABLE → BODY → ACTION →
PHENOTYPE → TREATMENT**

Soil and production condition the material beginning of food.

Industrial transformation constructs the final product.

Price and distribution organise the feasible set of options.

The subject makes tactical choices within that field.

Food enters the body.

The body responds.

That response may feed back into the conditions of later action.

The phenotype becomes visible.

Clinical intervention is directed at the body.

Pharmacology may alter the biological node while the constitutive food and social environment remains unchanged.

Conditions may be produced or intensified within systems; action is performed by the subject; consequences become visible in the body; treatment costs return to the subject and the health system.

Pharmacological individualisation arises here:

not because treatment is delivered to an individual, but because explanation and intervention are closed around the individual body.

This is the feedback.

This is the Metabolic Ambush.

The final judgements are these:

Diagnosis is not the beginning.

Statistics are not the body.

Normal glucose is not metabolic normality.

Insulin resistance is not merely diabetes.

Obesity is not the history of the body.

A dietary pattern is not a self-authored personal menu.

The similarity of sucrose and HFCS does not establish harmlessness; their difference does not establish the unique toxicity of HFCS.

Free-sugar exposure cannot be explained without considering molecule, dose, energy, matrix, repetition, and access together.

Addiction-like consumption is not merely a failure of will.

Food may carry environmental, industrial, and historical burdens that require measurement.

Detection of exposure is not proof of clinical harm at every dose.

Psychosocial stress does not determine action, but it may change its probability through biological and behavioural pathways.

A correct prescription is not necessarily an actionable prescription.

The clinical effect of GLP-1-based treatment is real; its structural limit is real too.

Medicine may alter the biological node; it cannot, by itself, transform the constitutive material world.

Treatment must not be rejected, but it must not replace investigation of the conditions.

Choice is real; the field of what can be chosen may also be constructed.

The act belongs to the subject; not all the conditions of the act are the subject's creation.

And therefore:

THE HISTORY OF HUMAN HEALTH DOES NOT BEGIN IN HOSPITAL.

The Metabolic Ambush lies not only in what a person eats, but in what they can access; in how the conditions that make them desire it again are produced; and in the statistical judgements through which their embodied reality is made visible or invisible.

Systems may produce or intensify the condition; the subject performs the act; the body bears the consequence; pharmacology intervenes at the biological node. Metabolic justice begins by treating disease without ceasing to ask why its costs are repeatedly transferred to the subject's body.

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Extended Keywords

Metabolic Ambush; Reality Paradigm; Constructed Food Reality; Statistical Judgement; Judgment Syntax; Compulsory Inclusion; Access-Conditioned Dietary Pattern; Insulin Resistance; Hyperinsulinaemia; Food Affordability; Food Environment; Free Sugars; Added Sugars; Sucrose; High-Fructose Corn Syrup; HFCS; Sugar-Sweetened Beverages; Food Matrix; Energy Surplus; Food Addiction; Ultra-Processed Food Addiction; Reward; Craving; Psychosocial Stress; Biological Preconditioning; Environmental Food Contaminants; PFAS; Microplastics; GLP-1 Receptor Agonists; Semaglutide; Tirzepatide; Treatment Withdrawal; Pharmacological Individualisation; Metabolic Cost Transfer; Obesity Critique; Metabolic Phenotype; Biological Depth; Constitutive Agency; Tactical Agency; Responsibility Transfer; Agricultural Soil; Food Engineering; Food Processing; Food Formulation; Onopticon; Homovictimus.