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New Zealand Charitable Trust

RECLAIMING HEALTH:

REVERSAL, REMISSION & REWIRING.

UNDERSTANDING & ADDRESSING THE PRIMARY DRIVERS OF NEW ZEALAND'S METABOLIC & MENTAL HEALTH CRISIS.

THE PHYSICIANS AND SCIENTISTS FOR GLOBAL RESPONSIBILITY NEW ZEALAND CHARITABLE TRUST (PSGRNZ).

ISBN 978-1-0670678-2-3

For 25 years,
PSGR New Zealand (PSGRNZ)
have provided scientific &
medical information &
analysis in the service of the
public's right to be
independently informed on
issues relating to
human & environmental
health.

Suggested citation.

PSGRNZ (2026) Reclaiming Health: Reversal, Remission & Rewiring. Understanding & Addressing the Primary Drivers of New Zealand's Metabolic & Mental Health Crisis. Bruning, J.R., Physicians & Scientists for Global Responsibility New Zealand. ISBN 978-1-0670678-2-3

PSGRNZ extends its sincere appreciation to the public health academics and medical practitioners who patiently and constructively contributed feedback across multiple iterations of this document.

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'The voice of nutrition in New Zealand is alarmingly quiet'.[1]

INTRODUCTION

PSGRNZ advances this Report as a catalyst for informed discussion and constructive debate on the health-reform responses required to address New Zealand's metabolic and mental-health challenges. The Report has two central aims: first, to synthesise the evidence identifying the key drivers of the global surge in metabolic and mental-health disorders; and second, to outline evidence-based pathways capable of restoring individuals, families, and communities to health.

A substantial body of scientific research now demonstrates that much of today's chronic disease burden is preventable and, in many cases, reversible. This is a consequential finding. It restores agency to individuals and communities and offers clinicians renewed purpose at a time when health systems remain heavily oriented toward symptom management rather than causal prevention.

Yet despite medical advances and increasing health system investment, public agencies and medical practitioners have been unable to reduce the burden of metabolic and mental illness. These conditions are now most commonly expressed as multimorbidity, the co-existence of multiple, interacting conditions. Legacy approaches, and the 'best international evidence' routinely relied upon by government, are increasingly contradicted by contemporary scientific literature that integrates nutrition, metabolism, inflammation, and neurobiology. This disconnect has material consequences for health outcomes, workforce participation, and public expenditure.

Nutrition is foundational to protecting metabolic and mental health, and nutrition plays a key role in recovery from common conditions. Both functions remain marginal in government health strategy. This Report examines the evidence showing that cumulative carbohydrate burdens, high intakes of ultra-processed foods, and widespread micronutrient insufficiencies play an outsized role in promoting New Zealand's chronic-disease epidemic. These factors contribute directly to hyperglycaemia, hypertension, systemic inflammation, and impaired brain function, operating upstream of many commonly diagnosed conditions.

The cumulative carbohydrate burden emphasises daily exposures to rapidly digestible starches, often in the form of processed carbohydrates, which affects not only the body but also the brain. A consistent body of evidence shows that reducing blood glucose and triglyceride levels, primarily through lowering carbohydrate intake, reduces risk not only of type 2 diabetes and cardiovascular disease, as well as many psychiatric and neurocognitive conditions. This evidence remains largely absent from official dietary policy.

Current dietary guidelines place disproportionate emphasis on carbohydrate intake while understating the physiological importance of fat and protein. As a result, many officials and members of Parliament remain insufficiently aware of the extent to which high-carbohydrate dietary patterns drive insulin spikes, hyperinsulinemia, and chronic inflammation. This omission persists despite clear evidence that insulin resistance and inflammatory pathways sit upstream of type 2 diabetes, hypertension, cardiovascular disease, periodontal disease, and many mental-health conditions, often concurrently.

Multimorbidity is the defining crisis. More New Zealanders now live with multiple chronic conditions than with any single diagnosis, and the associated costs, clinical, social, and economic, are super-additive. As metabolic and neurobiological dysfunction has intensified, prescribing rates have risen sharply. Yet polypharmacy is rarely associated with improved health or wellbeing and frequently compounds harm where medications address symptoms without resolving underlying dysfunction.

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[1] Coad J and Pedley K. (2020). Nutrition in New Zealand: Can the Past Offer Lessons for the Present and Guidance for the Future? *Nutrients,* 12:3433; DOI:10.3390/nu12113433

New Zealand's health system is increasingly out of step with the evidence base. Laws, policies, institutional cultures, and regulatory frameworks have not been systematically updated to reflect advances in metabolic and nutritional science. Officials have frequently relied on offshore 'consensus' positions that stabilise existing approaches rather than interrogate them. Independent, comprehensive reviews of the scientific literature have not occurred at scale, while science-policy and funding frameworks have created barriers to public-good research capable of addressing these gaps.

There is, however, significant scope for reform. One purpose of this Report is to contrast the breadth of evidence in the scientific literature with current Ministry of Health approaches. While health targets emphasise service access and utilisation, performance indicators increasingly focus on wellbeing. This policy-indicator misalignment leaves upstream prevention, particularly nutrition, largely out of scope. This is visible across white papers, ministerial briefings, consultation documents, and official responses to public inquiry.

PSGRNZ, as a New Zealand charitable trust, lays down a challenge:

New Zealand can reverse its metabolic and mental-health crisis,
but doing so requires rewiring health policy, general practice, and research systems.

Reform cannot occur if problems are acknowledged abstractly, without understanding how existing systems perpetuate them. Human biology is fundamentally dependent on vitamins and minerals, and officials have the discretion and responsibility to identify obstructive legislation and evaluate contemporary nutritional evidence. Accordingly, this Report identifies outdated scientific assumptions that no longer serve New Zealanders; highlights the absence of targeted biomarker screening that limits clinicians' ability to assess metabolic dysfunction, nutrient deficiency, or toxicity; and examines legislative settings that automatically classify nutrients as drugs once biochemical pathways are identified, an approach that can impede health-promoting interventions.

Reform is already underway in homes, clinics, and communities. The burden imposed by carbohydrate-rich diets has been scientifically and politically difficult to address, yet clinicians, health coaches, researchers, and patients have developed practical strategies for adopting nutrient-dense whole-food diets that are not burdensome, do not spike glucose or insulin and frequently reverse insulin resistance.

Effective reform must be multifaceted.

Health coaches provide personalised dietary support, recognising that individuals vary in their metabolic response to carbohydrates. Daily dietary decisions can mitigate, and often reverse, conditions long considered permanent, including type 2 diabetes. As glucose and insulin stabilise, a wide range of symptoms frequently subside, challenging the orthodoxy of 'one medication per symptom'. Health coaches can be integrated into clinical, hospital, and community settings, supporting individuals to navigate addictive food patterns that undermine health.

A broader reform agenda also requires the restoration and protection of academic and research freedom public good research for human and environmental health, alongside a willingness to challenge entrenched assumptions. Contemporary, interdisciplinary evidence from nutrition, metabolism, and neurobiology must be permitted to inform public policy. Biological and health-science curricula at primary, secondary, and tertiary levels require updating to reflect the central role of diet and metabolic health.

This Report concludes with recommendations for reform, including paradigm-shifting changes in how New Zealand approaches carbohydrate-heavy diets, insulin resistance, and hyperinsulinemia. The Reform section is structured in four parts: (1) diet-first approaches in local communities; (2) educational reform; (3) institutional and regulatory reform and (4) science-system reform.

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'This report is an important moment for New Zealand public health. For too long, the 'voice of nutrition' has been whispered when it should have been shouted. And then the whispers have focused on old, dated hypotheses and science that was flawed. The PSGRNZ rightly identifies that the bulk of our poor health, in both chronic disease and poor mental health is metabolic. It offers a clear blueprint for progress in addressing this. It offers a chance to reverse many chronic diseases and prevent them happening in the first place. These are goals long whispered in NZ and global health, but now we have the blueprint to get on with this important work.'

Grant Schofield PhD, Professor of Public Health, Director, Human Potential Centre. Auckland University of Technology, N.Z..

'The Physicians and Scientists for Global Responsibility have made clear the reasons for the worldwide pandemic of metabolic syndrome, and the role that the Western Diet plays in its pathogenesis. Fix the food and you fix health, healthcare, and society all at once.'

Robert Lustig, MD, MSL, paediatric endocrinologist, Emeritus Professor of Pediatrics, University of California, San Francisco, U.S.A..

'We need evidence-based system changes if we are to combat the twin epidemics of obesity and diabetes. I hope this report sparks needed conversation – and action.'

Leonardo Trasande, MD, MPP, Jim G. Hendrick, MD Professor of Pediatrics, Director, NYU Center for the Investigation of Environmental Hazards, Professor of Population Health, NYU Grossman School of Medicine, Professor of Health Policy, NYU Wagner School of Public Service, U.S.A..

'This document summarises the key science and clinical findings relating to the harms of excessive consumption of sugar, refined carbohydrates and ultra-processed foods. We have gone past the point where there can be any doubt that these food-like substances are at the heart of the multiple crises of chronic ill health both physical and mental. The burden of these effects is surely no longer tolerable to individuals or society. The answer is a simple one but will take concerted and consistent political will to implement. The same will it took to tackle the harms of tobacco. I hope New Zealand takes this chance to be a leader and show the rest of the World what can be achieved for its people.'

Dr Jen Unwin, BSc, MSc, DPsy, FBPsS. Chartered clinical and health psychologist with over 35 years experience, mostly in the NHS. Co-Founder, Food Addiction Solutions, U.K..

'The discovery that diabetes and poor metabolic health can be reversed by dietary means should have revolutionised modern medicine but hasn't. This report summarises much of the important evidence outlining the centrality of our diet for improving health and how we can be less reliant on drugs. I hope this document is a catalyst for the change in health policy we desperately need.'

Dr Simon Thornley, MBChB, MPH(hons), PhD, Senior Lecturer Epidemiology & Biostatistics, University of Auckland, N.Z..

'For over half a century, and over the same time period as the prevalence of obesity and obesity-related diseases skyrocketed, our nutrition recommendations have changed little. And yet, nutrition is recognized as a key contributor to these illnesses, and substantial research has supported changes to our nutritional approach for better health. This document by the PSGRNZ highlights both the long extant and the emerging evidence substantiating an approach that reduces starch and sugar intake, particularly the processed varieties, to reduce the burden of illness in New Zealand (and worldwide).'

William S. Yancy Jr., MD, Professor of Medicine. Medical Director, Duke Lifestyle & Weight Management Center, Co-director, Duke Primary Care Research Consortium, North Carolina, U.S.A..

'The majority of people in the developed world now have poor metabolic health, as defined by the presence of type 2 diabetes, central obesity, elevated blood pressure, raised triglycerides, or fatty liver disease. For example, O'Hearn et al., Journal of the American College of Cardiology, 2022. This nationally representative U.S. study found that in 2017–2018, only 6.8% of adults had optimal cardiometabolic health, meaning that over 93% had at least one abnormality in weight, blood pressure, glucose, lipids, or clinical cardiovascular disease, with the greatest declines seen in adiposity and glucose control over the past two decades. This is mainly a reflection of insulin resistance and in my practice responds best to controlling the intake of both ultra processed foods and refined carbohydrates. This including 'brown' bread. We have 32 peer reviewed papers on this, including one that shows carb restriction to be as good as GLP-1 injections (Wegovy).'

Dr David Unwin FRCGP. RCGP National Champion for Collaborative Care and Support Planning in Obesity & Diabetes, RCGP clinical expert in diabetes, Honorary Senior Lecturer, Edge Hill Medical School. Merseyside, UK. Founder member of The Public Health Collaboration

'What PSGRNZ articulates here aligns with what researchers increasingly recognize: chronic disease is fundamentally a metabolic problem, and dietary carbohydrate burden is a primary lever. New Zealand has an opportunity to lead.'

Benjamin T. Bikman, Ph.D. Professor, Department of Cell Biology, Brigham Young University, Utah, U.S.A..

'This report offers a clear, evidence-based framework for addressing diet-related chronic disease, an issue with significant health and economic impacts. Reclaiming Health defines how our current food environment contributes to illness and how existing policies can make progress challenging. Practical solutions are within reach. By taking thoughtful steps now, we have an opportunity to improve health outcomes, reduce healthcare costs, and support a stronger economy. Acting sooner rather than later will help ensure meaningful change.'

Julia Rucklidge, Professor of Psychology, Director of Te Puna Toiora, the Mental Health and Nutrition Research Lab at the University of Canterbury, N.Z..

'Congratulations to PSGRNZ for producing this paper which is an essential step in changing the understanding of the main causes of disease. This document will lead the transition from just managing the resulting symptoms to clinicians treating the underlying cause and empowering individuals to take control of their own health.'

Dr Glen Davies MBChB, Dip Obs, FNZCGP, FASLM, FACNEM

'To address the crisis in cardiometabolic health, I fully support PSGRNZ's call for a more adaptable approach to dietary guidance, one that is putting adequate nourishment first and is able to move beyond today's narrow definition of 'a healthy diet.'

Frédéric LEROY, Professor of food science & (bio)technology. Vrije Universiteit Brussel (VUB), Belgium.

'I cannot emphasize sufficiently that this report from the Physicians and Scientists for Global Responsibility is of biblical proportions. It is unquestionably the most carefully constructed and complete document ever compiled on this topic anywhere in the world. I can say this with both scientific conviction and personal authority. Because, between 2014 and 2018, I was subjected to a 4-year long public hearing in South Africa into my professional conduct for promoting, on social media, the exact dietary changes presented in this report. By presenting just a miniscule of the material contained herein, I was justly exonerated on all 13 charges. Reading this document leaves us with either of two choices. We either continue to travel the path of dietary iniquity. Or we do that which is right and just for the people of New Zealand (and ultimately the world). There is no other option. The evidence is now transparent; it is before your eyes. The choice is yours.'

Emeritus Professor Timothy David Noakes OMS, MBChB, MD, DSc, PhD (h.c.), FACSM (h.c.), FFSEM Ire (h.c.), FFSEM UK. Cape Town, South Africa.

CONTENTS

PART I. FOUNDATIONS: METABOLIC DYSFUNCTION & THE RISE OF MULTIMORBIDITY

1. THE PROBLEM: CASCADING METABOLIC IMPAIRMENT DRIVING MULTIMORBIDITY

The Global Burden of Disease Study calculates years lived with disability (YLDs), and years of life lost (YLLs) to arrive at disability-adjusted life-years (DALYs). The data continue to show escalating metabolic and mental health disease burdens across the world.

> *Globally between 2010 and 2021, among the 25 leading Level 3 causes, age-standardised DALY rates increased most substantially for anxiety disorders (16·7%), depressive disorders (16·4%), and diabetes (14·0%).*[2]

This is the first of two PSGRNZ Reports which lay a groundwork to suggest that dietary stressors play a major role in driving the unpredictable, complicated *and* complex outcomes that are prevalent in modern society, and which lead to over-burdened medical systems.

1. Diets high in refined carbohydrates and ultraprocessed food, which directly elevate blood glucose and lipid levels. The addiction-provoking impact on dopamine receptors can result in these diets being prioritised over nourishing foods which promote metabolic, including digestive tract health.
2. Insufficient levels of dietary micronutrients that are key cofactors in metabolic and mental processes.

PSGRNZ's Reports outline pathways for reform, highlighting prominent case studies, that can lead to reversal and mitigation of the complex conditions that individuals present with, when they visit their clinician or general practitioner (GP).

The metabolic and mental health crisis is global. But what does metabolic mean? The human *metabolism* is the coordinated network of chemical, cellular, and mitochondrial processes that sustain life and maintain the body's internal balance. Human *metabolic* function changes and adapts across the lifespan, influenced by developmental, hormonal, and physiological shifts.

Diet plays a key role in sustaining a healthy metabolism. Disruptions in gut function influence the metabolism through immune activation, altered microbial metabolites, and changes in nutrient absorption, affecting both brain function and mitochondrial health. Mitochondria are central regulators of cellular energy metabolism and redox balance, and emerging research increasingly links mitochondrial dysfunction to the pathophysiology of mental disorders.

These factors interact to suppress or provoke inflammatory and oxidative stress responses, which can also produce knock-on, cascading effects, further amplifying metabolic impairment across body systems.

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[2] Ferrari, Alize J *et al.* (2021) Global incidence, prevalence, years lived with disability (YLDs), disability-adjusted life-years (DALYs), and healthy life expectancy (HALE) for 371 diseases and injuries in 204 countries and territories and 811 subnational locations, 1990–2021: a systematic analysis for the Global Burden of Disease Study 2021. *The Lancet,* 403(10440)2133 - 2161

The interrelated factors rarely result in the diagnosis of a single condition. It is more common to have multiple conditions (multimorbidity) than a single condition.[3] People across the Western world are diagnosed at younger and younger ages with multiple conditions.

Multimorbidity, the co-occurrence of three or more chronic conditions[4], occurs a decade earlier in deprived communities.[5] [6] The societal cost of multimorbidity is super-additive.[7]

Metabolic syndrome is one example of how metabolic disturbances drive multimorbidity. Yet people who present with metabolic syndrome, which includes the overlapping conditions of hypertension, dyslipidaemia, type 2 diabetes mellitus, obesity, and inflammation, also are more likely to be diagnosed with a mental illness or brain-related disorder.[8] [9] [10] [11] Metabolic dysfunction is strongly correlated with risk for brain-related conditions and periodontal disease, yet these associations are rarely raised by health agencies.[12] [13]

In many individuals, metabolic disturbances precede the onset of psychiatric symptoms and formal diagnosis. Clinical presentation in a doctors' clinic by a patient, can reflect the complex interplay of genetic and epigenetic predispositions, dietary nutrient status, toxic exposures, and familial patterns. Lifestyle factors such as sleep disruption and physical inactivity further modulate this relationship, and the interaction is bidirectional: mental disorders can exacerbate metabolic dysfunction via neuroendocrine and behavioural pathways.[14]

Emerging lines of evidence indicate that the metabolic and mental-illness crisis is amplified by a class of food products, ultra-processed foods, that are engineered to be hyper-palatable and addictive. Ultra-processed foods can layer on top of diets that are already high in rapidly digestible starches.

Carbohydrate processing exists along a continuum, from minimally processed (e.g. unpeeled, cooked potatoes, rolled oats, and brown rice), to moderately processed (e.g. white rice or sourdough bread), and highly processed, refined carbohydrates, such as breakfast cereals, snack bars, crackers, and reconstituted breads (where the grain has been fractionated into refined flour, starch, bran, and germ).

Refinement increases glycaemic volatility, the risk of hyperinsulinaemia, and downstream metabolic stress, even when calorie content is similar. While minimally processed carbohydrate-based foods are generally compatible with health, regular intakes of moderately processed and refined carbohydrates

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[3] Russell *et al* (2019). Multimorbidity in Early Childhood and Socioeconomic Disadvantage: Findings From a Large New Zealand Child Cohort. *Academic Pediatrics,* 20(7),P619-627.
[4] Skou ST, Mair FS, Fortin M. *et al.* (2022). Multimorbidity. *Nat Rev Dis Primers* 8, 48. DOI: 10.1038/s41572-022-00376-4
[5] Head A, Fleming K, Kypridemos C, et al. (2021). Multimorbidity: the case for prevention *J Epidemiol Community Health* 2021;75:242–244. DOI:10.1136/jech-2020-214301
[6] Skou ST, Mair FS, Fortin M. *et al.* (2022). Multimorbidity. *Nat Rev Dis Primers*.
[7] Blakely T, Kvizhinadze G, Atkinson J, Dieleman J, Clarke P. (2019). Health system costs for individual and comorbid noncommunicable diseases: An analysis of publicly funded health events from New Zealand. *PLoS Med*. 16(1):e1002716. DOI: 10.1371/journal.pmed.1002716. PMID: 30620729.
[8] Otokunefor O, & Atoe K. (2025). The Nexus Between Metabolic Syndrome and Mental Health Disorders: A review. Open Journal of Medical Research (ISSN: 2734-2093), 6(1), 15-32. https://doi.org/10.52417/ojmr.v6i1.824
[9] John AP, Koloth R, Dragovic M, Lim SCB. (2009) Prevalence of metabolic syndrome among Australians with severe mental illness. MJA 2009; 190: 176–179
[10] Kim, JR., Kim, HN. & Song, SW. Associations among inflammation, mental health, and quality of life in adults with metabolic syndrome. *Diabetol Metab Syndr* 10, 66 (2018). DOI: 10.1186/s13098-018-0367-9
[11] Penninx, B. W. J. H., & Lange, S. M. M. (2018). Metabolic syndrome in psychiatric patients: overview, mechanisms, and implications. *Dialogues in Clinical Neuroscience*, *20*(1), 63–73. Doi: 10.31887/DCNS.2018.20.1/bpenninx
[12] Palmer CM. (2025) Beyond comorbidities: metabolic dysfunction as a root cause of neuropsychiatric disorders. *BJPsych* Advances. Published online 2025:1-3. doi:10.1192/bja.2024.74
[13] Gobin R, Tian D Liu Q Wang J. (2020). Periodontal Diseases and the Risk of Metabolic Syndrome: An Updated Systematic Review and Meta-Analysis. Front. Endocrinol. Volume 11. DOI:10.3389/fendo.2020.00336
[14] Palmer CM. (2025) Beyond comorbidities: metabolic dysfunction as a root cause of neuropsychiatric disorders.

create a cumulative metabolic burden, increasing the frequency of blood-glucose spikes. Ultra-processed foods are most strongly associated with food addiction.

Food addiction was first described in 1956. Food addiction is associated with dependency behaviours relating ultra-processed, refined, or high-glycaemic index carbohydrates. [15] [16]

Medical doctors often lament that their patients do not stick to a 'healthy diet'. Some foods, particularly those high in sugar and refined carbohydrates, can activate the brain's reward circuitry in ways that parallel mechanisms observed in substance addiction. This occurs in part through stimulation of the mesolimbic dopamine pathway, extending from the ventral tegmental area (VTA) to the nucleus accumbens, thereby reinforcing craving and repeated intake. Separately, chronic metabolic dysregulation (like sustained high insulin levels and associated leptin resistance) can impair normal appetite and reward signalling, potentially exacerbating dysfunctional eating behaviours; but the latter mechanism is part of broader metabolic research and is not established as one of the core addiction mechanisms.[17]

Ultraprocessed foods that are high in fats and refined carbohydrates, are the foods that are most closely associated with food addiction. Ultraprocessed foods are formulations of low-cost ingredients, mostly of exclusive industrial use, that result from a series of industrial processes. These processes involve the fractioning of whole foods into substances which are often derived from a few high-yield crops. Some of the substances can undergo hydrolysis, or hydrogenation, or other chemical modifications. Colours, flavours, emulsifiers and other additives are frequently added to make the final product palatable or hyper-palatable and ensure a long shelf life.[18]

Human bodies are not so much complicated as *complex*. The overlapping drivers work synchronistically to set human bodies on an illness trajectory: Systemic metabolic dysfunction gives rise to a cascade of symptoms and drives multimorbidity. Like any system with compromised structural integrity, the body's metabolic network becomes unstable and susceptible to cascading failures.

Ultraprocessed foods are not uniformly addictive, and treating them as one regulatory category is poorly supported by evidence and easily exploited by industry. A focus which reduces the cumulative burden of moderately processed and refined carbohydrates, to improve blood-sugar and insulin level, which takes account of individual insulin sensitivity, will concurrently result in less consumption of the ultraprocessed food groups that are most addictive, those that are high in sugar and refined carbohydrates.[19]

This Report draws from multiple levels of investigation, including cellular and mechanistic studies, case reports, cohort studies, audits of clinical data and population-level (epidemiological) research. The consistency and strength of findings are supported by systematic reviews and meta-analyses, which evaluate the consistency and strength of findings across studies. Meta-analyses help to determine whether the accumulated evidence supports, refutes, or remains inconclusive regarding a particular hypothesis.

Traditional dietary guidelines have not drawn from such a broad base of evidence, instead relying primarily on population-level epidemiological studies and randomised controlled trials.

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[15] Unwin J, Delon C, Giæver H, Kennedy C, *et al*. (2022). Low-carbohydrate and psychoeducational programs show promise for the treatment of ultra-processed food addiction. *Front. Psychiatry* 13:1005523. DOI: 10.3389/fpsyt.2022.1005523

[16] Sethi Dalai S, Sinha A, Gearhardt A. (2020). Low carbohydrate ketogenic therapy as a metabolic treatment for binge eating and ultraprocessed food addiction. *Curr Opin Endocrinol Diabetes Obes*. 27:275–82. DOI: 10.1097/MED.0000000000000571

[17] Lustig RH (2025). The battle over "food addiction". Front. Psychiatry 16:1621742. DOI:10.3389/fpsyt.2025.1621742

[18] Monteiro CA, Cannon G, Levy RB (2019). Ultra-processed foods: what they are and how to identify them. *Public Health Nutrition*, 22(5):936–941. DOI:10.1017/S1368980018003762.

[19] Ludwig DS (2025). Ultraprocessed Food on an Ultrafast Track. *NEJM* 393:1046-1049. DOI: 10.1056/NEJMp250869

Carbohydrate consumption as a driver of unstable blood glucose, elevated triglycerides (fat molecules in the blood), hyperinsulinemia and, ultimately, insulin resistance (the insulin pathway) is well established in the scientific literature. Insulin resistance is a byproduct of elevated insulin. This knowledge has prompted researchers and doctors to study or adopt low-carbohydrate approaches aimed at stabilising blood glucose and insulin and reducing triglyceride levels.[20] These insulin lowering diets, of which the ketogenic diet may be the most well-characterised, are based on low-carbohydrate, high-fat and moderate-protein foods. The ketogenic diet induces the production of ketone bodies by mimicking the breakdown of a fasting state.[21] [22]

From 2015 Onwards – Escalating Evidence.

In 2015 Richard Feinman and colleagues proposed that dietary carbohydrate restriction should be the first approach in diabetes management[23], arguing that a fundamental reappraisal of dietary recommendations was overdue. They cited several reasons:

1. General failure to halt the epidemic of diabetes under current guidelines.
2. The specific failure of low-fat diets to improve obesity, cardiovascular risk, or general health.
3. Constant reports of side effects of commonly prescribed diabetic medications, some quite serious.
4. Most importantly, the continued success of low carbohydrate diets to meet the challenges of improvement

Feinman *et al.* reasoned that this approach would lower blood glucose and reduce the risk of hyperglycaemia. They highlighted the role of increasing carbohydrate consumption in promoting obesity, weight loss is not the central issue (i.e. many people with type 2 diabetes are not overweight), yet when weight loss is required low-carbohydrate diets consistently outperformed low-fat diets for weight reduction. The authors further noted that replacing carbohydrates with protein is 'generally beneficial', and they challenged long-standing assumptions about dietary fat, arguing that total and saturated fat intake do not correlate with cardiovascular disease risk. They emphasised that 'plasma saturated fatty acids are controlled by dietary carbohydrate more than by dietary lipids' pointing to a metabolic rather than dietary origin. Crucially, the authors emphasised:

> *Adherence to low-carbohydrate diets in people with type 2 diabetes is at least as good as adherence to any other dietary interventions and is frequently significantly better.*

Despite presenting a substantial evidence base and identifying multiple downstream benefits, the conclusions of Feinman *et al.* have not been incorporated into major clinical guidelines. In the decade 2015-2025, their position has been corroborated by an expanding body of trials and mechanistic studies that demonstrate that people experience improved glycaemic control, reduced insulin requirements, and favourable lipid profiles under low-carbohydrate and ketogenic dietary patterns.

The Feinman paper does not appear to be referenced in Ministry of Health guidance or related agency documents. Yet New Zealand researchers did recognise the potential for a different approach to diabetes management and population health. Notably, Professor Grant Schofield and colleagues at Auckland University of Technology published a paper in the New Zealand Medical Journal supporting a low-

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[20] Unwin D. (2024). Reducing overweight and obesity; so how are we doing? *BMJ Nutrition, Prevention & Health*. 2024;:e000836. DOI:10.1136/bmjnph-2023-000836

[21] Nojek P, Zawół M, Zimonczyk M, *et al.* (2024) Ketogenic diet and metabolic health: A review of its impact on type 2 diabetes and obesity. Analysis of research on the ketogenic diet in the context of treating metabolic disorders. J Educ Health Sport. 2024;71:55923. DOI: 10.12775/JEHS.2024.71.55923.

[22] Baylie T, Ayelgn T, Tiruneh M, Tefsa KH (2024). Effect of Ketogenic Diet on Obesity and Other Metabolic Disorders: Narrative Review. *Diabetes, Metabolic Syndrome and Obesity,* 17:1391–1401, DOI: 10.2147/DMSO.S447659.

[23] Feinman RD, Pogozelski WK, Astrup A *et al.* (2015). Dietary carbohydrate restriction as the first approach in diabetes management: Critical review and evidence base. *Nutrition,* 31:1-13. DOI: /10.1016/j.nut.2018.12.002

carbohydrate approach and challenging the prevailing high-starch, low-fat dietary guidelines. The paper concluded:

> *We suggest that clinical dietary advice for the treatment of diabetes, as well as population prevention guidelines, be urgently revised.*[24]

New Zealand researchers have tried to raise attention to the carbohydrate (or starch) burden as a driver of poor metabolic health. Low-carbohydrate dietary responses to high blood glucose and triglyceride levels first reached mainstream media attention in New Zealand when Professor Grant Schofield and Dr Caryn Zinn, and chef Craig Dodger published the book *What The Fat* (WTF) book in 2015. The book was controversial, yet received critical acclaim, and has since been republished in various formats. The book remains widely available.

A few years earlier, New Zealand researchers theorised that carbohydrate cravings were associated with the dopaminergic pathway and food addiction. [25] [26] [27] In a groundbreaking paper, New Zealand researchers also explored the role of dopamine in reward and psychosis, considering the potential use of food as a substitute to induce dopamine release, which would then contribute to the weight gain that commonly follows antipsychotic drug use, in people with psychotic illness. The authors speculated that:[28]

> *'food may be a key stimulant of this disordered pathway, and altering diet may improve psychosis and reduce the need for antipsychotic treatment. If blocking the effects of free dopamine reduces psychotic symptoms, then reducing dopamine release is likely to induce a similar effect.'*

Although low-carbohydrate and food addiction research were identified as promising fields for investigation from 2008 onwards, and received a major 'injection' in 2015, some ten years later, these lines of research have not been extensively pursued by New Zealand's public universities, including public health and medical faculties, despite advancing population-level burdens of metabolic disease.

A substantial and robust body of research now reveals that type 2 diabetes mellitus (T2DM) is neither inevitably chronic nor irreversible, and that early reversal of prediabetes and T2DM is both feasible and associated with wider health benefits.[29] [30] [31] As poor diets frequently precede multimorbidity and multifactorial disease states, when diet and nutrition is addressed, a serendipitous, domino effect can occur and other health markers can improve, as blood glucose levels improve.

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[24] Schofield G, Henderson G, Thornley S, Crofts C. (2016) Very low-carbohydrate diets in the management of diabetes revisited. *NZMJ*, 129:1432. ISSN 1175-8716.

[25] Thornley, S.; McRobbie, H.(2009). Carbohydrate withdrawal: is recognition the first step to recovery? N. Z. Med. J., 2009, 122, 133-134.

[26] Thornley, S.; McRobbie, H.; Eyles, H.; Walker, N.; Simmons, G. (2008). The obesity epidemic: is glycemic index the key to unlocking a hidden addiction? *Med. Hypotheses*, 71, 709-714.

[27] Thornley, S, McRobbie H. (2011). Sickly Sweet: Sugar, Refined Carbohydrate, Addiction and Global Obesity (Nutrition and Diet Research Progress). Nova Novinka.

[28] Thornley S, Russell B and Kydd R. (2011) Carbohydrate reward and psychosis: an explanation for neuroleptic induced weight gain and path to improved mental health? *Curr Neuropharmacol*. 9(2):370-5

[29] Unwin D, Khalid AA, Unwin J, Crocombe D, Delon C, Martyn K, *et al*. (2020). Insights from a general practice service evaluation supporting a lower carbohydrate diet in patients with type 2 diabetes mellitus and prediabetes: a secondary analysis of routine clinic data including HbA1c, weight and prescribing over 6 years. *BMJ Nutr Prev Health*. 3:285–94, DOI:10.1136/bmjnph-2020-000072

[30] Unwin D, Delon C, Unwin J, *et al*. (2023). What predicts drug- free type 2 diabetes remission? Insights from an 8- year general practice service evaluation of a lower carbohydrate diet with weight loss. *BMJ Nutrition, Prevention & Health* 2023;0:e000544. DOI:10.1136/bmjnph-2022-000544

[31] Zinn C, Campbell JL, Fraser L, Davies G, Hawkins M, Currie O, Cannons J, Unwin D, Crofts C, Stewart T, et al. (2025) Carbohydrate Reduction and a Holistic Model of Care in Diabetes Management: Insights from a Retrospective Multi-Year Audit in New Zealand. *Nutrients*.17(24):3953. DOI:10.3390/nu17243953

Studies consequently show that health coaching can be integrated into everyday clinical practice, to support shifts away from poor dietary habits and addictive patterning that can overwhelm and hinder the best of intentions. Health coaching interventions apply a three-pronged approach: whole food, carbohydrate reduction; a health coach, behaviour-change-based delivery approach; and community- or peer-based initiatives.[32] Health coaching has been integrated into New Zealand Primary Health Organisations (PHOs). However, the current PHO work-scope does not extend to diet and nutrition coaching. In contrast, the health coaching that is discussed in this paper, explicitly integrates diet and nutrition support and education, with the central objective of improving metabolic, including mental health.

The January 2026 U.S. Dietary Guideline Shift.

Dietary guidelines not only shape the everyday choices of the general public; they also guide public-sector catering decisions and clinical advice, with effects that reverberate across society. Menus developed for government institutions, including schools, hospitals, and the military, are typically designed to align with guideline directives, while dietary recommendations by medical practitioners will, by convention, adhere to guidelines.

On 7 January 2026, the United States (US) Department of Health and Human Services (HHS) and the US Department of Agriculture (USDA) released substantially revised dietary guidelines, the 'most significant reset of federal nutrition policy in decades'.[33]

> *American households must prioritize diets built on whole, nutrient-dense foods—protein, dairy, vegetables, fruits, healthy fats, and whole grains.* [34]

HHS and USDA scientific and promotional materials draw attention to important but historically under-examined issues relating to macronutrient (protein, fat and carbohydrate) intake and the health impact of industrial processing. The *Overview of Evidence Accepted and Rejected from the Dietary Guidelines Advisory Committee (DGAC) Report* illustrates the extent to which many previously taken-for-granted 'healthy options' were rejected by the Advisory Committee as inconsistent with current evidence.[35]

The Scientific Foundation for the Dietary Guidelines for Americans, 2025–2030 clarifies several concepts that have previously been under-represented or downplayed. It notes, for example, that many foods described as 'healthy carbohydrates' are more accurately classified as refined grains, and that many low-fat products are highly processed and may therefore be less nourishing than less-processed alternatives. The *Scientific Foundation* paper acknowledges that legacy guidelines and health claims may have inadvertently directed people away from healthier products to less healthy products. They cited the example of minimally processed, full-fat yoghurt with no additives, which, when reformulated as 'low-fat' or 'fat-free', typically incorporates added sugars, starches, and other chemical additives. The document advises against artificial ingredients and advocates against added sugars, including their widespread inclusion in grain-based snack foods that were formerly viewed as healthy.

The U.S. guideline shift marks a constructive departure from the historic over-emphasis on carbohydrates and the relative under-recognition of the health benefits of protein and healthy fats. Historically, the U.S. acceptable macronutrient distribution range (AMDR) was established to balance competing metabolic considerations within a physiologically acceptable range. For example, in the 2005 dietary reference intake discussion, diets very high in carbohydrates were acknowledged to increase risk for coronary heart disease (CHD) and T2DM:

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[32] Zinn C, Campbell JL, Fraser L. *et al*. (2025) Carbohydrate Reduction & a Holistic Model of Care in Diabetes Mngmnt.
[33] HHS (January 7, 2026). Kennedy, Rollins Unveil Historic Reset of U.S. Nutrition Policy, Put Real Food Back at Center of Health. https://www.hhs.gov/press-room/historic-reset-federal-nutrition-policy.html
[34] HHS & USDA (Jan 2026). Dietary Guidelines for Americans, 2025–2030. Page 2. https://cdn.realfood.gov/DGA.pdf
[35] HHS & USDA (2026). The Scientific Foundation for the Dietary Guidelines for Americans, 2025–2030. P.iii-viii. https://cdn.realfood.gov/Scientific%20Report_1.8.26.pdf

> *High carbohydrate diets frequently cause greater insulin and plasma glucose responses than do low carbohydrate diets. These excessive responses theoretically could predispose individuals to the development of type 2 diabetes because of prolonged overstimulation of insulin secretion'; versus the risk of weight increase from excess fats in the diet.* [36]

At the same time, concerns were raised in the 2005 paper about excessive dietary fat contributing to weight gain.

As the *Scientific Foundation* states, the recommended daily allowance/intake levels and the AMDR serve complementary purposes:

> *The RDA prevents deficiency (e.g., preventing loss of lean body mass or negative nitrogen balance), while the AMDR identifies a range of intakes compatible with health and nutrient adequacy.* [37]

Importantly, they distinguish between the longstanding objective of preventing nutrient deficiency and the emerging evidence on intake levels, by age, sex, and life stage, that support optimal health. The AMDR framework recognises that intakes above minimum deficiency-prevention thresholds may confer additional health benefits.

Figure 1. Dietary Guidelines for Americans, 2025–2030. January 2026.

The revised guidelines place renewed emphasis on protein as an essential macronutrient and re-establish a broad intake range compatible with health. This represents a subtle but important shift. These shifts provide U.S. government institutions and medical practitioners with greater latitude in dietary planning and clinical guidance.

Historically, attention has tended to focus on the lower end of the acceptable macronutrient distribution range (AMDR) for protein, around 10% of total energy intake. The updated position recognises that protein intakes across a wider range, from 10–35% of total energy, can support maintenance of lean mass and metabolic health. [38] (New Zealand dietary guidelines do not appear to specify a comparable macronutrient distribution range.)

[36] Institute of Medicine. 2005. Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids. Washington, DC: The National Academies Press. DOI: 10.17226/10490. p.784.
[37] HHS & USDA (2026). The Scientific Foundation for the Dietary Guidelines for Americans, 2025–2030. P.37.
[38] HHS & USDA (2026). The Scientific Foundation for the Dietary Guidelines for Americans, 2025–2030. P.37-40.

In practice, adults roughly consume 1.0 g/kg per day of protein. By comparison the recommended daily allowance for protein for U.S. adults 18 years and over has historically been set at 0.8 g/kg[39], while the New Zealand and Australian reference values range from 0.84-0.94 g/kg.[40]

The new guidelines place heightened emphasis on distinguishing whole grains from refined grain products, explicitly outlining the health risks associated with refined carbohydrates that often contain added sugars and industrial additives. They recommend substantial reductions in highly processed carbohydrates and reduce recommended whole-grain intake to 2-4 servings per day.

The *Scientific Foundation* provides practical clarity on how whole grains may be differentiated from less healthy refined carbohydrate products, noting that *'most true whole-grain foods contain at least 1 gram of fibre for every 8 grams of carbohydrate.'*[41] Notably, neither the *Dietary Guidelines for Americans* nor the *Daily Servings by Calorie Level* reference 'cereal' as a recommended category.[42] [43] Instead, breakfast cereals are more commonly characterised as refined or processed foods to be limited or avoided.

Importantly, the Appendices recognise that a low-carbohydrate dietary pattern is scientifically justified option for people who are overweight or obese with metabolic syndrome or T2DM.[44]

Unrefined saturated fats are increasingly positioned as compatible with health. The new guidelines endorse full-fat milk and more generally frame unprocessed fats as health-supportive.:

> *'Healthy fats are plentiful in many whole foods, such as meats, poultry, eggs, omega-3-rich seafood, nuts, seeds, full-fat dairy, olives, and avocados. When cooking with or adding fats to meals, prioritize oils with essential fatty acids, such as olive oil. Other options can include butter or beef tallow.*

Despite this shift in framing, the formal recommendations remain constrained by legacy limits.:

> *In general, saturated fat consumption should not exceed 10% of total daily calories.*

This threshold reflects the position in the 2020–2025 Dietary Guidelines, which encouraged substitution of meats, butter, and dairy with a wide range of plant-based alternatives.[45] However, the 10% limit appears inconsistent with the evidence reviewed by the Advisory Committee and with Scientific Foundation statements and the analyses presented in the appendices.[46] For example, *The Scientific Foundation* states:

> *Overall, the RCT evidence does not provide causal support for reducing saturated fat below 10% of energy or replacing saturated fat with linoleic acid–rich oils to prevent CHD or death*.[47]

The information contained in the Appendices provide the scientific evidence that underpins guidelines positions. Two separate views in the Appendices found that current saturated fat recommendations which limit intake to below 10% of total daily calories have little scientific foundation. The first review (Appendix

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[39] Wolfe RR, Cifelli AM, Kostas G, Kim IY. (2017). Optimizing protein intake in adults: Interpretation and application of the Recommended Dietary Allowance compared with the Acceptable Macronutrient Distribution Range. *Adv Nutr.* 8(2):266–275. DOI:10.3945/an.116.013821

[40] NHMRC (2005). Nutrient Reference Values for Australia and New Zealand Including Recommended Dietary Intakes. Protein. https://www.eatforhealth.gov.au/sites/default/files/2022-10/n35-protein_0.pdf

[41] HHS & USDA (2026). The Scientific Foundation for the Dietary Guidelines for Americans, 2025–2030. P.20.

[42] USDA (January 2026). Dietary Guidelines for Americans, 2025–2030. https://cdn.realfood.gov/DGA.pdf

[43] HHS & USDA (2026). Daily Servings By Calorie Level. https://cdn.realfood.gov/Daily%20Serving%20Sizes.pdf

[44] HHS & USDA (2026). The Scientific Foundation For The Dietary Guidelines For Americans. Appendices. P.199 https://cdn.realfood.gov/Scientific%20Report%20Appendices_1.8.26.pdf

[45] 2025 Dietary Guidelines Advisory Committee. 2024. Scientific Report of the 2025 Dietary Guidelines Advisory Committee: Advisory Report to the Secretary of Health and Human Services and Secretary of Agriculture. U.S. Department of Health and Human Services. Page 5. DOI: 10.52570/DGAC2025

[46] HHS & USDA (2026). The Scientific Foundation For The Dietary Guidelines For Americans. Appendices. P.22-40.

[47] HHS & USDA (2026). The Scientific Foundation for the Dietary Guidelines for Americans, 2025–2030. Page 30. https://cdn.realfood.gov/Scientific%20Report_1.8.26.pdf

4.6) analysed randomised controlled trials to identify causal evidence that saturated fat intakes below 10% of total energy prevent coronary heart disease or all-cause mortality. The authors found no convincing evidence to support this hypothesis. [48]

The second review (Appendix 4.7) adopted a different approach, using Bayesian methods to examine whether saturated fat intake influenced all-cause mortality or cardiovascular disease (CVD) risk, including stroke. This latter review found that analyses and systematic reviews consistently conflated the more atherogenic trans fats, which are known to increase CVD risk, with saturated fats, and that the evidence base could not distinguish saturated fat effects from those of trans fatty acids. [49]

Following the release of the new guidelines, nutritionist Nina Teicholz, founder of the U.S.-based Nutrition Coalition, argued that retention of the 10% saturated-fat cap may disproportionately affect populations reliant on government food programmes, as these settings are most likely to restrict saturated fats while continuing to rely on refined seed oils. Teicholz further notes that the guidelines' positive framing of unprocessed fats is difficult to reconcile in practice, i.e., in the daily diet. For example, she observes that consuming one cup of full-fat yoghurt together with a chicken thigh cooked with the skin on in a tablespoon of butter would bring daily saturated-fat intake close to the recommended ceiling.

Teicholz also highlights an internal tension in the guidelines, arguing that the 10% threshold creates a paradox: meeting higher protein targets through commonly consumed whole-food sources such as beef, pork, or chicken thighs with skin would exceed the saturated-fat limit early in the day.[50]

The new U.S. guidelines, and the scientific evaluations that accompany them, provide renewed analytical attention to issues that have previously received limited emphasis and that are relevant to individual and population health outcomes. The release of the U.S. guidelines immediately prior to the launch of PSGRNZ's Reclaiming Health paper is notable, as the underlying *Scientific Foundation* analyses in many respects both reflect and independently corroborate key elements of the evidence base and reasoning advanced in this New Zealand-based work.

2. THE TOTAL CARBOHYDRATE BURDEN & INDIVIDUAL VULNERABILITY.

This *Reclaiming Health* paper places a strong emphasis on cumulative carbohydrate burdens, because a growing body of international scientific and clinical literature identifies excess dietary carbohydrate intake of moderately processed and refined carbohydrates as a central driver of metabolic syndrome. Strong evidence suggests that this pattern has been reinforced by dietary guidelines that have historically encouraged carbohydrate consumption while understating the role of healthy fats and proteins.

As outlined in the Reform section, a key opportunity lies in recalibrating dietary guidelines to bring fats and proteins back into public view, alongside the integration of health coaching and substantial reinvestment in nutrition education. This would represent a shift away from guideline frameworks primarily designed to prevent nutrient deficiency toward approaches which may support satiety, appetite regulation, and metabolic stability, optimise metabolic health across the life course, and reduce risk for obesity.

Importantly, the paper does not argue that obesity is driven exclusively by carbohydrate intake. Excess energy consumption can and does contribute to weight gain. Rather, *Reclaiming Health* situates carbohydrate exposure within a broader scientific landscape. Multiple models seek to explain obesity pathogenesis. These include the energy balance model (EBM), the carbohydrate-insulin model (CIM), [51] and

[48] HHS & USDA (2026). The Scientific Foundation For The Dietary Guidelines For Americans. Appendices. P.230.
[49] HHS & USDA (2026). The Scientific Foundation For The Dietary Guidelines For Americans. Appendices. P.248-259.
[50] Teicholz N (Jan 7, 2026). Butter Is Not Back: The Broken Promise on Saturated Fats. Substack.
[51] Hall, KD, Sadaf Farooqi I, Friedman JM et al (2022). The energy balance model of obesity: beyond calories in, calories out. The American Journal of Clinical Nutrition. 115(5):1243-1254. DOI: /10.1093/ajcn/nqac031

the more recently articulated REDOX model.[52] Taken together, the EBM, CIM and REDOX models offer complementary insights into the pathophysiology of obesity, rather than mutually exclusive explanations.

The EBM posits that changes in food environments, particularly the widespread availability and aggressive marketing of inexpensive, energy-dense, ultra-processed, and highly palatable foods, have driven obesity by increasing consumption beyond physiological energy requirements. Foods that are low in fibre and protein and offered in large portion sizes are thought to disrupt neural satiety signalling. The EBM highlights that external food-related cues, and the properties of the foods themselves, disrupt healthy neural signalling, and result in excess consumption and promoting excess accumulation of body fat.[53]

By contrast, the carbohydrate–insulin model (CIM) of obesity metabolic consequences of diets that are predominantly high in refined, rapidly digestible carbohydrate-containing foods, including fructose rich foods and beverages. This model proposes that such diets alter fuel partitioning, directing energy substrates away from oxidation and toward storage in adipose and other tissues, thereby promoting fat accumulation even in the absence of overt caloric excess.[54]

The REDOX model adds a further layer of explanation, proposing that obesity may arise from disturbances in cellular and systemic redox balance. Altered oxidation–reduction signalling can disrupt metabolic and hormonal regulation, interfere with insulin action and energy sensing, and impair adipose tissue function. In this framework, obesity emerges from perturbed signalling and feedback mechanisms shaped by environmental, dietary, and metabolic exposures, rather than from caloric excess alone.[55]

PSGRNZ's emphasis on the carbohydrate-insulin model is not driven solely by obesity pathophysiology, but by the breadth of evidence linking excess carbohydrate intake to elevated insulin, triglycerides, blood pressure, and adverse mental-health outcomes. Accordingly, *Reclaiming Health* differs from conventional approaches by drawing on mechanistic studies alongside case series, cohort data, and real-world clinical evidence to illustrate the multifactorial nature of metabolic and mental ill-health. Obesity may precede the other markers of metabolic syndrome, but in many cases, the other markers precede obesity.

The paper also highlights an under-examined dimension: addictive eating patterns associated with cumulative refined carbohydrate exposure, including but not limited to ultra-processed foods. These patterns contribute substantially to non-adherence to dietary guidelines, often precede metabolic syndrome and obesity, yet remain poorly integrated into academic and policy frameworks. While fats and proteins have been progressively marginalised within dietary guidance, the EBM model of obesity offers limited insight into how individuals might sustainably manage hunger and satiety through caloric restriction, particularly in the presence of glycaemic volatility and reward-driven, addictive eating behaviours.

While there is sound scientific evidence supporting greater regulation of sugar, defaulting to an exclusive focus on ultra-processed foods (UPFs) as a discrete regulatory target risks creating a political quagmire, as not all UPFs are addictive.

As such, many of the recommendations for reform, outlined in Chapter 12, extend well beyond an individual-level approach. The proposed pathways for reform are intended to improve key metabolic parameters that increase risk across a spectrum of metabolic and mental health conditions. Evidence

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[52] Heindel JJ, Lustig RH, Howard S. et al. (2021). Obesogens: a unifying theory for the global rise in obesity. *Int J Obes* 48, 449–460. DOI:10.1038/s41366-024-01460-3

[53] Magkos, F., Sørensen, T.I.A., Raubenheimer, D. et al. (2024). On the pathogenesis of obesity: causal models and missing pieces of the puzzle. Nat Metab 6, 1856–1865 (2024). DOI:10.1038/s42255-024-01106-8

[54] Magkos, F., Sørensen, T.I.A., Raubenheimer, D. et al. (2024). On the pathogenesis of obesity: causal models and missing pieces of the puzzle. Nat Metab 6, 1856–1865 (2024). DOI:10.1038/s42255-024-01106-8

[55] Heindel JJ, Lustig RH, Howard S. et al. (2021). Obesogens: a unifying theory for the global rise in obesity.

examined in the paper suggests that the approaches set out in the *Reform* section can deliver substantial metabolic and health benefits, again with particularly strong effects in lower-income populations.

PSGRNZ's recommendations involve structural changes designed to support dietary transition across New Zealand. The evidence reviewed indicates that these approaches deliver significant gains, particularly among lower-income groups.

The Carbohydrate-Insulin Model.

Nearly two billion people worldwide are now overweight or obese.[56] The conventional understanding holds that obesity drives type 2 diabetes mellitus (T2DM), and that together, these conditions set off a cascade of risks for a striking array of diseases and syndromes. In practice, insulin resistance develops first in obese and non-obese people. Beta cells are then unable to compensate.[57] However, as New Zealand doctors reversing diabetes have found, beta cell 'relative' failure is rare. Their experience aligns with other research which shows that pancreatic beta cell function and insulin secretion improves with dietary shifts.[58]

When eaten in quantities that exceed immediate energy demands and glycogen storage capacity, starchy carbohydrates are readily converted into body fat, fuelling weight gain and metabolic strain. An increasing range of randomised control trials support the suggestion that reductions in carbohydrate rather than low-fat intakes, may be more strongly associated with weight loss and reduction in risk for obesity.[59]

Dietary carbohydrates include sugars and starches, both of which are ultimately broken down in the digestive tract by enzymes into their simplest form, glucose. Starches vary in their digestibility: they may be rapidly digestible, slowly digestible, or resistant to digestion, and their characteristics can be significantly altered by domestic cooking methods and industrial processing. As with sugars, excessive consumption of rapidly digestible starches is associated with adverse health outcomes. Understanding differences in starch digestibility, and how quickly blood glucose levels rise after consuming starchy foods, is an important aspect of public health.[60]

The resultant glucose is absorbed into the bloodstream, raising blood sugar levels (reflected in HbA1c over time). In response, the pancreas releases insulin, a hormone that signals cells to take up glucose for immediate use or storage. Muscle and other body tissues use glucose as a rapid fuel source, particularly during physical activity, while liver and muscle cells store excess glucose as glycogen, a compact and readily mobilised energy reserve.

However, the body's glycogen storage capacity is limited. Once these reserves are full, continued high glucose availability, especially from carbohydrate-based meals and snacks, triggers the liver to convert excess glucose into fatty acids through a process known as *de novo* lipogenesis (DNL). These fatty acids are combined with glycerol to form triglycerides, which are packaged by the liver into very-low-density lipoproteins (VLDL) and released into the bloodstream for transport as energy.

Triglycerides in the bloodstream are a normal component of metabolism, serving as a transport form of fat for energy use or storage. After a meal, dietary fats are absorbed by intestinal cells and packaged into chylomicrons, large, triglyceride-rich lipoprotein particles that enter the circulation via the lymphatic

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[56] World Obesity Federation (October 2023). World obesity atlas 2023. *World Obesity.* https://www.worldobesity.org/resources/resource-library/world-obesity-atlas-2023

[57] Rachdaoui N. (2020). Insulin: The Friend and the Foe in the Development of Type 2 Diabetes Mellitus. International *Journal of Molecular Sciences.* 21(5):1770. DOI:10.3390/ijms21051770

[58] Lim EL, Hollingsworth K, Aribisala BS, *et al.* (2011). Reversal of type 2 diabetes: Normalisation of beta cell function in association with decreased pancreas and liver triacylglycerol. *Diabetologia,* 54:2506–2514. DOI: 10.1007/s00125-011-2204-7

[59] Public Health Collaboration. Survey of Randomised Control Trials Comparing Low-Carb Diets Of Less Than 130g Carbohydrate Per Day To Low-Fat Diets Of Less Than 35% Fat Of Total Calories. https://phcuk.org/evidence/rcts/

[60] Yang Z, Zhang Y, Wu Y, Ouyang J. (2023). Factors influencing the starch digestibility of starchy foods: A review. *Food Chemistry.* 406:135009. DOI:10.1016/j.foodchem.2022.135009

system. At the same time, the liver produces VLDL to carry triglycerides synthesised from excess carbohydrate or protein. These particles circulate through the bloodstream, where triglycerides may be delivered to adipose tissue for long-term storage as body fat or utilised by muscle tissue as an alternative energy source when required.[61] [62] [63] [64]

Summary papers addressing the physiological role of VLDL are scarce. Normal physiological function is typically treated as background knowledge and not considered worthy of synthesis. Journals, funding bodies, and regulators have historically prioritised disease endpoints, hazard identification, and modifiable risk factors. In physiological terms, VLDL secretion is a normal and essential hepatic function that protects hepatocytes from lipid overload, distributes endogenously synthesised energy substrates to peripheral tissues, and supports cellular membrane integrity and mitochondrial function; pathology arises primarily from chronic dysregulation of VLDL production and clearance rather than from VLDL itself.

VLDL is upstream of low-density lipoproteins (LDL). LDL is largely a metabolic product of VLDL following triglyceride removal. LDL play important physiological roles that are often downplayed. The primary cargo of LDL is cholesterol, and its principal physiological function is to deliver cholesterol for incorporation into cell membranes, synthesis of steroid hormones and bile acids, and support of myelin and synaptic function. There is no fixed biological amount of cholesterol per LDL particle and LDL particles vary widely in size and cholesterol content. An excess LDL burden will often reflect upstream VLDL dysregulation rather than a primary cholesterol excess.

The physiological roles of VLDL and LDL described here are foundational to lipid biochemistry and human metabolism and are widely accepted in the scientific literature; however, contemporary reviews overwhelmingly frame these lipoproteins through disease-risk paradigms rather than synthesising their normal biological functions.

Both injected insulin and pancreas-derived insulin promote the transport of triglycerides from the bloodstream into tissues, predominantly adipose tissue, even when the original dietary source is carbohydrate rather than fat. The precise mechanisms remain incompletely understood.

Insulin, the master regulator of energy metabolism, is central to this process. Insulin not only lowers blood glucose by facilitating its uptake, insulin also promotes fat storage by encouraging triglyceride uptake into fat cells, inhibiting fat breakdown. Under healthy conditions, this clearance is efficient, and triglyceride-rich lipoproteins do not remain elevated for long.

Over time, consistently high carbohydrate intakes, particularly from refined, high-glycaemic sources, can lead to chronically elevated compensatory insulin levels (hyperinsulinaemia), increased circulating triglycerides, and progressive fat accumulation. If carbohydrate intake (and thus hyperinsulinaemia) is not meaningfully reduced, this process can continue until pancreatic insulin secretion becomes impaired, resulting in persistently elevated blood glucose (HbA1c) and triglyceride levels.

As circulating triglyceride concentrations rise, the risk and severity of poorly controlled type 2 diabetes mellitus (T2DM) increase, alongside heightened risk of atherosclerotic disease, arterial occlusion, and

[61] Samuel VT, Shulman GI. (2016). The pathogenesis of insulin resistance: integrating signaling pathways and substrate flux. *J Clin Invest.,* 126(1):12-22. DOI: 10.1172/JCI77812. Epub 2016 Jan 4. PMID: 26727229; PMCID: PMC4701542.
[62] Li, M., Chi, X., Wang, Y. *et al.* (2022). Trends in insulin resistance: insights into mechanisms and therapeutic strategy. *Sig Transduct Target Ther., 7*:216. DOI:10.1038/s41392-022-01073-0
[63] Samuel VT, Schulman GI (2012). Mechanisms for Insulin Resistance: Common Threads and Missing Links. *Cell*. 148(5):852-871. DOI: 10.1016/j.cell.2012.02.017.
[64] Lee SH, Park SY, Choi CS. (2022). Insulin Resistance: From Mechanisms to Therapeutic Strategies. *Diabetes Metab J,* 46(1):15-37. DOI: 10.4093/dmj.2021.0280.

myocardial infarction.[65] [66] Scientists have increasingly questioned the marginal benefits of many cholesterol-lowering drugs when cholesterol is treated as a surrogate marker for cardiovascular disease (CVD). In contrast, sustained elevations in blood glucose and insulin typically precede, and contribute to, rising triglyceride concentrations, increasing risk for both CVD and diabetes.[67]

A substantial and growing body of research indicates that glycaemic instability, insulin resistance, compensatory hyperinsulinemia, and associated low-grade inflammation underpin much of the contemporary burden of chronic disease. Together, these processes drive progressive metabolic dysfunction and neurodegeneration, limiting the ability of individuals and families to achieve and sustain optimal health. Pathophysiological injury begins at the mitochondrial level, where impaired mitochondrial function disrupts cellular energy production, increases oxidative stress, and propagates metabolic dysfunction across insulin-sensitive tissues.[68]

Genome-wide association studies have indicated that more than 65 genes are associated with an elevated risk of T2DM. These genes are involved in regulating the metabolic pathways of glucose homeostasis, insulin signalling, and sensitivity.[69] Generic dietary recommendations may be unsuitable for people with these genes, and such groups may benefit from dietary approaches that shield them from over-consumption of refined carbohydrates.

Some people (for example many South Asian populations) develop type 2 diabetes mellitus (T2DM) at a lower body mass index (BMI) and at lower levels of apparent adiposity than other groups.[70] People differ (including by ancestry) in how much subcutaneous fat they can safely store before 'spillover' to liver, pancreas, and muscle (ectopic fat) drives insulin resistance.[71] [72] [73]

When modern diets high in refined carbohydrates intersect with these biological differences, the tipping point into metabolic disease can be reached more rapidly. Health scholars have proposed that cutoff values for BMI may need to be revised to due to the risk that the current recommendations for obesity under-recognise the risk of developing T2DM in minority ethnic populations.[74]

Complementary interventions alongside dietary changes may further support the lowering of blood glucose. Dr Alpana Shukla has led trials on meal sequencing (also called food order, carbohydrate-last

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[65] Slyper AH. (1994) Low-Density Lipoprotein Density and Atherosclerosis: Unraveling the Connection. *JAMA*. 1994;272(4):305–308. DOI:10.1001/jama.1994.03520040067042

[66] Endo K, Tanaka M, Sato T, *et al.* (2025). High Level of Estimated Small Dense Low-Density Lipoprotein Cholesterol as an Independent Risk Factor for the Development of Ischemic Heart Disease Regardless of Low-Density Lipoprotein Cholesterol Level - A 10-Year Cohort Study. *Circ J.* 89(8):1182-1189. DOI: 10.1253/circj.CJ-24-0770.

[67] Demasi M, Lustig RH, Malhotra A. (2017). The cholesterol and calorie hypotheses are both dead — it is time to focus on the real culprit: insulin resistance. *The Pharmaceutical Journal*. 14 July, 2017.

[68] Chen Y, Liu X, Liu Y. *et al*. (2025) Mitochondrial quality control in diabetes mellitus and complications: molecular mechanisms and therapeutic strategies. *Cell Death Dis* 16: 652. DOI:10.1038/s41419-025-07936-y

[69] Valaiyapathi B, Gower B, Ashraf AP. (2020) Pathophysiology of Type 2 Diabetes in Children and Adolescents. *Curr Diabetes Rev*; 16: 220-229 DOI: 10.2174/1573399814666180608074510.

[70] Mohan V. (2024). Lessons Learned From Epidemiology of Type 2 Diabetes in South Asians: Kelly West Award Lecture 2024. *Diabetes Care.* 48 (2): 153–163. DOI: 10.2337/dci24-0046

[71] Vishvanath L and Gupta RK. (2019). Contribution of adipogenesis to healthy adipose tissue expansion in obesity. *J Clin. Invest.* 129(10):4022-4031. DOI: 10.1172/JCI129191

[72] Wernstedt Asterholm I, Tao C, Morley TS *et al*. (2014). Adipocyte Inflammation Is Essential for Healthy Adipose Tissue Expansion and Remodeling. *Cell Metabolism*, 20(1):103 – 118. DOI: 10.1016/j.cmet.2014.05.005

[73] Taylor R (2024). Understanding the cause of type 2 diabetes. *The Lancet Diabetes & Endocrinology,* 12(9):664 – 673, DOI: 10.1016/S2213-8587(24)00157-8

[74] Caleyachetty R, Barber TM, Mohammed NI, *et al.* (2021). Ethnicity-specific BMI cutoffs for obesity based on type 2 diabetes risk in England: a population-based cohort study. *Lancet Diabetes Endocrinol.* 9(7):419-426. DOI: 10.1016/S2213-8587(21)00088-7.

eating, or nutrient pre-loading) to control post-prandial blood glucose and insulin responses.[75] Time restricted eating (or fasting) in conjunction with a low carbohydrate diet is increasingly supported by studies[76] [77] [78], however, consideration needs to be given to the challenges inherent in transitioning to a fasted state and the intersecting challenge of food addiction (discussed in later chapters).

These processes also exert a distinct intergenerational impact. Infants born to mothers with T2DM or elevated insulin levels during pregnancy are more likely to be large for gestational age and to have increased adiposity at birth. Excess maternal glucose readily crosses the placenta, stimulating the foetal pancreas to increase insulin secretion. In the foetus, insulin functions as a potent growth factor, promoting accelerated growth and increased fat deposition. Although maternal insulin itself does not cross the placenta, maternal insulin resistance enhances placental transfer of glucose and lipids, indirectly driving foetal hyperinsulinaemia and adiposity, effectively acting as a metabolic amplifier across generations.[79] [80] [81]

An increasing range of studies, from mechanistic and biomarker studies to case studies and trials provide firm scientific evidence for carbohydrates as a primary driver of obesity and metabolic disease via this *insulin pathway*, rather than the historic consensus position that calorific consumption is the primary driver of obesity.[82] [83] A simple focus on calorie restriction may not be the most effective approach for reducing risk parameters when the carbohydrate-insulin pathway is taken into consideration.[84] Relatedly, most calorie-related research and policy does not address craving and food-addiction-related issues and the role of satiety and the regulation of appetite, when addressing dietary behaviour and health.

Insulin resistance is not driven by excess carbohydrate intake alone. Factors such as chronic stress, elevated cortisol and epinephrine, inadequate or disrupted sleep, exposure to environmental chemicals, and certain medications can contribute to the development of insulin resistance.

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Metabolic Syndrome & Type Two Diabetes Mellitus (T2DM).

A growing scientific literature consistently associates insulin resistance and hyperinsulinaemia with a range of conditions that are increasingly prevalent in modern societies, including type 2 diabetes mellitus, cardiovascular disease, cellular senescence and cancer[85], and neurodegenerative diseases. [86 87 88 89]

Metabolic syndrome describes a cluster of interrelated conditions: including hypertension, dyslipidaemia, obesity, type 2 diabetes mellitus, and chronic inflammation, that share common underlying mechanisms. The carbohydrate–insulin pathway represents a central mechanistic axis influencing the development and progression of metabolic syndrome.

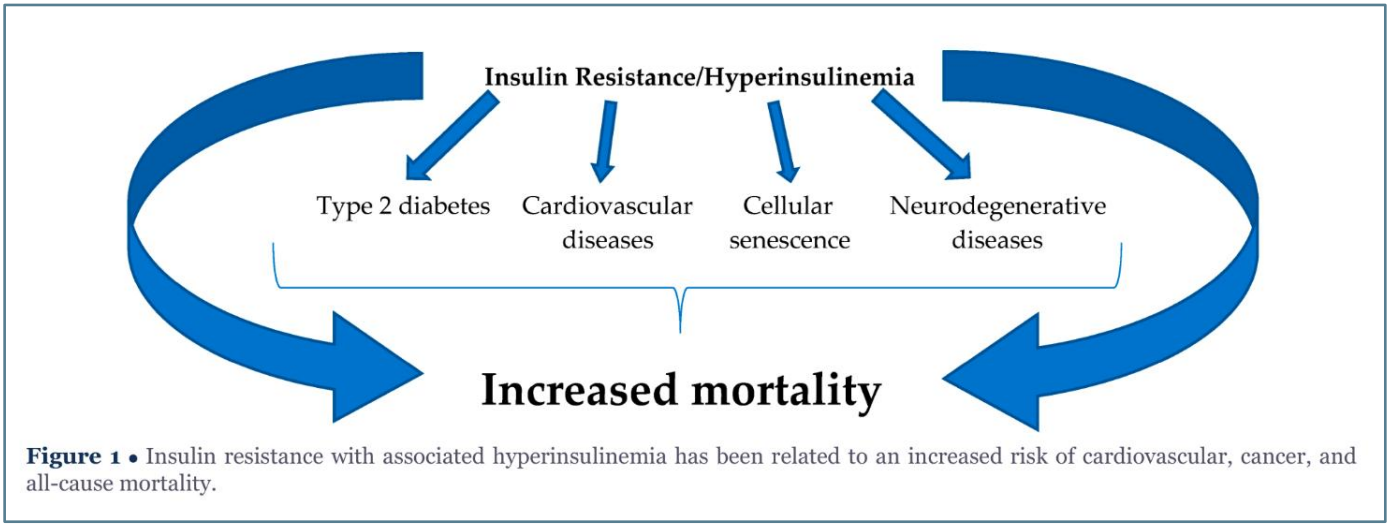

Figure 1 • Insulin resistance with associated hyperinsulinemia has been related to an increased risk of cardiovascular, cancer, and all-cause mortality.

Figure 2. Fazio S, Fazio V, Affuso F. The link between insulin resistance, hyperinsulinemia and increased mortality risk. *Academia*.

Hyperinsulinaemia, insulin resistance, and impaired metabolic function often precede a broad spectrum of inflammatory, metabolic, and brain-related conditions, contributing to microvascular dysfunction and increasing the risk of cardiovascular disease[90], cancer, visual impairment[91 92 93], neurodegeneration[94], and

[85] Shen S and Iyengar NM. (2022). Insulin-Lowering Diets in Metastatic Cancer. *Nutrients*,14:3542. DOI:10.3390/nu14173542.
[86] Flatt JP. (1996). Glycogen levels and obesity. *Int J Obes Relat Metab Disord.* Mar;20 Suppl 2:S1-11. PMID: 8646265.
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[89] Shen S and Iyengar NM. (2022). Insulin-Lowering Diets in Metastatic Cancer. *Nutrients*,14:3542. DOI:10.3390/nu14173542
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[94] Gomes Gonçalves N, Vidal Ferreira N, Khandpur N, *et al.* (2023). Association Between Consumption of Ultraprocessed Foods and Cognitive Decline. *JAMA Neurol.* 80(2):142–150. DOI:10.1001/jamaneurol.2022.4397

premature mortality.[95] [96] [97] Clinical features commonly associated with insulin resistance include acanthosis nigricans, metabolic-associated fatty liver disease (MAFLD), hyperandrogenism in females, and polycystic ovary syndrome (PCOS).[98]

People consuming diets high in sugars, refined carbohydrates, and ultra-processed foods are at increased risk of developing overlapping features of metabolic syndrome alongside a range of brain-related conditions.[99] [100] [101] An appreciation of the carbohydrate–insulin pathway helps explain why carbohydrate quality, quantity, and timing are critical not only for glycaemic control, but also for lipid regulation, adiposity, and overall metabolic health. [102] [103] [104]

T2DM is diagnosed when HbA1c is ≥ 50 mmol/L or fasting glucose is ≥ 7 mmol/L in repeated tests. Prediabetes is diagnosed when HbA1c is between 41 – 49 mmol/mol or fasting glucose 6.1 – 6.9 mmol/L or 2-hour glucose on GTT 7.8 – 11 mmol/L. [105] People with type 1 diabetes mellitus (T1DM) must contend with a pancreas that does not work, i.e. secrete insulin.

T2DM constitutes up to 96% of diabetes cases globally. A 2021 *The Lancet* analysis reported that more than 90% of the age-standardised diabetes prevalence rate across major regions was due to type 2 diabetes.[106]

T2DM is increasing in prevalence. Current New Zealand data on diabetes is based on estimates held with the Virtual Diabetes Register (VDR). In 2024, about 348,500 people were estimated to have diabetes in Aotearoa New Zealand. The estimated age-standardised prevalence of diabetes has increased from 36.6 (in 2013) to 47.0 per 1000 population, with the highest prevalence in Pasifika (137.2 per 1000 Pasifika population), Indian (103.6 per 1000 Indian population) and Māori (82.4 per 1000 Māori population) communities.[107] The register does not discern between T1DM and T2DM, nor does it disclose shifts in prevalence by age group over time.

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[95] Lane MM, Gamage E, Du S, Ashtree DN, *et al*. (2024). Ultra-processed food exposure and adverse health outcomes: umbrella review of epidemiological meta-analyses *BMJ* 384 :e077310 DOI:10.1136/bmj-2023-077310

[96] Ede G (2024). Change Your Diet, Change Your Mind: A Powerful Plan to Improve Mood, Overcome Anxiety, and Protect Memory for a Lifetime of Optimal Mental Health. *Dimensions*.

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[98] Pappachan JM, Fernandez CJ, Ashraf AP. (2024) Rising tide: The global surge of type 2 diabetes in children and adolescents demands action now. *World J Diabetes* 15(5):797-809. DOI: 10.4239/wjd.v15.i5.797.

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[100] Lane M M *et al*. (2024). Ultra-processed food exposure and adverse health outcomes: umbrella review.

[101] Hall, KD, Ayuketah A, Brychta R, et al. (2019). Ultra-Processed Diets Cause Excess Calorie Intake and Weight Gain: An Inpatient Randomized Controlled Trial of Ad Libitum Food Intake. Cell Metabolism, Volume 30, Issue 1, 67 - 77.e3

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[103] DiNicolantonio JD, OKeefe JH (2017) Added sugars drive coronary heart disease via insulin resistance and hyperinsulinaemia: a new paradigm: *Open Heart* 4:e000729. DOI: 10.1136/openhrt-2017-000729

[104] Reaven G (1988). Role of Insulin Resistance in Human Disease. *Diabetes* 37 (12): 1595-1607. DOI:10.2337/diab.37.12.1595

[105] Diabetes New Zealand. What tests can be done to find out if I have diabetes? https://www.diabetes.org.nz/diabetes-diagnosis

[106] Ong, Kanyin Liane *et al.*(2021). Global, regional, and national burden of diabetes from 1990 to 2021, with projections of prevalence to 2050: a systematic analysis for the Global Burden of Disease Study 2021 *The Lancet,* 402(10397);203 -234.

[107] Health New Zealand. Virtual Diabetes Register and web tool. https://www.tewhatuora.govt.nz/for-health-professionals/data-and-statistics/diabetes/virtual-diabetes-register-web-tool#key-findings-from-the-2024-virtual-diabetes-register

It is that thought that the prevalence of diabetes has been increasing by 7% per year.[108] A 2025 paper calculated that:

> *Aotearoa New Zealand will experience a significant increase in the absolute volume of prevalent diabetes, rising by nearly 90% to more than 500,000 by 2044. The age-standardised prevalence of diabetes will increase from around 3.9% of the population (268,248) to 5.0% overall (502,358). The prevalence and volume of diabetes diagnoses will increase most drastically for Pacific peoples, most notably Pacific females for whom diabetes prevalence is projected to increase to 17% of the population by 2044.*[109]

The current annual cost of T2DM in New Zealand is estimated to be $2.1 billion. PWC calculated that the annual cost would increase by 63% to $3.5 billion in the next 20 years.[110] PWC drew attention to the additive costs of a diagnosis in youth:

> *the personal and economic impact of the disease is most detrimental when a person is diagnosed early in life. When comparing the lifetime cost of someone diagnosed with type 2 diabetes at age 25 years ($565k) to the lifetime cost of someone diagnosed at age 75 years ($44k), the cost differential is $521k or a factor of 13. This is significant given the shift towards younger cohorts of New Zealanders developing type 2 diabetes.* [111]

Coronary Artery / Heart Disease Risk.

Cardiovascular risk is only one aspect of the broader metabolic risk landscape. Coronary artery disease (a form of cardiovascular disease) occurs when these processes take place within the coronary arteries supplying the heart.

A diagnosis of T2DM represents the tip of a 'risk iceberg', signalling a spectrum of disease risks that stem from chronically elevated HbA1c and triglyceride levels, including vascular, renal, hepatic, and neurological complications. An increasing range of studies consistently demonstrate that carbohydrate restrictive diets improve cardiovascular health, reducing triglyceride levels, blood pressure and other inflammatory markers.[112]

A high triglyceride-to-high-density lipoprotein ratio predicts cardiovascular risk and is a recognised surrogate marker of insulin resistance. This ratio often improves rapidly with carbohydrate reduction. By contrast, low-density lipoprotein cholesterol (LDL-C), the cholesterol content within LDL particles, does not reliably reflect cardiometabolic risk in isolation and may poorly predict cardiovascular disease in the presence of insulin resistance and metabolic dysfunction. Individuals with identical LDL-C values can have markedly different particle numbers and risk profiles, which helps explain why LDL-C often tracks poorly with outcomes when insulin resistance and inflammation are present.

Cardiac problems arise when triglycerides remain chronically elevated due to excessive production (often from high carbohydrate diets, insulin resistance, or liver overactivity) or impaired clearance (as in metabolic syndrome or genetic lipid disorders). Persistently high triglycerides mean a constant presence of

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[110] PwC New Zealand (2021). The Economic and Social Cost of Type 2 Diabetes. https://healthierlives.co.nz/wp-content/uploads/Economic-and-Social-Cost-of-Type-2-Diabetes-FINAL-REPORT_Secure-5.pdf Accessed 19/11/25.

[111] PwC New Zealand (2021). The Economic and Social Cost of Type 2 Diabetes

[112] Feng S, Liu R, Thompson C *et al.* (2025). An increasing range of studies consistently reveal that carbohydrate restrictive diets improve cardiovascular health, reducing triglyceride levels, blood pressure and other inflammatory markers. *The American Journal of Clinical Nutrition,* 122(5)1461-1478. DOI: 10.1016/j.ajcnut.2025.09.012

triglyceride-rich lipoproteins and their remnants in circulation. These remnants are particularly dangerous because they can penetrate the arterial wall, where they contribute to the build-up of atherosclerotic plaque.

The heart disease risk grows as fatty deposits in the blood (atheroma) attach to artery walls which over time, become hardened and stiff. In the arterial wall, remnants are taken up by macrophages, forming lipid-laden 'foam cells'. Over time, this accumulation of fat, cholesterol, and inflammatory cells creates plaques that narrow and stiffen arteries, a process called atherosclerosis. Triglyceride-rich particles also promote low-grade inflammation, oxidative stress, and endothelial dysfunction (damage to the artery's inner lining), all of which accelerate plaque growth and instability.

Unstable atherosclerotic plaques can rupture, exposing their lipid-rich contents to the bloodstream and triggering clot formation (thrombosis). If such a clot develops in a coronary artery, it can abruptly block blood flow to part of the heart muscle, resulting in a myocardial infarction (heart attack).

Elevated triglycerides are not merely markers of metabolic imbalance; they actively contribute to the pathological cascade underlying coronary artery disease. Triglycerides and blood pressure rise in parallel because they share upstream metabolic drivers, particularly insulin resistance, endothelial dysfunction, and vascular inflammation. Managing triglycerides is therefore a central component of cardiovascular risk reduction, alongside controlling blood pressure, blood glucose, and insulin instability.

Saturated Fats and Cholesterol – guilty by association?

Cholesterol has been used as a surrogate target for the prevention of heart disease. The heart-disease hypothesis postulates that reducing dietary saturated fat lowers serum cholesterol, thereby reducing cardiovascular risk.[113] Keeping LDL-C low by restricting saturated fat was an underpinning rationale for low-fat and low-saturated-fat diets. Carbohydrate-restricted diets may be associated with higher cholesterol levels but typically occur in a context of otherwise low metabolic risk.[114] [115]

New Zealand's dietary guidelines directly influence the scope and framing of the questions officials include in national dietary surveys. A New Zealand Ministry of Health report, *Adults' Dietary Habits* (2022), illustrates the Ministry's disproportionate emphasis on dietary fat reduction, alongside a relative lack of attention to adequate protein intake and an absence of consideration of cumulative carbohydrate intake.

The report refers to 'fat' fifty-seven times predominantly in the context of promoting low-fat alternatives, while 'protein' is mentioned only three times, each instance limited to noting that nuts, seeds, and legumes are protein sources, with no discussion of animal-derived protein. The carbohydrate macronutrient class is not discussed at all. In addition, no differentiation is made between vegetable classes (for example, starchy vegetables such as potatoes versus leafy green vegetables such as silverbeet), and total daily protein intake is not estimated or reported.[116]

Insulin is a tiny but highly potent molecule, while cholesterol is a much larger molecule. Gary Taubes meticulous research has shed light on why and how the more easily detectable cholesterol created the heart disease risk, while the invisible insulin was neglected.[117] Contemporary beliefs that cholesterol is the driving factor for heart disease risk, rather than carbohydrate-mediated lipidosis (high triglyceride levels),

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[113] DuBroff R, de Lorgeril M. (2021). Fat or fiction: the diet- heart hypothesis. *BMJ Evid Based Med* 26:3–7

[114] Norwitz NG, Feldman D, Soto-Mota A, *et al.* (2021). Elevated LDL Cholesterol with a Carbohydrate-Restricted Diet: Evidence for a Lean Mass Hyper-Responder Phenotype. *Curr Dev Nutr*. 30;6(1):nzab144. DOI: 10.1093/cdn/nzab144.

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[116] Ministry of Health (2022). Adults' Dietary Habits – Findings from the 2018/19 and 2019/20 New Zealand Health Survey. Wellington: Ministry of Health. ISBN 978-1-99-110065-8

[117] Taubes, G. (2024). Rethinking Diabetes. *Allen and Unwin.* Chapter 7.

may be a consequence of the limitation of early testing technologies and later studies which may have exaggerated the relationship of high cholesterol with mortality risk. [118]

This was the origin of the belief that high saturated-fat diets contribute to heart disease risk. It wasn't until after 1960 that researchers would appreciate how insulin levels in individuals with T2DM would spike far higher than that of healthy populations after consuming carbohydrates.[119]

Reviews consistently highlight the lack of robust evidence supporting an association between saturated fat intake and adverse cardiovascular outcomes, emphasising inconsistent findings and the context-dependent nature of reported effects.[120] [121] [122] [123]

In 2014 a U.K. based group assessed the relative risk of consumption of saturated, monosaturated, and polyunsaturated fats. The group concluded:

> *Current evidence does not clearly support cardiovascular guidelines that encourage high consumption of polyunsaturated fatty acids and low consumption of total saturated fats*.[124]

In A 2015 review identified that saturated fats were not associated with all-cause mortality, CVD, CHD, ischemic stroke, or T2DM, while finding that trans fats were associated with all-cause mortality, total CHD, and CHD mortality.[125] A later Cochrane review (2015, updated 2020) determined that reducing saturated fat intake for at least two years causes a potentially important reduction in combined cardiovascular events.[126] New Zealand researchers evaluated that review, finding that the relative risk became non-significant when using more robust assumptions. [127]

Two 2025 reviews have further confirmed the absence of convincing evidence implicating saturated fat in cardiovascular disease. Steen *et al.* found only low to moderate certainty, i.e. no significant evidence that that reducing saturated fat intake might reduce risk for at-risk groups. For people with low baseline risk, absolute reductions were below the threshold of importance.[128] Yamada *et al.* concluded that:

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[118] Ramsden CE, Zamora D, Majchrzak-Hong S, *et al.* (2016). Re-evaluation of the traditional diet-heart hypothesis: analysis of recovered data from Minnesota coronary experiment (1968-73). *BMJ* 2016;353:i1246. DOI:10.1136/bmj.i1246.

[119] Taubes, G. (2024). Rethinking Diabetes. *Allen and Unwin.* Chapter 7.

[120] Volek JS, Phinney SD, Forsythe CE, *et al.* (2009). Carbohydrate restriction has a more favorable impact on the metabolic syndrome than a low fat diet. *Lipids.* 44(4):297-309. DOI: 10.1007/s11745-008-3274-2

[121] Harcombe Z. (2017). Dietary fat guidelines have no evidence base: where next for public health nutritional advice? Br *J Sports Med* 51:769–774. DOI:10.1136/bjsports-2016-096734

[122] Aramburu A, Dolores-Maldonado G, Curi-Quinto K *et al.,* (2024). Effect of reducing saturated fat intake on cardiovascular disease in adults: an umbrella review. *Front. Public Health.* 12:2024. DOI: 10.3389/fpubh.2024.1396576

[123] Dehghan M, Diaz R, Mente A. *et al*. (2025). Associations of fats and carbohydrate intake with cardiovascular disease and mortality in 18 countries from five continents (PURE): a prospective cohort study. *The Lancet*, 390:10107;2050 – 2062.

[124] Chowdhury R, Warnakula S, Kunutsor S, et al. (2014). Association of dietary, circulating, and supplement fatty acids with coronary risk: a systematic review and meta-analysis. *Ann Intern Med,* 160:398–406. DOI:10.7326/M13-1788

[125] de Souza RJ, Mente A, Maroleanu A, (2015). Intake of saturated and trans unsaturated fatty acids and risk of all cause mortality, cardiovascular disease, and type 2 diabetes: systematic review and meta-analysis of observational studies. *BMJ*. 2015 Aug 11(351):h3978. DOI: 10.1136/bmj.h3978.

[126] Hooper L, Martin N, Jimoh OF, *et al.* (2020). Reduction in saturated fat intake for cardiovascular disease. *Cochrane Database Syst Rev.* 8(8):CD011737. DOI: 10.1002/14651858.CD011737.pub3.

[127] Thornley S, Schofield G, Zinn C, Henderson G. (2019). How reliable is the statistical evidence for limiting saturated fat intake? A fresh look at the influential Hooper meta-analysis. *Internal J Med.* 49(11)1418-1424. DOI: 10.1111/imj.14325

[128] Steen JP, Klatt KC, Chang Y, *et al.* (2025). Effect of Interventions Aimed at Reducing or Modifying Saturated Fat Intake on Cholesterol, Mortality, and Major Cardiovascular Events: A Risk Stratified Systematic Review of Randomized Trials. *Ann Intern Med.* DOI:10.7326/ANNALS-25-02229

The findings indicate that a reduction in saturated fats cannot be recommended at present to prevent cardiovascular diseases and mortality. [129]

Studies may fail to adequately control for dietary fat quality and carbohydrate burdens, which may act as significant confounders of outcomes.[130] The health effects of fats depend not just on their type (saturated, monounsaturated, polyunsaturated), but on the extent of processing and refining. Unprocessed or minimally processed fats, such as those found in extra virgin olive oil, nuts, seeds, avocados, and oily fish, consistently show protective effects for heart and metabolic health. Evidence also suggests that replacing refined carbohydrates with unprocessed fats yields better outcomes than simply reducing fat intake.

The example of eggs illustrates how public health messaging can become misdirected. From the 1970s through the early 1990s, dietary cholesterol was widely portrayed as a driver of elevated blood cholesterol, and eggs were framed as directly harmful to heart health. These restrictions peaked in the 1980s and early 1990s, before being revised as evidence showed that dietary cholesterol has only a modest and variable effect on serum cholesterol and cardiovascular risk; however, the associated cultural risk perception has persisted. More recent evidence increasingly highlights the nutritional value of eggs, with a large Australian cohort study of older adults finding that frequent egg consumption was associated with a lower risk of cardiovascular disease and all-cause mortality.[131]

Cholesterol is a sterol (lipid) that plays metabolic key roles, including cell signalling and hormone synthesis. Cholesterol is present in all vertebrates, while most invertebrates (including insects, molluscs and crustaceans) do not synthesize cholesterol but source it from their diet.[132] [133] This fundamental role of cholesterol across all systems suggests that early diagnostic testing for plaque in sclerotic arteries would naturally, consistently detect elevated cholesterol levels. This did not mean that cholesterol was the driver of heart disease.

Cholesterol is synthesized from acetyl-CoA in a multi-step pathway and cholesterol is most concentrated in organs involved in membrane biogenesis, hormone synthesis, cellular signalling and lipoprotein assembly. Lipoproteins are the shipping containers that carry cholesterol to where it is required.[134] The membrane of every cell requires cholesterol to make and maintain them. The liver controls homeostasis,

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[129] Yamada S, Shirai T, Inaba S, *et al*. (2025). Saturated Fat Restriction for Cardiovascular Disease Prevention: A Systematic Review and Meta-analysis of Randomized Controlled Trials. *JMA J.* 8(2):395-407. DOI: 10.31662/jmaj.2024-0324.

[130] Arnesen EK, Laake I, Veierød MB, Retterstøl K. (2024) Saturated fatty acids and total and CVD mortality in Norway: a prospective cohort study with up to 45 years of follow-up. *Br J Nutr.*132(4):1-13. DOI: 10.1017/S0007114524001351.

[131] Wild H, Gasevic D, Woods RL, *et al*. (2025). Egg Consumption and Mortality: A Prospective Cohort Study of Australian Community-Dwelling Older Adults. *Nutrients*. 17(2):323. DOI:10.3390/nu17020323

[132] Behmer, S.T., & Nes, W.D. (2003). Insect sterol nutrition and physiology: a global overview. *Advances in Insect Physiology,* 31:1–72. DOI: 10.1016/s0065-2806(03)31001-x

[133] Goad, L.J. (1981). Sterol biosynthesis and metabolism in marine invertebrates. *Pure and Applied Chemistry*, 53(4):837–852. DOI: 10.1351/pac198153040837

[134] Lund-Katz S and Phillips MC (1986). Packing of cholesterol molecules in human low-density lipoprotein *Biochemistry* 1986 25 (7):1562-1568. DOI: 10.1021/bi00355a016

and the main sites are in the liver, intestine and brain but the adrenal glands, gonads, skin, kidney, lungs, spleen and muscle can also synthesise cholesterol.[135] [136] [137] [138] [139]

Cholesterol's fundamental role in the brain is well established. Cholesterol is the major building block of myelin sheaths and cholesterol is essential to maintain its compact structure. Cholesterol is crucial for synapse formation and function, influencing neurotransmitter release and receptor organisation, and hence plays a key role in plays a central role in early brain development.[140] [141] [142]

Higher HDL-cholesterol is associated with greater longevity.[143] Low low-density lipoprotein cholesterol (for example below 70 mg/dL) may be associated with health risks including mortality while high levels (such as over 200 mg/dL) may be health promoting.[144] [145]

Statins are HMG-COA reductive inhibitors and are taken to reduce cholesterol levels, with the impression that this will reduce cardiovascular-related mortality. However, primary and secondary prevention trials suggest that the median postponement of death for may be only 3.2 and 4.1 days, respectively.[146] Statins may be associated with reduced cholesterol levels that are detrimental to brain health. Trials that failed to demonstrate that by lowering cholesterol coronary heart disease would be prevented, may have been downplayed,[147] excluded and suppressed.[148] Animal studies have revealed that statins can lower cholesterol levels in the brain.[149] [150] [151]

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[135] Bloch K. (1992). Sterol structure and membrane function. *PNAS*, 89(15):6942–6945. DOI: 10.1073/pnas.89.15.69
[136] Brown MS, & Goldstein JL. (1974). Familial hypercholesterolemia: Defective binding of lipoproteins to cultured fibroblasts associated with impaired regulation of 3-hydroxy-3-methylglutaryl coenzyme a reductase activity, Proc. Natl. Acad. Sci. U.S.A. 71(3)788-792, DOI:10.1073/pnas.71.3.788
[137] Simons K and Ikonen E (2000). How Cells Handle Cholesterol. *Science,* 290(5497), 1721–1726.DOI: 10.1126/science.290.5497.1721
[138] Dietschy JM & Turley SD (2002). Control of cholesterol turnover in the mouse: a model for human lipoprotein metabolism. *Biochimica et Biophysica Acta,* 1529(1–3):171–187. DOI: 10.1016/S1388-1981(01)00160-8
[139] Belabed, M., Park, M.D., Blouin, C.M. et al. (2025). Cholesterol mobilization regulates dendritic cell maturation and the immunogenic response to cancer. *Nat Immunol* 26:188–199. DOI:10.1038/s41590-024-02065-8
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[142] Björkhem I & Meaney S (2004). Brain cholesterol: long secret life behind a barrier. *Arteriosclerosis, Thrombosis, and Vascular Biology,* 24(5):806–815. DOI: 10.1161/01.ATV.0000120374.59826.1b
[143] Ravnskov U, de Lorgeril M, Diamond DM, *et al*. (2020). The LDL paradox: Higher LDL Cholesterol is Associated with Greater Longevity. *A Epidemiol Public Health,* 3(1): 1040.
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The Inflammatory Cascade that Drives Multimorbidity, & High Sensitivity C-reactive Protein.

Inflammation can be provoked by an acute injury, or accrue slowly over time from toxic exposures which, if they occurred rarely, would not build up to inflammation at the system level, whether an organ, tissue or a whole-body response.

Persistent high refined carbohydrate exposures drive inflammation in the body. As the cascade diagram below shows, it does not happen all at once but builds over time. The cascade occurs from a convergence of hyperinsulinemia, glycaemic volatility, ectopic fat accumulation, adipocyte stress and macrophage infiltration. [152] [153] [154]

High blood glucose (hyperglycaemia) is toxic to cells and to the mitochondria. T2DM is recognised as an inflammatory disease and glycation plays an important role. The frequent intake of sugary or starchy foods over time results in high glucose levels, which then stick to proteins in the blood and tissues in a process called glycation. This is somewhat like a slow biological version of caramelisation, where sugars react with proteins without enzymes. These altered proteins, known as advanced glycation end products (AGEs), can build up in tissues and bind to special receptors on cells called RAGE (Receptors for Advanced Glycation End Products).

Once activated, these receptors trigger a chain reaction: the release of oxidative molecules, the activation of inflammatory cytokines, and the recruitment of immune cells to the site.

Health authorities that urge populations to shift away from saturated fats have often failed to adequately assess the inflammatory potential of processed fats. Processed fats including industrial trans fats, hydrogenated oils, and refined seed oils commonly used in ultra-processed foods, are consistently associated with increased risk of cardiovascular disease, metabolic syndrome, and chronic inflammation. These fats frequently undergo chemical processing or high-heat treatment, generating harmful by-products that can disrupt lipid metabolism and impair endothelial function.

Context is important: saturated fats derived from heavily processed meats and packaged foods may confer greater risk than saturated fats from whole-food sources such as dairy and fresh meat. The role of high-quality, minimally processed fat sources in supporting metabolic health has been under-recognised in nutrition policy.[155] [156]

Low-grade systemic inflammation is increasingly recognised as a clinically relevant contributor to cardiovascular risk. High-sensitivity C-reactive protein (hsCRP) is a well-validated acute-phase inflammatory biomarker, synthesised by the liver in response to pro-inflammatory cytokines (notably interleukin-6). While hsCRP rises rapidly following acute infection or trauma, persistent elevation is thought to reflect ongoing low-grade inflammation. Elevated CRP concentrations are consistently associated with an increased risk of coronary heart disease and broader cardiovascular events.

Dietary patterns influence inflammatory status. Diets characterised by a high carbohydrate burden and a high intake of ultra-processed foods are associated with higher CRP concentrations, whereas low-carbohydrate dietary patterns are generally associated with reductions in CRP and other inflammatory

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[156] Liu AG, Ford NA, Hu FB. *et al.* (2017) A healthy approach to dietary fats: understanding the science and taking action to reduce consumer confusion. *Nutr J* 16, 53. DOI:10.1186/s12937-017-0271-4

markers.[157] [158] [159] [160] [161] In 2009, researchers developed and validated the Dietary Inflammatory Index (DII), demonstrating that shifts toward a more anti-inflammatory dietary pattern were associated with significant reductions in hs-CRP.[162]

In September 2025, the American College of Cardiology updated their guidance, with new ACC recommendations including universal screening of C-reactive protein levels in all patients.:

> *Measurement of hsCRP (>3 mg/L) can be used in routine clinical practice to identify primary prevention individuals at increased inflammatory risk as long as the patient is not acutely ill.*[163]

Inflammation, as assessed by hsCRP, may not only be a more accurate predictor of risk for future cardiovascular events and death than hyperlipidaemia assessed by low-density lipoprotein cholesterol (LDLC), it may highlight risk in populations currently overlooked by standard screening approaches. Contemporary preventive cardiology frameworks, focussed on hypertension, dyslipidaemia, diabetes mellitus, and smoking, do not routinely account of the inflammatory status of the individual.[164]

Evidence from large population studies supports this position. A United Kingdom study, the largest analysis to date, involving 448,653 UK Biobank participants without known atherosclerotic cardiovascular disease, found that:

> *hsCRP independently enhances CV risk stratification, and the predictive performance of hsCRP was comparable to or greater than traditional risk factors such as systolic blood pressure or LDL-C.*
>
> *…individuals with hsCRP levels >3 mg/L had a 34% higher risk of MACE, a 61% and 54% increased risk of CV death and all-cause death compared to those with hsCRP <1 mg/L.*

Notably, '*the association of hsCRP with all endpoints was consistent across subgroups.*'[165] A separate U.K. cohort study investigating the usefulness of baseline serum hsCRP as a predictor of long-term cardiovascular events in stable patients with hypertension, found that participants in the top third of hsCRP

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[165] Kurt B, Reugels M, Schneider KM, *et al.* (2025). C-reactive protein and cardiovascular risk in the general population, *European Heart Journal,* 2025;ehaf937, DOI:10.1093/eurheartj/ehaf937

experienced a substantially greater incidence of cardiovascular events and all-cause mortality, compared to the lowest third.[166]

Comparable findings have been reported in the U.S. In a prospective cohort study of 12,530 initially healthy women followed for 30 years, women with persistently elevated hsCRP concentrations had a significantly higher risk of future cardiovascular events, independent of traditional risk factors.[167]

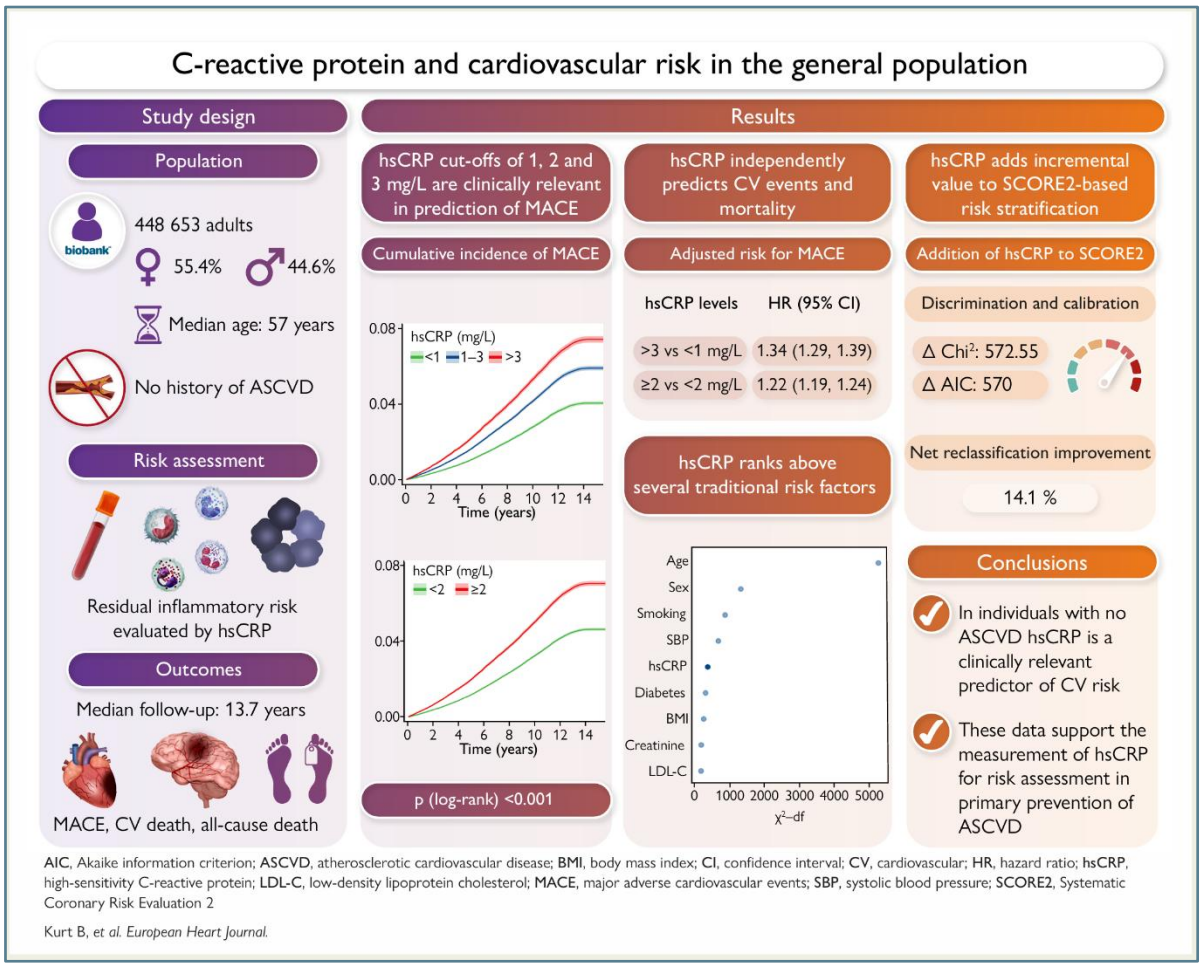

Figure 3. Kurt B, Reugels M, Schneider KM, et al. (2025). C-reactive protein and cardiovascular risk in the general population. *European Heart Journal*.

In New Zealand, high-sensitivity CRP (hsCRP) is available, however, it is used more selectively, predominantly for cardiovascular risk stratification, and is not universally ordered. It may be requested by GPs or specialists, but it is not part of routine population screening in NZ.

[166] Hartley A, Rostamian S, Kaura A *et al.* (2025). The relationship of baseline high-sensitivity C-reactive protein with incident cardiovascular events and all-cause mortality over 20 years. *eBioMedicine* 2025;117:105786. DOI: 10.1016/j.ebiom.2025.105786

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HYPERINSULINAEMIA & INFLAMMATORY CASCADE – can precede obesity.

1. **1. High refined carbohydrate intake.**

- Frequent intake of high-glycaemic carbs leads to large postprandial glucose spikes.
- Pancreas releases *large insulin pulses* to bring glucose down.
- *No inflammation yet.* Sets the hormonal scene (high insulin → low glucagon → low fat oxidation).
- Sensitive individuals may show transient oxidative stress and low-grade inflammatory markers.

2. **2. Chronically elevated insulin levels (hyperinsulinemia).**

- Even in normoglycaemia, insulin may remain elevated for hours.
- Over time, tissues become less responsive → early insulin resistance.
- *Early signs of inflammation begin here:* Chronic hyperinsulinemia suppresses autophagy (mTOR) and, together with hyperglycaemia/FFAs, increases ROS, altering immune cell signalling.
- Acceleration of non-enzymatic glycation of circulating proteins.
- Endothelial dysfunction and low-grade inflammation (e.g., IL-6, CRP) can precede fat gain.

3. **3. Insulin promotes fat storage (visceral adiposity).**

- Inhibits lipolysis (fat breakdown) and stimulates lipogenesis (fat creation) in adipose tissue, especially visceral fat. Preferential deposition impacted by genetics, gender, stress etc.
- *Inflammation ramps up here.* Repeated postprandial hyperglycaemia accelerates AGE formation; glycated proteins activate RAGE receptors, triggering NF-κB and downstream inflammatory cytokine release, which exacerbates tissue hypoxia.
- Immune cells (esp. macrophages) infiltrate visceral fat. Adipose remains endocrine but becomes increasingly pro-inflammatory (from macrophage infiltration, cytokine.).
- Macrophage infiltration reinforces adipose inflammation, amplifying insulin resistance.

4. **4. Weight gain and metabolic inflexibility**

- Persistent hyperinsulinemia shifts caloric partitioning toward fat storage.
- Appetite regulation can be disrupted via leptin and ghrelin and hypothalamic inflammation.
- Ongoing inflammation fuels impaired mitochondrial function, exacerbates insulin resistance, and promotes hepatic steatosis (fatty liver).
- Glycation accelerates in liver, muscle, and vascular tissues; AGEs crosslink extracellular matrix proteins, increasing vascular stiffness. Repeated postprandial hyperglycaemia accelerates glycation.
- Immune cells in adipose and vessel walls respond with inflammatory amplification.

5. **5. Obesity emerges & glycation drives inflammation — downstream of insulin load**

- Hyperinsulinemia precedes and drives weight gain in many individuals; obesity is often an outcome, not the origin. Especially in youth, where beta-cell resilience is limited.
- Chronic exposure to glycation products increases oxidative stress, stiffens the vasculature, and damages renal filtration structures and pancreatic β-cells.
- Inflammation is now systemic: -
  – Endothelial cells, liver (via NAFLD), skeletal muscle (via lipotoxicity), kidney and β-cells.
  – Adipose tissue macrophages release increase IL-6 levels, stimulating hepatic CRP production.

6. **6. T2DM develops once beta-cell function falters**

- In youth, β-cell decline can be more rapid and often severe.
- Persistent hyperglycaemia entrenches glycation, vascular injury, and chronic inflammation.
- Glucotoxicity, lipotoxicity, and immune-mediated inflammation drive β-cell apoptosis and multi-organ complications (retinopathy, nephropathy, neuropathy, CVD).

While blood testing remains the conventional standard, urine and saliva present promising, less resource-intensive alternatives, especially for screening inflammatory markers in vulnerable populations, who may be reluctant or unable to undergo venipuncture.[168] [169]

People diagnosed with type 2 diabetes mellitus (T2DM) are substantially more likely to develop multiple co-existing conditions, and multimorbidity is the norm rather than the exception in this population. Increasingly, scientific research identifies a shared spectrum of upstream risk factors and overlapping pathophysiological pathways that drive the range of conditions commonly associated with a T2DM diagnosis.[170]

Glycation is thought to be a key contributor to the development of diabetes-related complications, contributing to the increased risk of multiple, overlapping health conditions. In parallel, recent meta-analyses and pooled evidence consistently demonstrate that elevated hsCRP is associated with an increased risk of incident type 2 diabetes and cardiometabolic events. The development of T2DM and cardiovascular disease share common inflammatory aetiologies, for which hsCRP serves as a well-validated biomarker. Elevated hsCRP concentrations are associated with a higher risk of both prediabetes and established T2DM.[171] [172] [173] [174]

Over time, this persistent low-grade inflammatory milieu, effectively captured by hsCRP, contributes to progressive microvascular damage, particularly affecting the eyes, kidneys, and peripheral nerves.[175] [176] [177] [178]

Without such an appreciation, guidelines may over-emphasise downstream correlates, such as obesity as a primary driver, and under-emphasise the contribution of the cumulative carbohydrate burden to elevated blood glucose, inflammation, insulin resistance and eventual diabetes.

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[177] Nowotny K, Jung T, Höhn A. *et al.* (2015). Advanced glycation end products and oxidative stress in type 2 diabetes mellitus. *Biomolecules*. 5(1):194-222. DOI: 10.3390/biom5010194.
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3. BRAIN HEALTH: CONSISTENTLY ASSOCIATED WITH METABOLIC DYSFUNCTION

An extraordinary amount of scientific research reveals how conditions previously considered exclusively brain-related, commence as metabolic dysfunction. Metabolic dysfunction is inherent to the pathophysiology of mental illness.[179] [180] [181] [182] [183] [184] [185] [186]

The data arises from multiple levels of investigation, including cellular and mechanistic studies, case reports, cohort studies, and population-level (epidemiological) research.

High refined carbohydrate and/or ultraprocessed food diets are conventionally low in bioavailable nutrients, and the relative deficiency in these diets increases risk for cascading and overlapping metabolic, mental and immune system illnesses. Metabolic syndrome is a common correlate when these pressures converge, driving inflammation, and creating feedback loops that can overwhelm the body's capacity to repair.

The central nervous system and the digestive tract are inter-dependent. A healthy microbiome is essential to optimise bi-directional neuroendocrine signalling, for sensory-motor reflexes, immune activation, gut brain cross-talk and hormonal signalling. [187] [188]

Biomarker studies have tracked relationships between diet quality, metabolic health, and risks for mental disorders including anxiety, depression, addiction and suicidality.[189] Risk factors can overlap, amplifying conditions or increasing the severity of symptoms and diseases.

> *Inadequate intake, systemic diseases, medical therapies, and genetic conditions can lead to deficiencies of specific nutrients, affecting both the central and peripheral nervous systems.*[190]

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[189] Yuan, S.; Zhu, T.; Gu, J. *et al.* (2025). Associations of Ultra-Processed Food Intake and Its Circulating Metabolomic Signature with Mental Disorders in Middle-Aged and Older Adults. *Nutrients* 17:1582. DOI:10.3390/nu17091582
[190] Muscaritoli M (2021). The Impact of Nutrients on Mental Health and Well-Being: Insights From the Literature. *Front. Nutr.* 8:656290. DOI: 10.3389/fnut.2021.656290

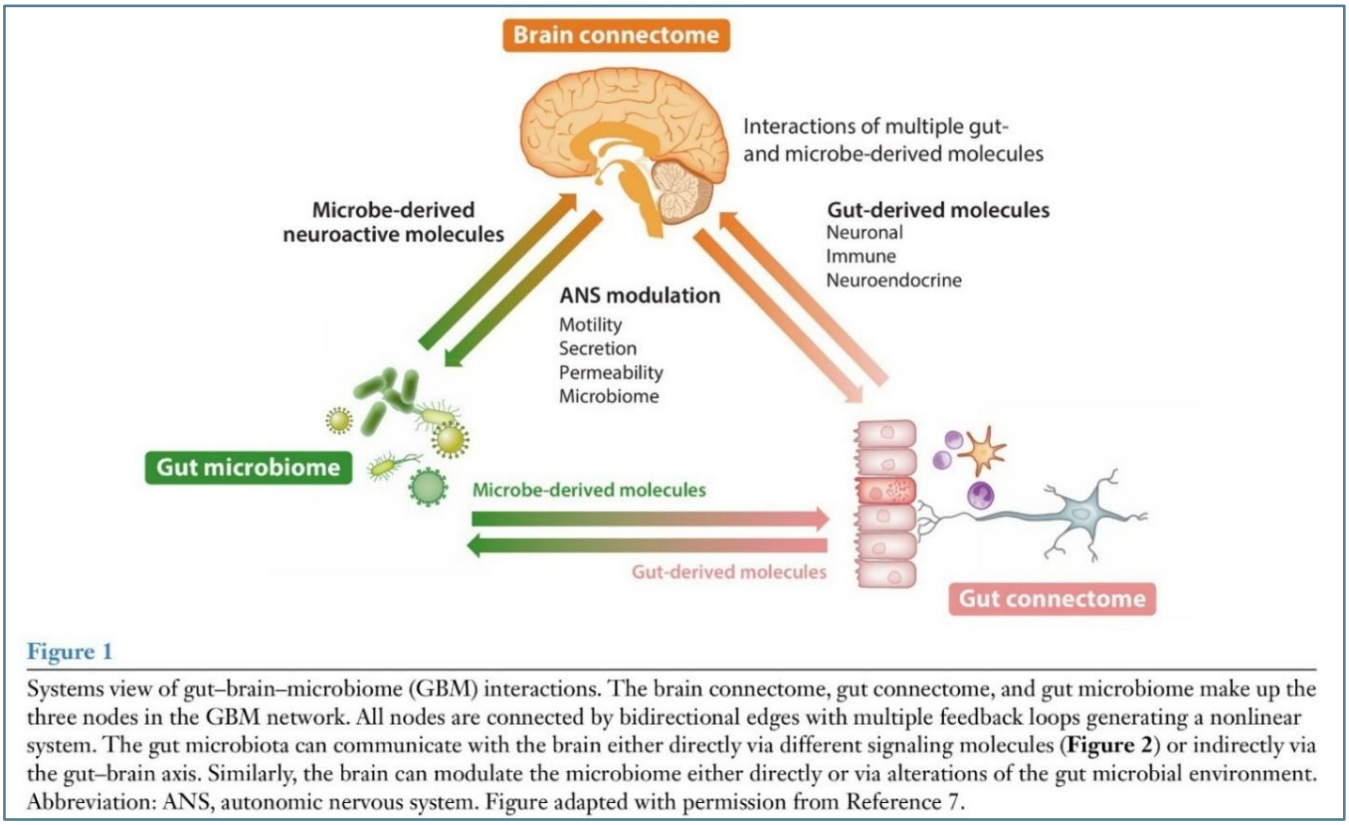

Figure 1

Systems view of gut–brain–microbiome (GBM) interactions. The brain connectome, gut connectome, and gut microbiome make up the three nodes in the GBM network. All nodes are connected by bidirectional edges with multiple feedback loops generating a nonlinear system. The gut microbiota can communicate with the brain either directly via different signaling molecules (**Figure 2**) or indirectly via the gut–brain axis. Similarly, the brain can modulate the microbiome either directly or via alterations of the gut microbial environment. Abbreviation: ANS, autonomic nervous system. Figure adapted with permission from Reference 7.

Figure 4. Mayer EA, Nance K, Chen S. (2022). The Gut–Brain Axis. *Annual Review of Medicine.*

When these factors overlap and the stressors accrue over years and decades, brain function can be severely impacted. In a book on mental health and mitochondrial function, Harvard-based Chris Palmer has argued that:

> *mental symptoms are like the canary in the coal mine: they are sometimes the first indication of metabolic and mitochondrial failure.*[191]

People with psychiatric diagnoses are rarely diagnosed with a single condition.

> *Multimorbidity is common in those with mental illness, and further deterioration of mental health, reduced quality of life, and premature mortality have been reported in those with multiple disorders*.[192]

Symptoms of mental illnesses can overlap. Therefore, one person may be diagnosed for multiple brain disorders and receive prescriptions for multiple psychiatric medications.[193]

For example, while patients with schizophrenia die 20 years earlier than healthy populations, the mortality risk is predominantly associated with cardiovascular risks. As this paper will discuss below, treatment for

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individuals with major depressive disorder, bipolar disorder, and schizoaffective disorder can include metabolic and low-carbohydrate approaches.[194 195 196 197 198 199]

The under-25 age group may be most severely affected. There is a:

> *'greater burden of physical multimorbidity in people with severe mental illness compared with those without is higher for younger cohorts, reflecting a need for earlier intervention.*[200]

Once a person is diagnosed with metabolic and psychiatric conditions, they will be prescribed a range of medical drugs for these conditions. The drugs may produce a range of side effects, which can necessitate additive prescriptions for other drugs. The drugs can also deplete the gut microbiome, which can create further disorder. Multiple drug regimens, or polypharmacy has substantially increased in recent decades.

> *In the US, for example, the prevalence of polypharmacy among adults aged 65 and older increased from 13% in 1998 to 43% in 2014,2425 with the most recent estimates from 2017-2018 at 45%.This increase was driven in particular by the growing use of cardioprotective and antidepressant drug treatments, and the highest prevalence of polypharmacy is seen among populations with heart disease.*[201]

The gut-brain connection.

An impaired gut microbiome can produce cascading, interrelated metabolic and mental health outcomes.

> *Diet is a foundational factor in shaping the gut microbiota, influencing its composition, diversity, and functionality, which in turn affects a wide range of health outcomes through complex microbial-host interactions.*[202]

Diet plays a central role in sustaining a healthy gut-brain-axis, the complex neuro-immuno-endocrine signalling pathway[203] that is fundamental for sustaining good mental health.[204]

> *Communication within this system is nonlinear, is bidirectional with multiple feedback loops, and likely involves interactions between different channels.*[205]

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Human bodies are complex and there are myriad ways poor diets and insufficient vitamin and mineral levels can affect brain function and mental health over time. Brain fog is associated with elevated glucose levels and gluten sensitivity.[206] Refined food diets and chemical exposures, genetic, and epigenetic stressors can increase risk for digestive disorders and impair nutrient processing and the synthesis of hormones, impair sleep patterns, cortisol regulation and promote fatigue and brain fog.

Key factors which contribute to inflammation and health decline include the following:

- Insulin resistance (IR) which plays a crucial role in the development and progression of metabolism-related diseases including diabetes, hypertension, tumours, and non-alcoholic fatty liver disease.[207]
- Frequent consumption of ultraprocessed foods that increase risk for insulin resistance.[208]
- Gluten-heavy diets that can impair digestive tract function and result in a cascade of events which include symptoms which fit the criteria for many mental illnesses.[209] [210] [211]
- Increasing burdens of synthetic chemicals that have toxic effects, which can be at higher levels in refined and ultraprocessed foods, including synthetically refined ingredients and additives, microplastics from packaging, and increasing use of pesticides in staple food crops.[212]
- The inflammatory potential of chemically refined vegetable oils.[213] [214]
- Suppression of ketone bodies which have anti-oxidative, anti-inflammatory, mitochondrial, neurological and cardio-protective features.[215] [216] [217]
- Lower than optimum (insufficient) intakes of micronutrients which are physiologically indispensable for metabolic and/or immunological and/or physiological health, including mental health.

Mitochondria play a central role in metabolic function, and the systematic impact of metabolic stress can be observed at a microscopic level in the mitochondria.[218] Psychiatrists diagnose mental illness based on

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overactive, underactive or absent brain functions. However, the dysregulation of the mitochondria can drive these symptoms. Five distinct cellular processes are involved, whereby cells can either become overactive, underactive, abnormal, defunct or dead, or unable to function correctly and in disrepair. [219]

A spectrum of related processes such as chronic inflammation, altered gut integrity and dysbiosis, and dysregulation of the HPA axis (the body's stress response system) can negatively affect metabolic homeostasis and mitochondrial function. They are all associated with risk for a psychiatric diagnosis. This includes impaired sleep and cortisol production.[220] [221] [222]

There is strong evidence that many if not most of the classic symptoms of depression[223] can be associated with a poor diet, metabolic syndrome and poor digestive tract functioning.[224] [225] [226] [227]

PSGRNZ do not downplay or underestimate the role of chronic stress, trauma and grief in driving temporary (ranging from weeks to years) poor mental health but instead draw attention to factors which may lead to a shortening, a reduction or reversal in the symptoms experienced by the person who is suffering. Evaluations of the role of higher-dose nutrients in times of trauma and stress, to identify whether people had improved outcomes, were more resilient and recovered more swiftly have been undertaken.

> *Following natural disasters of earthquake (Christchurch, Aotearoa/New Zealand, 2010–11) and flood (Calgary, Canada, 2013), controlled research showed statistically and clinically significant reductions in psychological distress for survivors who consumed minerals and vitamins (micronutrients) in the following months.*[228]

Accumulation of toxins in the brain may also alter brain function. For example, people with autism spectrum disorder appear to have difficulty regulating mitochondria-related processes of apoptosis, which leads impaired autophagy. This can increase risk for an accumulation of toxic products in the brains of individuals with autism.[229]

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Poor sleep can be associated with inadequate nutrition and high intakes of ultraprocessed food is associated with poor sleep-related outcomes.[230] [231] Sleep cycles play a key role in eliminating neurotoxic metabolites, waste products, that without clearing could contribute to dementia and poor brain health. Poor sleep cycles may lead to a reduction in the brain's capacity to clear toxic waste, creating negative feedback loops that further impair mental health.[232] [233]

New Zealand government officials do not undertake this work that deepens public information and general practitioner knowledge on the relationship between nutrition and brain health.[234] [235] [236] Without an official effort review the scientific literature and update agency staff, nutrients critical for health can be poorly and incorrectly categorised due to out-dated legacy perspectives.

Introducing Nutritional Psychiatry.

Nutritional psychiatry is a growing sub-specialty of psychiatry. Mechanistic, observational and interventional data increasingly demonstrates that diet is a modifiable risk factor for mental illness.[237] Studies researching nutrition and psychiatry have exploded in the past 15 years.[238]

> *'nutritional psychiatry encompasses the study of dietary and nutrient-based interventions for the prevention or treatment of mental disorders. The concept of nutraceuticals refers to non-toxic dietary extracts or supplements with scientifically validated benefits for promoting health and aiding in disease management.'*[239]

PSGRNZ emphasise that psychotherapy, connection and support play an integral part of healing and management of brain and mind-related challenges. Human connection is central to the healthy functioning of all of us. The greater outcome from psychotherapy, friendship and community engagement includes deeper self-understanding, enhanced self-agency, and greater social engagement. [240]

Nutritional psychiatry which integrates nutritional and dietary changes, complements traditional psychotherapy, may play a key role for people who are treatment resistant and may assist with recovery. Dietary modifications may be an underutilised tool for people diagnosed with a psychiatric condition.[241]

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An established and increasing scientific literature demonstrates that metabolic disruption and subclinical nutrient deficiency is a precursor and a companion to a wide range of metabolic and mental illness.[242] [243] [244] Deficiency across a spectrum of micronutrients, can follow months and years of inadequate intakes.[245] Studies consistently show that nutrient insufficiency is common in people with diagnosed with many brain-related conditions including depression and anxiety[246] [247] [248] and ADHD.[249] [250]

It is rarely one nutrient that bodies are missing and treatments with individual nutrients may result in inconsistent trial results. evidence is growing that dietary change and micronutrient supplementation which broadly raises nutrient intake levels may be more effective.[251] [252] [253]

Once ill, people are more likely to be diagnosed with multiple health conditions. Exposure to stressors such as trauma, can further promote systemic inflammation and disease risk, producing cascading harms for an individual.

Dietary shift exerts overlapping complex effects which can improve and repair gut microbiome function, lower the inflammatory burden, and increase nutrient intake. Practitioners in the field of metabolic and psychiatric nutrition, adopt a spectrum of flexible approaches that revolve around reducing carbohydrate intakes to reduce, mitigate and eliminate the markers of poor metabolic health which frequently underlie poor brain health. The approach necessarily involves psychological, behavioural and practical skills coaching.

Therefore, when people reduce ultraprocessed food intakes, glucose and gluten burdens, and shift to wholefood diets that are low in refined and chemically synthesised ingredients, reversal of multiple clinical parameters can occur. Dietary changes can include the elimination of foods that may play a triggering or mediating role in many chronic symptoms and conditions.[254] Biomarker testing, case studies and trials consistently report multiple positive outcomes across multiple clinical parameters.

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Low-carbohydrate diets can upregulate endogenous ketone body production by shifting the metabolism toward increased fat oxidation. This may be a key mechanism underpinning many of the observed improvements in metabolic and mental functioning. Ketone bodies are produced when the body shifts from using glucose to mobilising stored fat for energy, have been consistently shown to provide important benefits for brain function and health.

From birth, humans are physiologically adapted to tolerate periods of fasting and food scarcity, and can flexibly transition into a state of nutritional ketosis when carbohydrate availability is low.[255]

Ketone bodies may be produced naturally by the body or provided through external supplements. Growing research is revealing how these molecules influence metabolism and brain function, making their therapeutic potential an exciting and fast-moving area of science. [256] [257] [258] [259] [260]

Ketone bodies not only function as fuel, but also as signalling metabolites with applications in health and disease. Scientists and clinicians are therefore regarding exogenous sources of ketone bodies, such as through infusion of beta-hydroxybutyrate (BHB), as a potential therapeutic treatment to reduce blood glucose, and improve performance, endurance/resilience and health outcomes. [261] A recent review found that dosing regimens of BHB produced more consistent results in healthy than non-healthy populations.[262] [263] [264]

Much of the early work in psychiatric nutrition was undertaken in an effort to improve health outcomes of treatment-resistant patients. For example, psychiatrist Georgia Ede's approach was adopted after a French colleague, Dr Albert Danan, conducted a trial on 35 treatment resistant patients who were diagnosed with major depression, bipolar disorder (schizoaffective disorder). Patients were placed on a close supervision ketogenic diet. All were on multiple psychiatric medications, and all had been previously hospitalised. Many of the treatment resistant patients had high blood glucose, high blood pressure, high triglycerides and obesity and many could not work due to the psychiatric disability.[265]

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[261] Liao LP, Church LA, Melville H, *et al* (2025). Effect of ketone supplementation, a low-carbohydrate diet and a ketogenic diet on heart failure measures and outcomes: a systematic review and meta-analysis. *Heart*. DOI: 10.1136/heartjnl-2025-326082

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- By week three the 28 of the original 35 began improving metabolically and psychiatrically.
- 23 people with depression symptoms experienced substantial improvements in mood.
- All 10 people with schizoaffective disorder experienced substantial reduction in psychosis symptoms.
- 12 people (44%) achieved full clinical remission.
- 18 people substantially reduced psychiatric medication.
- All but one lost weight.

Danan's diet protocol was adapted from a protocol developed by a Dr Eric Westman at Duke University.[266] [267] The diet consisted almost exclusively of meat, seafood, poultry, eggs, vegetables, nuts and cheese and was well tolerated by the patients.[268]

Multiple disease or symptom parameters (and multimorbidity) can regress, following a dietary shift.[269] [270] In a case of a 38-year-old female diagnosed with post-traumatic stress disorder, ADHD, binge eating disorder, bipolar II disorder, depression, anxiety, and premenstrual dysphoric disorder was placed on an insulin lowering ketogenic diet.:

> *By week 12, all psychiatric symptoms resolved evidenced by quantitative reductions to 0 across all validated instruments. The patient consistently reported optimal symptom control when blood ketone levels were maintained between 3 and 5 mmol/L. Qualitative reports substantiated marked functional gains, including improved occupational engagement and social functioning.*[271]

The case above highlights the complex interplay between trauma, addictive behaviours and eating disorders. Diets high in rapidly absorbed carbohydrates can trigger sharp dopaminergic responses via mesolimbic reward pathways, while simultaneously driving spikes and crashes in blood glucose and insulin. Micronutrient insufficiency may play a significant role, particularly in younger adults.[272] [273] This combination of nutrient insufficiency, transient reward, followed by metabolic depletion and dysphoria, may heighten sensations of emptiness and reinforce repetitive seeking of the same foods. Compounding this, the widespread belief that dietary fat drives body fat can lead to unhealthy suppression of this essential macronutrient class, undermining satiety and further destabilising eating patterns. Vegetarianism may also be more commonly represented in eating disorder groups with the fat and protein intake under-

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represented.[274] Eating disorder literature rarely addresses the role of healthy saturated fats and proteins in supporting a return to adequate micronutrient status and in cutting short the addictive dopaminergic cycle.

When layered onto sociocultural pressures around body shape and health, pressures that disproportionately affect women, media influence and media, and the expansion of psychiatric categories, these interacting cultural, neurochemical, metabolic and psychosocial mechanisms may contribute meaningfully to the emerging pattern of eating-disorder vulnerability.[275] Low-carbohydrate and ketogenic diet researchers and clinicians have stepped into this field of research, with some success.[276] [277]

A cautionary approach is warranted. The person in the case study above carries a spectrum of risks and could revert to earlier dietary patterns and psychosis, or alternatively, the person may remain stable for the foreseeable future. Care involves navigation over time and people can be medication-supported and nutrition-supported and can taper off to drug-free states.

Nutritional psychiatry is stepping into the treatment void for many people who may choose not to take psychiatric drugs, and can address therapeutic gaps where people have found that conventional medical treatment has not suppressed symptoms (treatment failure), or where they have found adverse effects to be intolerable.

Are Symptoms of Inadequate Nutrition Misclassified as Psychiatric Disorders?

Psychiatric nutrition is a companion partner to conventional psychotherapy because nutrition enhances physiological health. Many of the 'classic' symptoms used to diagnose a psychiatric condition may have arisen due to insufficient nutrition or inadequate nutrient absorption over time, and poor mitochondrial (and cellular) health.

Many of the symptoms of depression[278], anxiety[279] and ADHD[280] that are listed in the *Diagnostic and Statistical Manual of Mental Disorders*[281], and that lead to a diagnosis and subsequent prescription, can be similarly attributable to dietary inadequacy, nutritional deficiencies and poor digestion.

The role of dietary nutrition in protecting from many of the symptoms of depression, including fatigue, insomnia, brain fog, is now well established.

- Depressed mood.
- Markedly diminished interest or pleasure in most or all activities.
- Poor appetite, weight loss, or weight gain.
- Insomnia or hypersomnia.
- Slowing down of mental or physical activities (for example, sluggishness or diminished hand-eye coordination.
- Fatigue or loss of energy.

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[274] Sergentanis TN, Chelmi M-E, Liampas A, *et al.* (2021). Vegetarian Diets and Eating Disorders in Adolescents and Young Adults: A Systematic Review. Children. 8(1):12. DOI:10.3390/children8010012

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[280] Mental Health Foundation (Sept 2022). ADHD in Children. https://mentalhealth.org.nz/conditions/condition/adhd-in-children

[281] Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, Text Revision (DSM-5-TR) https://doi/book/10.1176/appi.books.9780890425787

- Feelings of worthlessness or excessive or inappropriate guilt.
- Diminished ability to think or concentrate ("brain fog"), or indecisiveness.
- Recurrent thoughts of death; thinking about, planning, or attempting suicide.

Many of these categories might simply reflect inadequate nutrient intakes by age and/or gender, a differently functioning brain, a brain where discrete developmental periods mature at different stages (such as due to brain hemisphere differentiation) and/or deficiency in nutrients required for concentration and focus. These factors depend on complex interrelationships between diet, digestion, physical and social environmental exposures, genetics and methylation capacity.

Exercise is critical for optimum health, retention of healthy muscle and is associated with better mental health. However, fatigue, sleep loss, inadequate protein uptake and inadequate nutrition are often not factored in when people are urged to exercise. Over years, inadequate nutrition, although 'healthy' may result in fatigue in groups that have nutrient requirements that are greater than, or that diverge from, current guideline recommendations.

The pathways, mechanisms and evidence of reversal following dietary shifts provide a compelling body of evidence that nutrition can be, and for some psychiatrists, already is, a first line treatment.[282] [283] [284] Results from trials show that people with major depressive disorder can be helped by making dietary changes, and ketogenic diets may provide one such pathway.[285]

Childhood and adolescent behaviour that is considered non-normative and behaviourally different, when teachers and practitioners clinically diagnose the behaviour of ADHD, sets that child on a path where medical treatment and behavioural strategies are first line treatments, and nutritional status is a minor order issue. The diagnostic criterion for ADHD is difficult to navigate [286] and ambiguous, and the quantity of criteria that are established to confirm an ADHD diagnosis has been arbitrary and flexible.[287]

Proportionately, these issues are not judged as equivalent factors, and there is a knowledge vacuum on the nutrition 'side' while the path is smoothed on the 'medicalisation' side. I.e. access to prescription drugs following a diagnosis of poor brain/mental health is non-controversial, but dietary changes to reverse or mitigate a brain-related syndrome or diagnosis is much more controversial.

4. THE CARBOHYDRATE-DOPAMINE CYCLE: AMPLIFIED BY ULTRAPROCESSED FOOD

Hyperpalatability & Food Addiction.

While people do not become addicted to protein or vegetables, a high glycaemic carbohydrate intake (especially if spread over multiple meals/snacks) results in repeated postprandial glucose spikes. These glucose spikes are associated with dopamine release, and carbohydrate consumption impacts people differently.[288] Researchers are recognising that multifactorial drivers plausibly amplify health risk:

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[282] Palmer C. (2022). Brain Energy. Benbella Books.
[283] E.g. Google Scholar. Search: 'mental, illness, nutrition, diet'. https://scholar.google.com/scholar?start=10&q=mental+illness+nutrition+diet
[284] Ede G (2024). Change Your Diet, Change Your Mind: A Powerful Plan to Improve Mood, Overcome Anxiety, and Protect Memory for a Lifetime of Optimal Mental Health. *Dimensions*.
[285] Decker DD, Patel R, Cheavens J. *et al*. (2025). A pilot study examining a ketogenic diet as an adjunct therapy in college students with major depressive disorder. *Transl Psychiatry* 15:322;(2025). DOI: 10.1038/s41398-025-03544-8
[286] Peterson BS, Trampush J, Brown M, *et al*. (2024). Tools for the Diagnosis of ADHD in Children and Adolescents: A Systematic Review. *Pediatrics*, 153 (4): e2024065854. DOI:10.1542/peds.2024-065854
[287] Honkasilta J, Koutsoklenis A. (2022). The (Un)real Existence of ADHD—Criteria, Functions, and Forms of the Diagnostic Entity. *Front. Sociol.* 7, DOI: 10.3389/fsoc.2022.814763
[288] Wu, Y., Ehlert, B., Metwally, A.A. *et al*. (2025). Individual variations in glycemic responses to carbohydrates and underlying metabolic physiology. *Nat Med* 31:2232–2243. DOI: 10.1038/s41591-025-03719-2

> *High glycemic index carbohydrates elicit a rapid shift in blood glucose and insulin levels, akin to the pharmacokinetics of addictive substances. Akin to drugs of abuse, glucose and insulin signals in the mesolimbic system to modify dopamine concentration. Sugar elicits addiction-like craving and self-reported problem foods are rich in high glycemic index carbohydrates. These properties make high glycemic index carbohydrates plausible triggers for food addiction.*[289]

Uncertainty has prevailed over whether food addiction is behaviour-based or a function of exposure to substances (i.e. a substance use disorder).[290] [291] However the association of food addiction with industrially formulated ultraprocessed food consumption provides evidence for food addiction as a substance use disorder. Many addictive agents contain formulations of substances which reinforce the addictive potential. The combination of fat and refined carbohydrates in industrially formulated ultraprocessed foods, does not exist in naturally occurring foods. These factors may drive outcomes where:

> *consumption of ultraprocessed foods high in both fat and refined carbohydrates (e.g., Oreo cookies, M&Ms) leads to downregulation of dopamine receptors, binge eating, and willingness to obtain these foods despite negative consequences.*[292]

Naturally occurring, minimally processed foods (fruits, vegetables, nuts, milk and meat protein) have not been associated with food addiction.

Ultraprocessed foods were first formally categorised as group four under the NOVA food classification system. The NOVA system is based on extent of processing and additives in the end food product. Work is being undertaken to validate the NOVA system in countries to account for cultural differences in food products.[293] [294] [295]

Many hyper-palatable ultraprocessed foods containing unnaturally high levels of refined carbohydrates or added fats, including sweets and salty snacks are strongly implicated in the behavioural indicators of addiction.[296] They consistently contain unnaturally high doses of reinforcing ingredients that are effective at activating reward and motivation systems. These calorie-dense foods are optimised; they lack ingredients which slow the rate of absorption, water, fibre and protein. This increases the speed of uptake and impact.[297] [298]

Diets high in ultraprocessed food are associated with an increase in free sugars, total fats, and saturated fats, as well as a decrease in fibre, protein, potassium, zinc, and magnesium, and vitamins A, C, D, E, B12,

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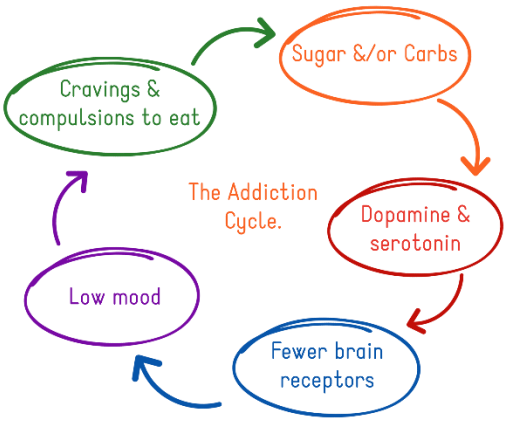
[289] Lennerz B, Lennerz JK. (2017) Food Addiction, High-Glycemic-Index Carbohydrates, and Obesity. Clin Chem. 2018 Jan;64(1):64-71. DOI: 10.1373/clinchem.2017.273532.
[290] Gordon EL, Ariel- Donges AH, Bauman V, et al. (2018) What is the evidence for "food addiction?" A systematic review. Nutrients 2018;10:477
[291] Gearhardt AN, & Schulte EM. (2021). Is Food Addictive? A Review of the Science. *Annu Rev Nutr*. 41:387-410. DOI: 10.1146/annurev-nutr-110420-111710.
[292] Gearhardt AN, & Schulte EM. (2021). Is Food Addictive? A Review of the Science. Page 398.
[293] Louzada MLC, Gabe KT. (2025) Nova food classification system: a contribution from Brazilian epidemiology. *Rev Bras Epidemiol*. 28: e250027. DOI:1590/1980-549720250027
[294] Freire WB, Tello BB Guerrón P. (2025) Validation of NOVA 27 ultra-processed food screener: adaptation and performance in Ecuador. *Public Health Nutrition*. 28(1):e105. doi:10.1017/S1368980025100475
[295] Monteiro CA, Cannon G, Moubarac JC, *et al.* (2018). The UN decade of nutrition, the NOVA food classification and the trouble with ultra-processing. *Public Health Nutr*. 21:5–17. DOI: 10.1017/S1368980017000234.
[296] Gearhardt, A. N., Bueno, N. B., DiFeliceantonio, A. G., *et al.* (2023). Social, clinical, and policy implications of ultra-processed food addiction. *BMJ*, 383. DOI: 10.1136/bmj-2023-075354
[297] Schulte, E.M., Avena, N.M., & Gearhardt, A.N. (2015). Which foods may be addictive? The roles of processing, fat and glycemic load. PLOS ONE, 10(2): e0117959. DOI:10.1371/journal. pone.0117959.
[298] Gearhardt AN, & Schulte EM. (2021). Is Food Addictive? A Review of the Science.

and niacin.[299] The proportion of ultraprocessed food in the diet increases the likelihood that people will gain weight.[300]

In a 2025 conference presentation[301] Dr Jen Unwin described the cycle the ultraprocessed food addiction trap:

- Hyperpalatability.
- Suppression of frontal lobe activity.
- Neurons that fire together, wire together.
- Damage to the mitochondria, leading to energy deficits.
- Negative reinforcement: a bad feeling state is temporarily relieved.



The Yale Food Addiction Scale (YFAS) is a clinical research tool that has been developed to measure food addiction by drawing from DSM-5 criteria for substance use disorder. In 2016, the YFAS 2.0 was released to reflect changes to the substance use disorder diagnostic criteria in the Diagnostic and Statistical Manual of Mental Disorders 5 (DSM-5).[302] Ultraprocessed foods include pizza, ice-cream, white bread biscuits and potato chips have been consistently associated with YFAS indicators.

Researchers have since progressed to develop a Food Addiction Symptom Inventory (FASI), a clinician-administered assessment of food addiction. This has been adapted from the Structured Clinical Interview for *Diagnostic and Statistical Manual of Mental Disorders, fifth edition* modules for diagnosing substance-use disorders.[303] Work has further been undertaken to develop simpler approaches to recognise food-related emotional expectancies including addiction that may be of benefit in clinical populations with high participant burdens.[304]

Scientists and researchers have recently moved closer to agreement on this issue.

In 2025 international clinicians and academics in the field conducted a consensus exercise and concluded that the term ultra-processed food addiction (UPFA) appropriately describes the disorder.[305] The consensus statements agreed to by the majority of participants were:

- There is enough evidence to justify the classification of new substance use disorder.
- The disorder should be referred to as ultra-processed food addiction (UPFA).

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[299] Martini D, Godos J, Bonaccio M. *et al*. (2021). Ultra-Processed Foods and Nutritional Dietary Profile: A Meta-Analysis of Nationally Representative Samples. *Nutrients*, 13, 3390. DOI: 10.3390/nu13103390

[300] Dicken SJ, Jassil FC, Brown A. *et al.* (2025). Ultraprocessed or minimally processed diets following healthy dietary guidelines on weight and cardiometabolic health: a randomized, crossover trial. *Nat Med*. 31(10):3297-3308. DOI:10.1038/s41591-025-03842-0

[301] Unwin J. The Growing Epidemic of Ultra Processed Food Addiction. Primary Healthcare Lifestyle Conference September 2025. Birmingham UK.

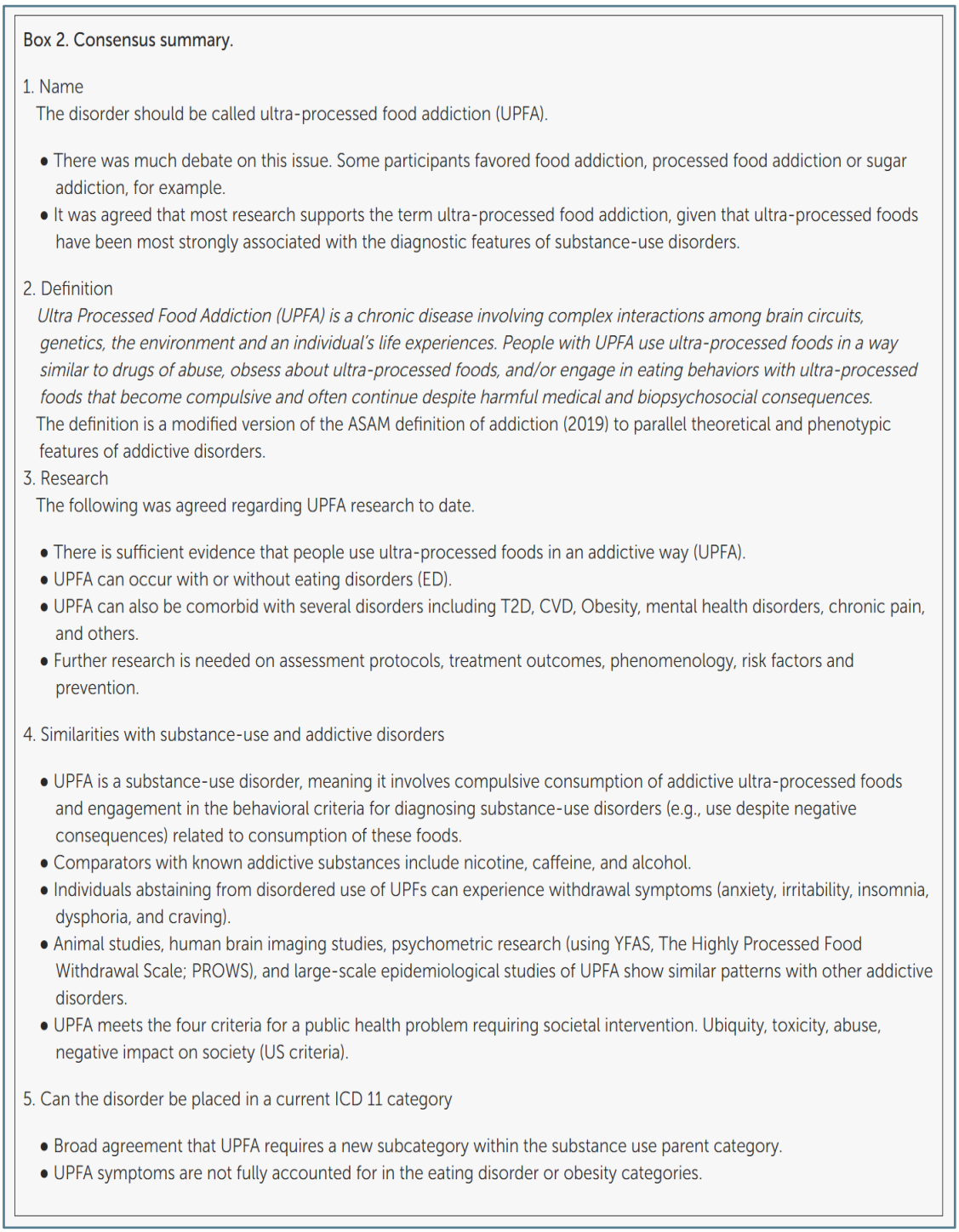
[302] Gearhardt AN., and Loch LK (2024) Assessment of Food Addiction: A DSM-5 Update, in Ashley N. Gearhardt, and others (eds), Food & Addiction: A Comprehensive Handbook, 2nd edn (New York, 2024; online edn, Oxford Academic, 19 Sept. 2024), DOI: 10.1093/oso/9780190671051.003.0001,

[303] LaFata EM, Worwag K, Derrigo K, *et al.* (2024). Development of the Food Addiction Symptom Inventory: The first clinical interview to assess ultra-processed food addiction. *Psychological Assessment, 36*(11), 654–664. DOI: 10.1037/pas0001340

[304] Cummings JR, Treharne N, Vaink, *et al.* (2025). Development and validation of a brief form of the Anticipated Effects of Food Scale. *Appetite*, 206:107843. DOI: 10.1016/j.appet.2024.107843

[305] Unwin J, Giaever H, Avena N, Kennedy C, Painschab M and LaFata EM (2025) Toward consensus: using the Delphi method to form an international expert consensus statement on ultra-processed food addiction. *Front. Psychiatry* 16:1542905. DOI: 10.3389/fpsyt.2025.1542905

- The symptoms that comprise addiction to other substances of abuse in the DSM and ICD describe UPFA symptoms.
- Given that UPFA is a substance use disorder: abstinence from a person's 'drug foods' will form the mainstay of therapy.
- FA exists on a continuum and can co-occur with eating disorders but is a distinct disorder.
- Recognition of the disorder will lead to more research and treatment options.



Box 2. Consensus summary.

1. Name

The disorder should be called ultra-processed food addiction (UPFA).

- There was much debate on this issue. Some participants favored food addiction, processed food addiction or sugar addiction, for example.
- It was agreed that most research supports the term ultra-processed food addiction, given that ultra-processed foods have been most strongly associated with the diagnostic features of substance-use disorders.

2. Definition

Ultra Processed Food Addiction (UPFA) is a chronic disease involving complex interactions among brain circuits, genetics, the environment and an individual's life experiences. People with UPFA use ultra-processed foods in a way similar to drugs of abuse, obsess about ultra-processed foods, and/or engage in eating behaviors with ultra-processed foods that become compulsive and often continue despite harmful medical and biopsychosocial consequences.

The definition is a modified version of the ASAM definition of addiction (2019) to parallel theoretical and phenotypic features of addictive disorders.

3. Research

The following was agreed regarding UPFA research to date.

- There is sufficient evidence that people use ultra-processed foods in an addictive way (UPFA).
- UPFA can occur with or without eating disorders (ED).
- UPFA can also be comorbid with several disorders including T2D, CVD, Obesity, mental health disorders, chronic pain, and others.
- Further research is needed on assessment protocols, treatment outcomes, phenomenology, risk factors and prevention.

4. Similarities with substance-use and addictive disorders

- UPFA is a substance-use disorder, meaning it involves compulsive consumption of addictive ultra-processed foods and engagement in the behavioral criteria for diagnosing substance-use disorders (e.g., use despite negative consequences) related to consumption of these foods.
- Comparators with known addictive substances include nicotine, caffeine, and alcohol.
- Individuals abstaining from disordered use of UPFs can experience withdrawal symptoms (anxiety, irritability, insomnia, dysphoria, and craving).
- Animal studies, human brain imaging studies, psychometric research (using YFAS, The Highly Processed Food Withdrawal Scale; PROWS), and large-scale epidemiological studies of UPFA show similar patterns with other addictive disorders.
- UPFA meets the four criteria for a public health problem requiring societal intervention. Ubiquity, toxicity, abuse, negative impact on society (US criteria).

5. Can the disorder be placed in a current ICD 11 category

- Broad agreement that UPFA requires a new subcategory within the substance use parent category.
- UPFA symptoms are not fully accounted for in the eating disorder or obesity categories.

Figure 5. Unwin J, Giaever H, Avena N, Kennedy C, Painschab M and LaFata EM (2025) Toward consensus: using the Delphi method to form an international expert consensus statement on ultra-processed food addiction. *Front. Psychiatry*

The 'dose' of both carbohydrate and fat appears to drive the addictive potential of an ultraprocessed food formulation. Di Feliceantonio et al. (2018) analysed neural responses to ultraprocessed foods containing fat *and* carbohydrate and equicaloric foods containing primarily fat *or* carbohydrate. The study found that reward-related activation peaked with foods high in both fats and carbohydrate, that the neural response was supra-additive, and that the effect was independent of liking.[306]

Prevalence of food addiction may more common than recognised. A recent review finding that 14% of adults and 12% of children, (a reported prevalence that is similar to alcohol addiction) struggle with refined and ultraprocessed food addiction.[307] Food addiction symptoms appear to better fit criteria for substance use disorder than behavioural addiction,[308] yet it is important to note that food addiction is higher in groups with other behavioural addictions. [309]

> *Individual risk factors interact with the addictive potential of a substance to determine the likelihood that a specific individual will become addicted. Individual risk factors that increase a propensity for addiction include a family history of addiction, cognitive control difficulties, trauma exposure, and depression.*[310 311]

Many groups, including children and adolescents, may struggle to limit consumption levels. A spectrum of challenges face children and adolescents if they are to reduce their dependency on high carbohydrate intakes. These not only concern the role of dopamine feedback in young minds, the prevalence of food addiction but relate to the risk of attrition. In a 12-week whole food, carbohydrate restricted dietary trial, drop-out rates were high (48%) with children's adherence influenced, positively and negatively, by levels of support from friends and family.[312]

Current Ultraprocessed Food Intakes Associated with Poor Health Outcomes.

While carbohydrates were always in diets, ultraprocessed foods drive addictive actions which further displace nutrient dense foods. Poor diets drive mental illness risk and western populations can on average consume 50% of their diets as ultraprocessed foods which are low in bioavailable nutrients. The proportion of the diet that is ultra-processed has grown markedly:

- New Zealand toddlers and pre-schoolers: Ultra-processed foods contributed to the 45% (12 months), 42% (24 months), and 51% (60 months) of energy intake to the diets of children.[313]
- U.S. young people under age 19 consume on average 67% ultraprocessed food in their diet.[314]

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[306] DiFeliceantonio AG, Coppin G, Rigoux L, Thanarajah SE, Dagher A, *et al*. (2018). Supra-additive effects of combining fat and carbohydrate on food reward. *Cell Metab*. 28(1):33–44.e3. DOI: 10.1016/j.cmet.2018.05.018

[307] Gearhardt, A. N., Bueno, N. B., DiFeliceantonio, A. G., *et al*. (2023). Social, clinical, and policy implications of ultra-processed food addiction. *BMJ,* 383. DOI: 10.1136/bmj-2023-075354

[308] Gordon EL, Ariel-Donges AH, Bauman V, Merlo LJ. (2018) What Is the Evidence for "Food Addiction?" A Systematic Review. *Nutrients,* 10(4):477. DOI: 10.3390/nu10040477.

[309] Gaspar-Pérez, A.; Granero, R.; Fernández-Aranda, F.; Rosinska, M.; Artero, C.; Ruiz-Torras, S.; Gearhardt, A.N.; Demetrovics, Z.; Guàrdia-Olmos, J.; Jiménez-Murcia, S. (2025) Exploring Food Addiction Across Several Behavioral Addictions: Analysis of Clinical Relevance. *Nutrients*, 17:1279. DOI:10.3390/nu17071279

[310] Gearhardt, A. N., & Schulte, E. M. (2021). Is food addictive? A review of the science. Annual Review of Nutrition,

[311] Lopez-Quintero C, de los Cobos JP, Hasin DS, *et al.* (2011). Probability and predictors of transition from first use to dependence on nicotine, alcohol, cannabis, and cocaine: results of the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC). *Drug Alcohol Depend*.115:120–30

[312] Zinn C, Schmeidel O, McPhee J *et al*. (2018). A 12-week, whole-food carbohydrate-restricted feasibility study in overweight children. *Journal of Insulin Resistance.* 3:1, DOI:10520/EJC-1053260947

[313] Fangupo LJ, Haszard JJ, Taylor BJ, Gray AR, Lawrence JA, Taylor RW. (2021).Ultra-Processed Food Intake and Associations With Demographic Factors in Young New Zealand Children. *J Acad Nutr Diet.* 121(2):305-313. DOI: 10.1016/j.jand.2020.08.088.

[314] Wang L, Steele EM, Du M, Pomeranz JL *et al*. (2021). Trends in Consumption of Ultraprocessed Foods Among US Youths Aged 2-19 Years, 1999-2018. *JAMA,* 326(6):519-530. DOI: 10.1001/jama.2021.10238.

- U.S. adults consume around 60% of their diet in ultra-processed food.[315]
- British children consume more than 60% of their calories as ultra-processed food.[316]
- Canadian children and adolescents consumed over half their dietary calories as ultra-processed food.[317]
- Australian children consume more than 42% of their calories as ultra-processed food, processed culinary ingredients (6.8% and minimally or unprocessed foods (35.4%).[318]

The addictive potential of these foods seems to present the challenge that generally accompanies dopamine-inducing technologies. For many people, stopping at a designated 'safe' level may not be feasible. Ultraprocessed food intakes which form more than 30% of daily calorie intake seems to increase risk.[319] While lower intakes (e.g., in the lowest quintile, often <10–20% of energy) generally serve as the reference group with no excess risk, a universal 'safe' threshold has not been established.

However, diets that are high in carbohydrates, even where ultraprocessed food component is less than 30% can promote repeated elevations in blood glucose levels, increasing risk for insulin resistance and T2DM. Other carbohydrate foods that are less processed, cumulatively add to the cumulative burden, increasing blood glucose and therefore triglyceride levels in the body. These can include rice, bread, pasta, starchy vegetables and fruits. The body burden of sugars and starches potentially cumulatively contribute to the potential for hyperinsulinemia and insulin resistance to present in an individual.

Current consumption levels of ultraprocessed foods are strongly associated with escalating harms which impair quality of life, well into adulthood. Most studies report a dose–response between ultra-processed food intake and adverse metabolic and mental-health outcomes. Diets high in ultraprocessed food are associated with premature mortality, inflammatory bowel diseases, impaired reproductive health and

[315] Juul F, Parekh N, Martinez Steele E, Monteiro CA, Chang VW. (2022). Ultra-processed food consumption among US adults from 2001 to 2018. *The American Journal of Clinical Nutrition*. 115(1):211-221. DOI: 10.1093/ajcn/nqab305 oi:
[316] Timmins G, O'Hare R. (June 14, 2021). Urgent action needed to reduce harm of ultra-processed foods to British children. Imperial College London. https://www.imperial.ac.uk/news/223573/urgent-action-needed-reduce-harm-ultra-processed/
[317] Polsky JY, Moubrac JC, Garriguet (2020). Consumption of ultra-processed foods in Canada. *Statistics Canada*. DOI: 10.25318/82-003-x202001100001-eng
[318] Machado PP, Steele EM, Levy RB, *et al.*(2019) Ultra-processed foods and recommended intake levels of nutrients linked to non-communicable diseases in Australia: evidence from a nationally representative cross-sectional study *BMJ Open* 2019;9:e029544. DOI: 10.1136/bmjopen-2019-029544
[319] Global Food Research Program (Nov 2023). Ultra-processed foods. A global threat to public health. https://www.globalfoodresearchprogram.org/wp-content/uploads/2023/11/GFRP_FactSheet_UltraProcessedFoods_2023_11.pdf?

metabolic fitness, obesity, non-alcoholic fatty liver disease, wheezing, poor sleep, metabolic syndrome (including T2DM) obesity, male and female fertility and cardiovascular risks.[320] [321] [322] [323] [324] [325] [326] [327] [328] [329]

Ultraprocessed food consumption increases health risk from conception on. For example, the greater intake of ultraprocessed food in pregnancy, the increased potential for T2DM.[330]

Ultraprocessed food intakes are associated with a broad range of neuropsychiatric outcomes. Studies correlate ultraprocessed food consumption with neurodegeneration, cognitive decline, dementia, and mood disorders.[331] Eight cohort studies now demonstrate a strong association of depression with ultraprocessed food intake.[332] Increasing evidence links aggression and antisocial behaviour with a poor diet.[333]

Scientists increasingly observe associations of poor dietary intakes with anxiety,[334] obsessive compulsive disorder (OCD)[335] and conditions involving psychosis.[336]

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[320] Nilson EAF, Delpino FM, Batis C, Machado PP et al (2025) Premature Mortality Attributable to Ultraprocessed Food Consumption in 8 Countries. *Am J Prev Med* 68(6)1091-1099. DOI: 10.1016/j.amepre.2025.02.018

[321] Barbaresko J, Bröder J, Conrad J *et al.*(2024). Ultra-processed food consumption and human health: an umbrella review of systematic reviews with meta-analyses. *Critical Reviews in Food Science and Nutrition*. 65(11) 1999-2007. DOI: 10.1080/10408398.2024.2317877

[322] Lane MM, Gamage E, Du S, *et al*. (2024) Ultra-processed food exposure and adverse health outcomes: umbrella review of epidemiological meta-analyses *BMJ* 384 :e077310, DOI:10.1136/bmj-2023-077310

[323] Gutierrez-Ortiz C., Guariguata L, Dénos C. *et al*. (2025). Impact of ultra-processed foods consumption on the burden of obesity and type 2 diabetes in Belgium: a comparative risk assessment. *BMC Public Health* 25, 1097. DOI: 10.1186/s12889-025-22304-3

[324] Su, X., Chen, G., Shi, S. *et al.* (2025). Association between ultra-processed foods and female infertility: a large cross-sectional study. *BMC Public Health* **25**, 2213 (2025). DOI:10.1186/s12889-025-23458-w

[325] Preston JM, Iversen J, Hufnagel A *et al.* (2025). Effect of ultra-processed food consumption on male reproductive and metabolic health. *Cell Metabolism*. 37:1-11. DOI: 10.1016/j.cmet.2025.08.004

[326] Wang ME, Llewellyn CH, Katsoulis M, *et al (*2025). Ten-year trajectories of ultra-processed food intake and prospective associations with cardiovascular diseases and all-cause mortality: findings from the Whitehall II cohort study. *Nutr J.* 24(1):79. DOI: 10.1186/s12937-025-01144-2.

[327] Juul, F., Martinez-Steele, E., Parekh, N. *et al.* (2025). The role of ultra-processed food in obesity. *Nat Rev Endocrinol* DOI: 10.1038/s41574-025-01143-7

[328] Adjibade M, Julia C, Allès B. *et al.* (2019). Prospective association between ultra-processed food consumption and incident depressive symptoms in the French NutriNet-Santé cohort. *BMC Med* 17:78 DOI: 10.1186/s12916-019-1312-y

[329] Mazloomi SN, Talebi S, Mehrabani S *et al,* (2023). The association of ultra-processed food consumption with adult mental health disorders: a systematic review and dose-response meta-analysis of 260,385 participants. *Nutr Neurosci*. 26(10):913-931. DOI: 10.1080/1028415X.2022.2110188. Epub 2022 Sep 12. PMID: 36094005.

[330] Yang J, Yin X, Tobias DK, *et al.* (2025). Ultra-processed Foods and Diet Quality in Association With Long-term Weight Change and Progression to Type 2 Diabetes Among Individuals With a History of Gestational Diabetes Mellitus-A Prospective Study. *Diabetes Care.* 48(10):1827-1836. DOI: 10.2337/dc25-0700.

[331] Lutz M, Arancibia M, Moran-Kneer J, Manterola M. (2015) Ultraprocessed Foods and Neuropsychiatric Outcomes: Putative Mechanisms. *Nutrients* 2025, 17, 1215. DOI:10.3390/nu17071215

[332] Mengist B, Lotfaliany M, Pasco JA. *et al.* (2025). The risk associated with ultra-processed food intake on depressive symptoms and mental health in older adults: a target trial emulation. *BMC Med* 23,172. DOI: 10.1186/s12916-025-04002-4

[333] Prescott SL, Logan AC, LaFata EM. *et al.* (2024) Crime and Nourishment: A Narrative Review Examining Ultra-Processed Foods, Brain, and Behavior. *Dietetics, 3*, 318-345. DOI:10.3390/dietetics3030025

[334] Sun M, He Q, Li G *et al.* (2023). Association of ultra-processed food consumption with incident depression and anxiety: a population-based cohort study. *Food Funct.*, 2023,14:7631-7641. DOI: 10.1039/D3FO01120H

[335] Dawson S, Rucklidge JJ, Schofield G. (2025). Whole Food and Ketogenic-Informed Dietary Interventions for OCD: A Metabolic Perspective. *Current Treatment Options in Psychiatry* 12:25. DOI: 10.1007/s40501-025-00361-0

[336] Martland R, Teasdale S, Murray RM, et al. (2023) Dietary intake, physical activity and sedentary behaviour patterns in a sample with established psychosis and associations with mental health symptomatology. *Psychological Medicine*. 53(4):1565-1575. DOI:10.1017/S0033291721003147

Dietary carbohydrates may be more associated with inflammation than has been formally recognised. In a 20-year cohort study, the follow-up in 2022 identified that chronic systemic inflammation appeared to affect the CVD risk of participants who had a higher carbohydrate intake more substantially, as compared to those with low intake. [337]

Satiety, Glycaemic Volatility and the Drivers of Addictive Eating.

Unlike meals based on refined carbohydrates where food is rapidly absorbed and individuals more swiftly experience symptoms of hunger, an optimum diet will assure between-meal satiety and support the regulation of appetite. It will not elicit addiction-like cravings, and it will prevent glucose 'lows'. These discrete differences are rarely discussed in depth, and it is possible that children and adults misinterpret or conflate homeostatic hunger (regulation of energy balance) with addictive cravings or a glucose 'lows'.[338]

- Satiety is known to be associated with the inter-meal period, through the suppression of hunger and the inhibition of further eating.
  Satiation describes within-meal inhibition and can be said to determine meal size and bring a particular eating episode to an end. [339]

- A glucose 'low', or reactive or postprandial hypoglycaemia can occur 2-5 hours after a high carbohydrate meal. Reactive hypoglycaemia is defined as: *recurrent episodes of hypoglycaemia occurring after consumption of carbohydrate-containing meals.*[340] Symptoms include fatigue, shakiness, irritability and cravings. This can occur independently of obesity or a T2DM diagnosis.[341] For people with T2DM, metformin alone may be insufficient. Continuous glucose monitoring (CGM) technology may help predict and prevent these events.[342]

- Food addiction is recognised as a substance use disorder with ultraprocessed foods recognised as an addictive agent that triggers neurobiological and behavioural responses including loss of control, craving, withdrawal, and tolerance that are similar to other addictive substances.[343] [344]

- Guidelines that provoke a glycaemic response and under-recommend protein may be particularly problematic for children and adolescents, as these groups often have underdeveloped yet

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[337] Giannakopoulou SP, Antonopoulou S, Chrysohoou C. *et al.* (2024). The Impact of Dietary Carbohydrates on Inflammation-Related Cardiovascular Disease Risk: The ATTICA Study (2002–2022). *Nutrients*, 16, 2051. DOI: 10.3390/nu16132051

[338] Lutter M, Nestler EJ. (2009). Homeostatic and hedonic signals interact in the regulation of food intake. J Nutr. 2009;139(3):629-632. DOI: 10.3945/jn.108.097618.

[339] Stribiţcaia, E., Evans, C.E.L., Gibbons, C. *et al.* (2020) Food texture influences on satiety: systematic review and meta-analysis. *Sci Rep* 10, 12929 DOI:10.1038/s41598-020-69504-y

[340] Harris S. (1924) Hyperinsulinism and dysinsulinism. *JAMA* 83(10):729–33. doi: 10.1001/JAMA.1924.02660100003002

[341] Hall M, Walicka M, Panczyk M, Traczyk I. (2021). Metabolic Parameters in Patients with Suspected Reactive Hypoglycemia. *Journal of Personalized Medicine*. 11(4):276. DOI:10.3390/jpm11040276

[342] Younes YR, Cron N, Field BCT *et al.* (2024). Proposed treatment strategy for reactive hypoglycaemia. *Front. Endocrinol.* Vol 15. DOI: 10.3389/fendo.2024.1332702

[343] Gearhardt, A. N., & Schulte, E. M. (2021). Is food addictive? A review of the science. *Annual Review of Nutrition*, 41: 387–410. DOI:10.1146/annurev-nutr-110420-111710

[344] Schiestl ET, Gearhardt AN, Wolfson J. (2023). The qualitative evaluation of food addiction across the lifespan. *Appetite*. 194:107170. DOI: https://doi.org/10.1016/j.appet.2023.107170

hyperactive inhibitory control systems in the brain, factors that mediate motor impulsivity and dietary restraint. These issues may amplify addictive-like eating behaviours.[345] [346] [347]

People who consume high quantities of starchy carbohydrates including breakfast cereals, supermarket bread (including 'whole-grain' when this only forms a limited portion of the bread ingredients), white rice, and ultraprocessed food are exposed to big glucose swings, known as reactive hypoglycaemia, which people who consume a relatively small amount of starchy carbohydrates each day, who are 'metabolically flexible', including people who consume a ketogenic diet, do not experience.

Reactive hypoglycaemia, post-prandial hyperinsulinaemia, or exaggerated insulin secretion after high-glycaemic meals involve pancreatic 'overshoot' responses. Importantly, reactive hypoglycaemia can precede insulin resistance, not just result from it, can occur in people with prediabetes and may predict diabetes.[348] [349] [350]

People who shift away from starchy carbohydrate-based diets, find that these swings reduce, ketones then counter-regulate in low-intake periods, as insulin secretion becomes extremely low and steady. This may also occur over time for people who have been diagnosed with T2DM, particularly in the early years of diagnosis.

Satiety is a multifactorial concept, but nutritionally rich food and macronutrient balance are integral to achieving satiety and reducing snacking and hunger cravings between meals. Studies consistently show that higher protein foods or meals deliver better satiety than energy matched foods with lower levels of protein which inhibit appetite in the period *after* consumption lessen the effect of sensations of hunger on motivation and mood. The order of fat and carbohydrate is contested, but as this paper discusses elsewhere, the degree of processing of these macronutrients, and the adherence to carbohydrate levels that do not spike blood sugar may be more important than prescribed ratios.[351]

Recent papers have shed light on the differential postprandial glycaemic responses (PPGRs) to carbohydrates (including pasta, rice and bread). A small study analysed the association between carbohydrate response type and metabolic traits to demonstrate how people vary in their response. Responses were associated with the level of insulin resistance presenting in those people.[352]

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[345] Hardee J, Phaneuf C, Cope LM, *et al*. (2020). Neural correlates of inhibitory control in youth with symptoms of food addiction. *Appetite* 148: 104578. DOI: 10.1016/j.appet.2019.104578

[346] Via E, Contreras-Rodríguez O. Binge-Eating Precursors in Children and Adolescents: Neurodevelopment, and the Potential Contribution of Ultra-Processed Foods. *Nutrients*. 15(13):2994.DOI: 10.3390/nu15132994

[347] Bennett C, Blissett J. (2020). Interactive effects of impulsivity and dietary restraint over snack intake in children. *Appetite*. 146:104496. DOI: 10.1016/j.appet.2019.104496

[348] Hofeldt FD. (1989) Reactive hypoglycemia. *Endocrinol Metab Clin North Am*. 18(1):185-201. PMID: 2645126.

[349] Shanik MH, Xu Y, Skrha J, Dankner R, Zick Y, Roth J. (2008). Insulin resistance and hyperinsulinemia: is hyperinsulinemia the cart or the horse? *Diabetes Care*. 31 Suppl 2:S262-8. DOI: 10.2337/dc08-s264. PMID: 18227495.

[350] Altuntaş Y. (2019) Postprandial Reactive Hypoglycemia. *Med Bull Sisli Etfal Hosp* 53(3):215–220. DOI: 10.14744/SEMB.2019.59455

[351] Chambers L, McCrickerd K, Yeomans MR. (2015). Optimising foods for satiety. *Trends in Food Science & Technology,* 41(2):149-160. DOI: 10.1016/j.tifs.2014.10.007

[352] Wu, Y., Ehlert, B., Metwally, A.A. *et al*. (2025) Individual variations in glycemic responses to carbohydrates and underlying metabolic physiology. *Nat Med* 31:2232–2243. DOI: 10.1038/s41591-025-03719-2

A two-week continuous glucose monitoring (CGM) study in young adults showed that identical meals (varying in carbohydrate content) produced distinct PPGR patterns across individuals, linked to differences in glycaemic variability, these results were not necessarily related to BMI or age.[353]

The physio-neurological effects of sustained carbohydrate exposure, including its potential to provoke unstable satiety, glycaemic volatility, and complex, neurobiologically mediated responses may precede and accompany food addiction. These patterns are increasingly associated with the development of obesity and T2DM.

Increasing diet-related chronic conditions may not so much be a reflection of simple 'noncompliance' to dietary recommendations, but the fundamental unsuitability of the guidelines for a significant portion of the population.

Dietary fibre plays a key role in satiety and health maintenance, protecting colonic health, supporting gut microflora, metabolite and hormone synthesis and preventing and suppressing inflammation.[354] Fibre is thought to contribute to satiety via affect satiety in many ways, depending on the fibre type, and relating to its ability to bulk foods, increase viscosity, gel in the stomach and ferment in the gut.

However, controversy and lack of consistency in study outcomes fail to elucidate which dietary fibres most clearly confer inflammatory risk or anti-inflammatory benefit, and dietary recommendations fail to make allowance for and protect individuals from gastrointestinal disorders.[355] [356]

Personal responsibility in 'healthy eating' which accords to current dietary guidelines may be discordant when people consume insufficient protein and higher levels of 'low fat' carbohydrates which do not assure between-meal satiety. High carbohydrate dietary recommendations may dovetail with food addiction, disordered sleep patterns and lack of energy.

The language of 'personal responsibility' and 'lifestyle choice' may require revisiting if the addictive potential of ultraprocessed food is widely understood.[357]

Practitioners are not only stepping in to offer health coaching and community programmes to support individuals in navigating the addictive potential and shift away from habitual patterns of food

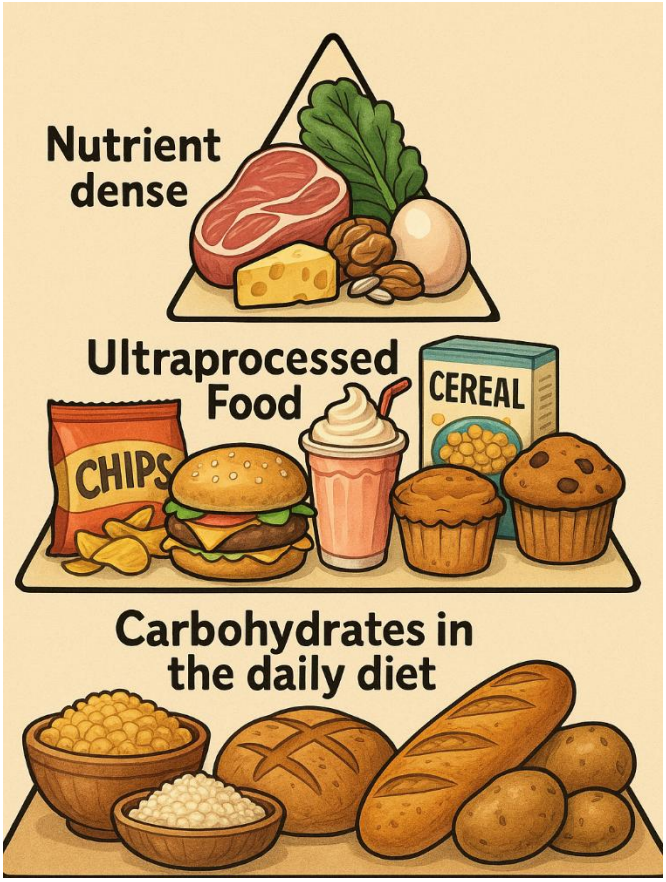

Figure 6. The Problem of Carbohydrate Addiction.

[353] Song J, Oh TJ, Song Y. (2023) Individual Postprandial Glycemic Responses to Meal Types by Different Carbohydrate Levels and Their Associations with Glycemic Variability Using Continuous Glucose Monitoring. *Nutrients*, 15:3571. DOI:10.3390/nu15163571

[354] Barber TM, Kabisch S, Pfeiffer AFH, Weickert MO. (2020) The Health Benefits of Dietary Fibre. *Nutrients.* 12(10):3209. DOI: 10.3390/nu12103209

[355] Kabisch S, Hajir J, Sukhobaevskaia V, Weickert MO, Pfeiffer AFH. (2025). Impact of Dietary Fiber on Inflammation in Humans. *Int. J. Mol. Sci.* 2025, 26, 2000. DOI:10.3390/ijms26052000

[356] Gill SK, Rossi M, Bajka B. *et al.* (2021) Dietary fibre in gastrointestinal health and disease. *Nat Rev Gastroenterol Hepatol* 18:101–116. DOI:10.1038/s41575-020-00375-4

[357] Gearhardt *et al.* (2023). Social, clinical, and policy implications of ultra-processed food addiction.

consumption, they are publishing case studies in the scientific literature to encourage other groups to offer similar services. [358]

These can allow people to address food addiction personally in their communities. Studies show, as we discuss below, that supportive environments can assist with addictive cravings, and facilitate skills development to navigate change away from ultraprocessed food consumption.

Policies that implement advertising restrictions, improve data capture, increase access to nutritious food for lower income groups and that deter lobbying will be amplified by the integration of practitioner-led and wrap-around community-based programmes.

5. ETHICAL CATASTROPHE: THE GREATER BURDEN ON LOW-INCOME GROUPS & YOUNG PEOPLE

Low-income populations not only have nutrient depleted diets but higher levels of exposure to stress and/or trauma. Food insecurity is defined by:

> '*the absence of sufficient, nutritionally adequate, safe foods, as well as the inability to acquire such foods in socially acceptable ways*.'[359]

Food insecurity is a persistent problem in New Zealand.[360] Māori and Pasifika populations are most vulnerable to food insecurity in New Zealand, while women experience food insecurity more than men.[361] Consequent stress is not only nutrition-related, but psychological, despite for example, Māori communities working together to support community members who require additional care.[362]

Foodbanks cannot provide people with adequate nutrition.[363] [364] Food insecure individuals often have limited access to nutrient-rich foods including fruits, vegetables, and meat protein, and much greater access to highly processed foods which are high in refined carbohydrates and processed fats.

New Zealand has a large research cohort exploring the increased risk of substance abuse with poor mental health. Food addiction is yet to be integrated into research programmes and university curricula.

While people across all socio-demographic scales will experience food addiction, people and families experiencing food insecurity and poor mental health are likely to be uniquely susceptible to food addiction over the longer term:[365]

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[358] Zinn C, Campbell JL, Fraser L. *et al*. (2025) Carbohydrate Reduction and a Holistic Model of Care in Diabetes Management: Insights from a Retrospective Multi-Year Audit in New Zealand. *Nutrients*.17(24):3953.
[359] Graham R, Hodgetts D, Stolte O., Chamberlain K. (2018). Hiding in plain sight: experiences of food insecurity and rationing in New Zealand. *Food, Culture & Society.* 21:3;384-401. DOI: 15528014.2018.1451043
[360] Ministry of Health (2019). Household Food Insecurity Among Children in New Zealand. https://www.health.govt.nz/publications/household-food-insecurity-among-children-new-zealand-health-survey
[361] Reynolds D, Mirosa M, Campbell H, (2020). Food and vulnerability in Aotearoa/New Zealand: A review and theoretical reframing of food insecurity, income and neoliberalism. New Zealand Sociology 35:1;123-152. DOI: 10.3316/INFORMIT.219515053019306
[362] Beavis BS, McKerchar C, Maaka J, Mainvil LA (2019). Exploration of Māori household experiences of food insecurity. *Nutrition & Diatetics* 76:344-352. DOI:10.1111/1747-0080.12477
[363] Dey K and Humphries M. (2014) Recounting food banking a paradox of counterproductive growth. ANZTSR https://apo.org.au/node/52943
[364] Riches, Graham. (2011) Thinking and Acting Outside the Charitable Food Box: Hunger and the Right to Food in Rich Societies. *Development in Practice*, 21(4/5):768–75. JSTOR, http://www.jstor.org/stable/41412998.
[365] Burrows T, Kay-Lambkin F, Purser K *et al* (2018). Food addiction and associations with mental health symptoms: a systematic review with meta-analysis. *Journal of Human Nutrition and Dietetics*, 31(4)544-572. DOI: 10.1111/jhn.12532

A similar pattern may exist with food addiction, such that individuals with food insecurity experience similar food addiction symptoms, but greater impairment and long-term negative consequences due to a lack of buffering from other socioeconomic supports. [366]

The chapter above demonstrates that in under-valuing dietary protein and over-emphasising dietary carbohydrates, dietary guidelines may be increasing risk for T2DM, obesity, and a spectrum of metabolic and mental illnesses, conditions which are disproportionately present in low socio-economic groups.

Diabetes Epidemic: The Ethics of Failing to Prevent T2DM in Children & Adolescents.

'Type 2 diabetes is likely to be the biggest global epidemic in human history'. [367] [368]

Childhood T2DM, as with adult onset T2DM, is the result of insulin resistance. T2DM is preceded by a prediabetes state (impaired glucose tolerance and/or impaired fasting glucose). Disease onset and progression proceeds much more rapidly than in adults to frank T2DM.[369] [370] Cardiovascular disease risk is elevated in younger populations with T2DM.[371]

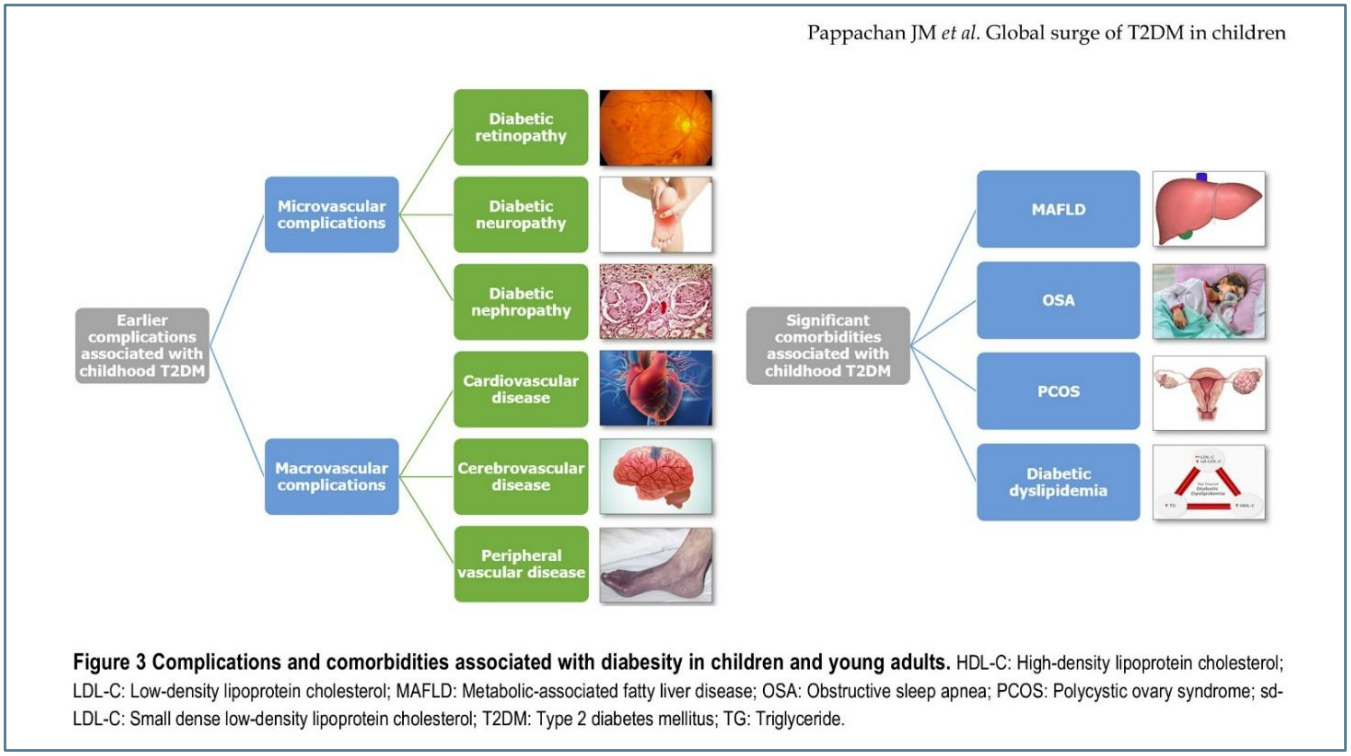

Figure 3 Complications and comorbidities associated with diabesity in children and young adults. HDL-C: High-density lipoprotein cholesterol; LDL-C: Low-density lipoprotein cholesterol; MAFLD: Metabolic-associated fatty liver disease; OSA: Obstructive sleep apnea; PCOS: Polycystic ovary syndrome; sd-LDL-C: Small dense low-density lipoprotein cholesterol; T2DM: Type 2 diabetes mellitus; TG: Triglyceride.

Figure 7. Pappachan JM, Fernandez CJ, Ashraf AP. (2024) Rising tide: The global surge of type 2 diabetes in children and adolescents demands action now. *World J Diabetes*.

[366] Parnarouskis L, Leung CW, Wolfson JA *et al* (2025). The lived experience of Co-occurring food insecurity and food addiction: A qualitative study. *Appetite*. 206:107818. DOI: 10.1016/j.appet.2024.107818

[367] Zimmet, P. Z. (2017). Diabetes and its drivers: the largest epidemic in human history? *Clinical Diabetes and Endocrinology*. 3(1). DOI:10.1186/s40842-016-0039-3

[368] PwC New Zealand (2021). The Economic and Social Cost of Type 2 Diabetes. https://healthierlives.co.nz/wp-content/uploads/Economic-and-Social-Cost-of-Type-2-Diabetes-FINAL-REPORT_Secure-5.pdf Accessed 19/11/25.

[369] Valaiyapathi B, Gower B, Ashraf AP. (2020) Pathophysiology of Type 2 Diabetes in Children and Adolescents. *Curr Diabetes Rev.* 16: 220-229 DOI: 10.2174/1573399814666180608074510

[370] Elder DA, Hornung LN, Herbers PM (2015). Rapid deterioration of insulin secretion in obese adolescents preceding the onset of type 2 diabetes. *J Pediatr* 166: 672-678. DOI: 10.1016/j.jpeds.2014.11.029.

[371] Daga N, Nasir K, Hamirani Y, *et al.* (2013). Prevalence and severity of coronary artery calcium in young persons with diabetes. *J Cardiovasc Comput Tomogr.* 7(4):241-7. DOI: 10.1016/j.jcct.2013.08.004.

Incidence rates of T2DM in children vary, between 285-734 per 100,000 population with 41,600 new cases confirmed in the year 2021.[372] Children who are diagnosed with T2DM are more likely to be obese. A global review and meta-analysis of 53 studies including 8942 participants in 2022, found that 75.27% of children with T2D had obesity, and 77.24% had obesity at diagnosis. [373]

The ethical dimensions arising from inadequate nutritional recommendations for children at risk of metabolic disease remain largely unexplored. Conventional reviews discussing the problem of obesity and diabetes do not generally discuss the aetiology of carbohydrates as an underlying factor in the development of these conditions. The role of bariatric surgery can be mentioned, but not carbohydrate restriction.[374] [375]

There are no formal guidelines recommending carbohydrate restriction in children to reduce or eliminate risk for elevated glucose and T2DM. A recent review by a U.S. based committee affiliated with the Indiana-based Riley Hospital, considered the potential risks of low-carbohydrate approaches, which included: growth deceleration, nutritional deficiencies, poor bone health, nutritional ketosis that cannot be distinguished from ketosis resulting from insulin deficiency, and disordered eating behaviours.[376]

Prevalence of Type 1 (T1DM) and Type 2 diabetes (T2DM) has been increasing. A recent study of adolescents found that T1DM was more prevalent in adolescent females, than males, while T2DM was more prevalent in adolescent males.[377] Increasing rates of T1DM suggest that environmental influences play a role. No single factor has been identified, and scientists have proposed that these may include nutrient insufficiency, impaired gut microbiota, psychosocial stress, and altered immune function.

The review did not consider the risk from long-term exposure to diabetes medication and health conditions that are tightly correlated with T2DM, particularly when it commences in childhood. These include higher risk for diabetic associated retinopathy, neuropathy and kidney disease; diabetic dyslipidaemia, hypertension and cardiovascular disease risk; cerebrovascular and peripheral vascular diseases; metabolic fatty liver disease; obstructive sleep apnoea, hyperandrogenism in females and/or polycystic ovary syndrome (PCOS).[378]

Prediabetes: The Quiet Threat Beneath the Surface.

Increasing rates of prediabetes may be one of the earliest and under-recognised symptoms of the chronic disease epidemic. Prediabetes is a sign of early pathophysiology that precedes T2DM, and prediabetic diagnosis is a condition associated with elevated glucose and inflammation which can be associated with

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[372] Wu H, Patterson CC, Zhang X *et al.* (2022). Worldwide estimates of incidence of type 2 diabetes in children and adolescents in 2021. *Diabetes Res Clin Pract.* 185: 109785. DOI: 10.1016/j.diabres.2022.

[373] Cioana M, Deng J, Nadarajah A, *et al,* (2022) The Prevalence of Obesity Among Children With Type 2 Diabetes: A Systematic Review and Meta-analysis. *JAMA Netw Open.* 1;5(12):e2247186. DOI: 10.1001/jamanetworkopen.2022.47186.

[374] Pappachan JM, Fernandez CJ, Ashraf AP. (2024) Rising tide: The global surge of type 2 diabetes in children and adolescents demands action now. *World J Diabetes* 15(5):797-809. DOI: 10.4239/wjd.v15.i5.797.

[375] Pramanik S, Mondal S, Palui R, Ray S. (2024) Type 2 diabetes in children and adolescents: Exploring the disease heterogeneity and research gaps to optimum management. *World J Clin Pediatr.* 13(2):91587. DOI: 10.5409/wjcp.v13.i2.91587.

[376] Neyman A, Hannon TS; Committee on Nutrition. Low-Carbohydrate Diets in Children and Adolescents With or at Risk for Diabetes. Pediatrics. 2023 Oct 1;152(4):e2023063755. doi: 10.1542/peds.2023-063755.

[377] Chen, X., Zhang, L. & Chen, W. (2025) Global, regional, and national burdens of type 1 and type 2 diabetes mellitus in adolescents from 1990 to 2021, with forecasts to 2030: a systematic analysis of the global burden of disease study 2021. *BMC Med* 23:48. DOI:10.1186/s12916-025-03890-w

[378] Pappachan JM, Fernandez CJ, Ashraf AP, (2024). Rising tide: The global surge of type 2 diabetes in children and adolescents demands action now. *World J Diabetes* 15(5):797-809. DOI: 10.4239/wjd.v15.i5.797.

poor mental health. Prediabetes and diabetes are associated with an elevated risk for depression and anxiety.[379] [380] [381] [382]

Professor Caryn Zinn recently highlighted that prediabetes risk is effectively downplayed by existing clinical approaches, arguing that the term signals a waiting room instead of a treatment window. Zinn argued that instead, prediabetes should be retitled early type 2 diabetes, which would then enable practitioners to refer for tailored dietary and lifestyle intervention.:

> *prediabetes is often a footnote in primary care, flagged inconsistently on lab reports, mentioned briefly or ignored for 'watchful waiting'. This passive approach fails to reflect the biological reality. Long before HbA1c reaches the diagnostic threshold for type 2 diabetes (T2D) (≥48 mmol/mol or ≥6.5%), the disease process is active: insulin resistance, hyperinsulinaemia and β-cell stress drive early microvascular and cardiovascular damage.*[383]

Prediabetes in children and adolescents is increasingly common.[384] [385] The U.S. Centre for Disease Control (CDC) recently identified that 32% of U.S. adolescents between 12-17 years had prediabetes.[386] [387] The CDC used 2023 data from CDC's National Health and Nutrition Examination Survey (NHANES). The CDC advised that the change in methodology had increased the recognised rates of the condition. Only 4 years before, the CDC had identified that 1 in 5 adolescents and 1 in 4 young adults were diagnosed with prediabetes. [388]

New Zealand Ministry of Health data on the prevalence of prediabetes and diabetes in children is difficult to find. New Zealand lacks national data on prediabetes,[389] however, data suggests that prediabetes in children is more prevalent in Pasifika and South Asian children.[390] Doctors and clinicians lack knowledge about prediabetes, and prediabetes is relatively understudied.[391]

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[379] Yu Y, Wan W (2024) Association between prediabetes and depression: A meta-analysis. *PLoS ONE* 19(8): e0307428. https://doi.org/10.1371/journal.pone.0307428

[380] Deschênes SS, McInerney A, Nearchou F, Byrne B, Nouwen A, Schmitz N. Prediabetes and the risk of type 2 diabetes: Investigating the roles of depressive and anxiety symptoms in the Lifelines cohort study. *Diabet Med*. 2023; 40:e15061. doi:10.1111/dme.15061

[381] Topaloğlu, U.S., Erol, K. Fatigue, anxiety and depression in patients with prediabetes: a controlled cross-sectional study. *Diabetol Int* 13, 631–636 (2022). https://doi.org/10.1007/s13340-022-00583-0

[382] Fisher, L., Skaff, M.M., Mullan, J.T., Arean, P., Glasgow, R. and Masharani, U. (2008), A longitudinal study of affective and anxiety disorders, depressive affect and diabetes distress in adults with Type 2 diabetes. *Diabetic Medicine*, 25: 1096-1101. https://doi.org/10.1111/j.1464-5491.2008.02533.x

[383] Zinn C. (2025). Zinn C. Prediabetes is pre-nothing: Call it early type 2 diabetes. *J. Metab. Health*. 8(1):a132. DOI: 10.4102/jmh.v8i1.132

[384] Han C, Song, Q, Ren Y, Chen X *et al*. (2022) Global prevalence of prediabetes in children and adolescents: A systematic review and meta-analysis. *Journal of Diabetes*. 14(7):434-441. Doi: 10.1111/1753-0407.13291

[385] Esquivel Zuniga R, & DeBoer MD. (2021). Prediabetes in Adolescents: Prevalence, Management and Diabetes Prevention Strategies. *Diabetes, Metabolic Syndrome and Obesity,* 14, 4609–4619. DOI: 10.2147/DMSO.S284401

[386] CDC Spotlight on Diabetes Data. Prediabetes in U.S. adolescents. https://gis.cdc.gov/grasp/diabetes/diabetesatlas-spotlight.html

[387] Prediabetes: A fasting plasma glucose (FPG) 100–<126 mg/dL or hemoglobin A1c (A1c) 5.7–<6.5% and the lack of a diabetes diagnosis. In ElSayed NA, Aleppo G, Aroda VR, Bannuru RR, Brown FM, Bruemmer D, *et al*. (2023) Classification and Diagnosis of Diabetes: Standards of Care in Diabetes-2023. *Diabetes Care*. 46(Suppl 1):S19-S40.

[388] Andes LJ, Cheng YJ, Rolka DB, Gregg EW, Imperatore G.(2020) Prevalence of Prediabetes Among Adolescents and Young Adults in the United States, 2005-2016. *JAMA Pediatr*. 174(2):e194498. DOI:10.1001/jamapediatrics.2019.4498

[389] Barthow C, Pullon S, McKinlay E, Krebs J, (2022) It is time for a more targeted approach to prediabetes in primary care in Aotearoa New Zealand. *Journal of Primary Health Care* 14, 372-377. DOI: 10.1071/HC22089

[390] Mazahery H, Gammon CS, Lawgun D, *et al* Pre-diabetes prevalence and associated factors in New Zealand school children: a cross-sectional study. *NZMJ* 12 March 2021, 134(1531) ISSN 1175-8716

[391] McKinlay E, Hilder J, Hood F, *et al*. (2022) Uncertainty and certainty: perceptions and experiences of prediabetes in New Zealand primary care – a qualitative study. *Journal of Primary Health Care,* 14,138-145. DOI: 10.1071/HC21066

New Zealand may have higher rates of prediabetes in children and adolescents than has been formally recognised.[392] A 2021 New Zealand study measured blood glucose (HbA1c) in 451 children, aged 8-11 years. Pre-diabetes was present in 71 (16%) children and was greatest in South Asian (n=13, 30%), Pacific Island (n=29, 27%) and Māori (n=10, 18%) children, compared with European children (n=10, 6.0%) (P< 0.001).[393]

The prevalence of prediabetes increases with age and approximately 67% of people regulatory take hypoglycaemic medication.[394] New Zealand's Ministry of Health 'Data Explorer' indicates that the prevalence of diabetes in 2023/2024 was 6.4% of the adults (over age 15) excluding pregnant women.[395]

Prediabetes and diabetes are more easily reversed at an early stage. When young people are diagnosed with prediabetes and T2DM, this sets the metabolic 'stage' for a spectrum of illnesses at an earlier stage than previous generations, which can then undermine health, wellbeing and productivity in the years to come.

Not Only Nutrition: Environmental Toxins and the Human Exposome.

While this paper focuses primarily on diet and nutrition, acute and chronic exposures beyond diet and nutrition form an essential part of the wider environmental-health framework shaping metabolic and mental wellbeing. Environmental and dietary exposures frequently interact, amplifying risk across the life course. Exposures occurring pre-conception, during pregnancy, infancy and youth can produce long-term deficits in cognition, health and earning capacity, contributing to increased disability, lower lifetime income and reduced quality of life.

Exposures can be tiny, in trace amounts, at parts per million, billion and/or trillion yet can have biologically meaningful effects. Environmental epigenetic factors modulate gene expression through interconnected mechanisms such as DNA methylation, histone modification and non-coding RNA regulation.[396] [397] Endocrine-disrupting chemicals, even at hormonally relevant low doses, can mimic or block normal hormone action, disrupt cellular signalling networks and alter transcriptional patterns. These processes intersect with metabolic pathways including glycolysis, oxidative phosphorylation and fatty-acid oxidation, all of which govern cellular phenotype and function. Exposures at sensitive developmental stages, particularly those affecting the central nervous system, can lead to long-lasting impairments and reduce quality of life.[398] [399] [400] These pathways can directly interfere with the control of food intake and metabolism:

> *including metabolic efficiency via effects on the development of the adipose tissue, pancreas, liver, gastrointestinal tract, brain and/or muscle, thereby resulting in an altered body weight set point or sensitivity for developing obesity across the lifespan and generations. In utero and early development*

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[392] Prediabetes diagnostic criterion is having a haemoglobin A1C (HbA1c) between 41 and 49 mmol/mol (5.8%–6.7%)
[393] Mazahery H, Gammon CS, Lawgun D, Conlon CA, Beck KL, von Hurst PR. (2021). Pre-diabetes prevalence and associated factors in New Zealand school children: a cross-sectional study. *N Z Med J.* 2021 Mar 12;134(1531):76-90.
[394] Health Quality & Safety Commission Te Tahu Hauora (July 2024). Diabetes. https://www.hqsc.govt.nz/our-data/atlas-of-healthcare-variation/diabetes/
[395] Ministry of Health. Indicator: Diabetes (diagnosed, excluding diabetes during pregnancy)
[396] Tiffon, C.(2018) The Impact of Nutrition and Environmental Epigenetics on Human Health and Disease. *Int. J. Mol. Sci.* 2018, *19*, 3425. DOI: 10.3390/ijms19113425
[397] Huang, B.; Jiang, C.; Zhang, R. (2014) Epigenetics: The language of the cell? *Epigenomics* 6:73–88.
[398] Patisaul HB, Adewale HB. (2009) Long-term effects of environmental endocrine disruptors on reproductive physiology and behavior. *Front Behav Neurosci.* 29(3):10. DOI: 10.3389/neuro.08.010.2009.
[399] Bruning J (2021) Innovation and Ignorance: How Innovation Funding Cultures Disincentivise Endocrine Disruption Research. Thesis. University of Auckland. Master of Arts, Sociology (research). https://researchspace.auckland.ac.nz/handle/2292/57929
[400] Kahn LG, Philippat C, Nakayama SF. et al. (2020) Endocrine-disrupting chemicals: implications for human health. *The Lancet Diabetes & Endocrinology,* 8(8):703 - 718

may be a highly sensitive time for the programming of fat storage due to permanent effects on gene expression and adipose tissue differentiation. [401]

Importantly, these environmental, nutritional, inflammatory, endocrine and epigenetic influences are not isolated. They frequently dovetail: nutritional insufficiency can heighten vulnerability to environmental toxins; endocrine-disrupting exposures can intensify metabolic instability; and chronic inflammation can magnify epigenetic and neurological impacts. Together, these interacting factors may precipitate or exacerbate both metabolic and mental-health conditions. This integrative view does not imply that mental health challenges are solely genetic, chemical or psychological; rather, they often emerge from a complex constellation of interrelated processes.

As a consequence, the simple excess energy balance theory of obesity is being replaced by a more complex approaches which encompass carbohydrate and obesogen exposure across the lifespan and which increasingly appear to provoke excess consumption. Personal and dietary changes including shifts away from refined and ultraprocessed foods consequently reduce exposure to obesogenic substances. [402]

Socially and environmentally mediated exposures are now recognised as far more influential drivers of chronic disease than discrete genetic traits. Genome-wide association studies (GWAS) demonstrate that genetic contributions to affect, behaviour and cognition arise from thousands of variants, each exerting extremely small effects. [403] In response to the limited explanatory power of genetics alone, the concept of the 'exposome' was proposed in 2005, the '*lifetime environmental exposures (physical, chemical, biological, psychosocial, social, behavioral, etc.) from conception to death*' was defined. This was further refined and expanded to the:

classification of the exposome into three overlapping domains that can change over time: the internal exposome (e.g., aging, oxidative stress, metabolism, gut microbiome), the general external exposome (e.g., climate, built environment), and the specific external exposome (e.g., chemical exposure, lifestyle, occupations). [404]

Socially and environmentally mediated exposures are a far greater driver of chronic disease than discrete genetic traits. The genome-wide association (GWA) studies demonstrated that:

'*genetic influences on individual differences in affect, behavior, and cognition are driven by thousands of DNA variants, each with very small effect sizes.*'

In 2005, after recognising the relatively small contribution of genetics to cancer risk, the concept of the human exposome, the '*lifetime environmental exposures (physical, chemical, biological, psychosocial, social, behavioral, etc.) from conception to death*' was defined. This was further refined and expanded to the:

classification of the exposome into three overlapping domains that can change over time: the internal exposome (e.g., aging, oxidative stress, metabolism, gut microbiome), the general external exposome (e.g., climate, built environment), and the specific external exposome (e.g., chemical exposure, lifestyle, occupations). [405]

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[402] Heindel JJ, Lustig RH, Howard S. *et al.* (2021) Obesogens: a unifying theory for the global rise in obesity.
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Industrial, agricultural and urban pollution drives early childhood deaths and promotes gastrointestinal diseases and non-communicable disease in children.[406] Health effects from pesticides,[407] [408] electromagnetic-fields,[409] [410] [411] [412] [413] , plastics,[414] [415] remain relatively unresearched and therefore unknown in New Zealand. General practitioners and clinicians lack clinically approved and funded pathways for testing and funded pathways for research are lacking. Priorities may not reflect real risks. Heavy metal poisoning may cause more heart disease than high cholesterol, but testing for cholesterol is normal while testing for heavy metals is not normal.[416] [417] [418]

People can be exposed to pollutants and toxins via industrial, agricultural, urban and workplace exposures. These exposures can then not only result in chronic illness, but can provoke and amplify poor mental health and neurodegenerative disorders.[419] Heavy metals[420] [421] [422] [423], particulate matter[424] [425], common

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[425] Fonken LK, Xu X, Weil ZM, *et al.* (2011). Air pollution impairs cognition, provokes depressive-like behaviors and alters hippocampal cytokine expression and morphology. *Mol Psychiatry*. 16(10):987-95, 973. DOI: 10.1038/mp.2011.76.

pesticides[426] [427] and household and general use substances have been identified as neurodevelopmental toxicants which increase risk to brains, from preconception onwards. [428]

This facet of health care is largely unrecognised in conventional medical practice. Integrative or functional medical practitioners often have a greater 'toolkit' to address the environmental contributors to poor health. These doctors have elected to pursue continuing professional development (CPD) outside the conventional 'mainstream', to gain a broader appreciation of the drivers of toxicity. This includes further education to support their capacity to evaluate and address the complex interrelationships between diet, digestion, physical and social environmental exposures, genetics and methylation capacity. When presented with complex, chronic conditions, functional medicine practitioners run serum and biomarker screening tests in addition to conventional screening to evaluate genetic variation and methylation capacity, and toxic stressors and work to eliminate toxicity, improve absorption in the digestive tract and enhance nutrition.

This dimension of health care remains only partially recognised within conventional medical practice and health agencies. Integrative and functional medicine practitioners tend to work with a broader clinical

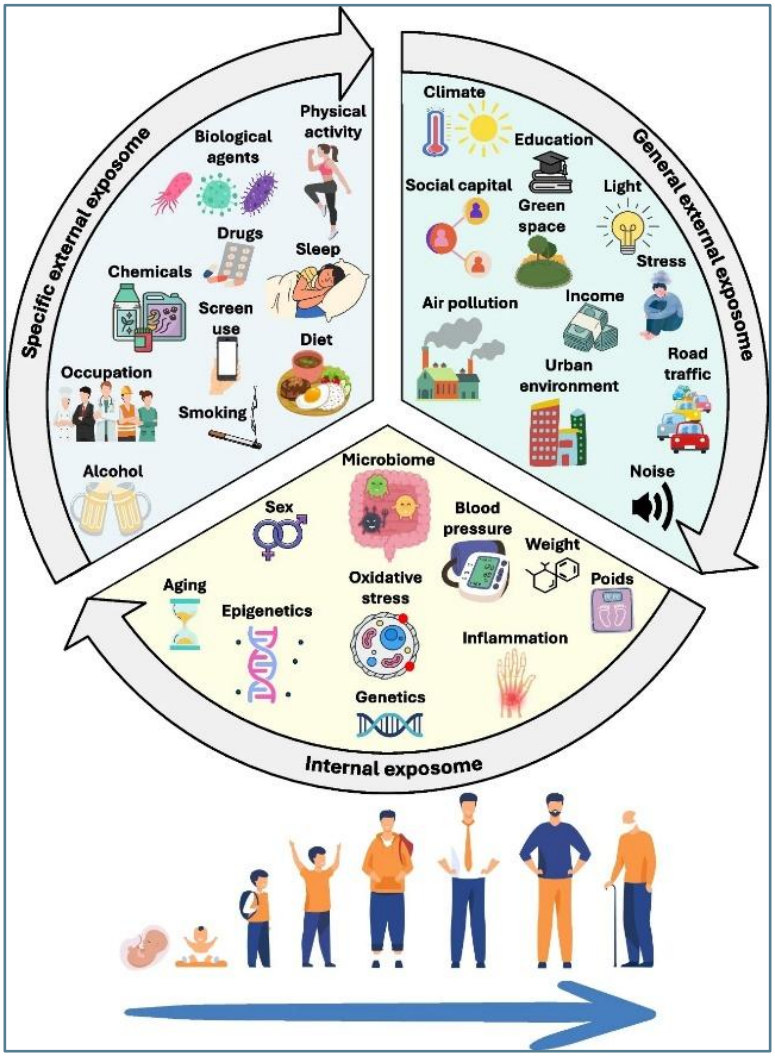

Figure 8. Petit, P., Vuillerme, N. (2025) Global research trends on the human exposome: a bibliometric analysis (2005–2024). *Environ Sci Pollut Res*.

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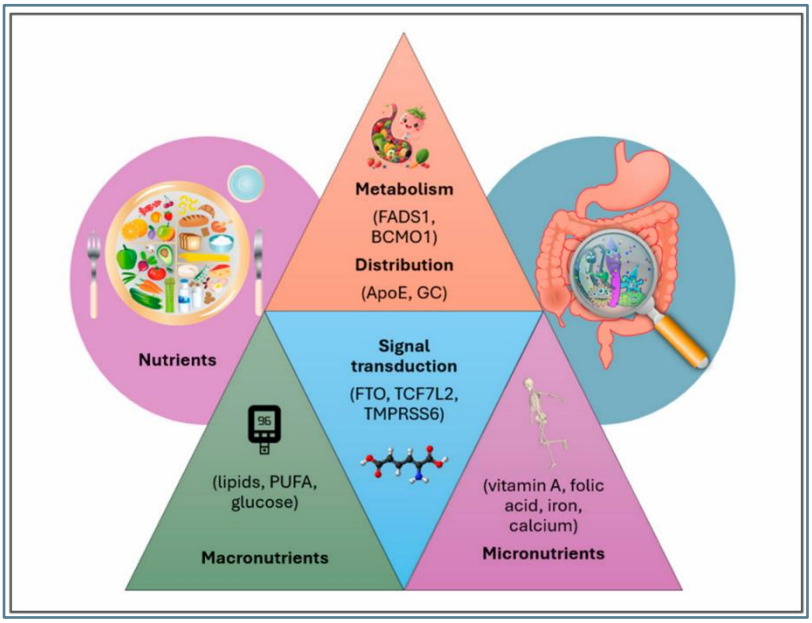
'toolkit' that allows them to engage more directly with environmental contributors to ill health. Many pursue additional continuing professional development outside standard CPD pathways through organisations such as Australasian College of Nutritional and Environmental Medicine, to deepen their understanding of the interrelated drivers of toxicity, metabolic instability and chronic inflammation. This training supports a more integrated evaluation of diet, digestion, physical and social environmental exposures, genetic variation and methylation capacity. When faced with complex, multi-system conditions, functional practitioners commonly use targeted serum and biomarker assessments to identify toxic stressors, address digestive-tract function, and optimise nutritional status.

In New Zealand, however, most functional medicine practitioners are required to send clinical samples overseas for toxicity and biomarker analysis, as no domestic infrastructure exists to provide these services. There is currently no Ministry of Health support or coordinated framework in the research sector to assist clinicians who manage complex chronic or acute presentations that may be initiated or exacerbated by toxic, endocrine, inflammatory or epigenetic environmental exposures.

The practical effect is that both physicians and patients are constrained by cost, and the public system has not provided affordable pathways to support appropriate metabolic or toxicity screening. This produces inequity: individuals on low incomes are often unable to investigate complex or chronic conditions, while those with greater financial means can access comprehensive testing. Although there is a recognised risk that repeated testing may at times be driven by health anxiety or diagnostic uncertainty, resulting in low-value or low-yield investigations, many functional medicine practitioners use a defined, evidence-guided panel of tests that they adjust according to the patient's history, exposures and clinical presentation.

The absence of a national research programme assessing human toxicity patterns has also constrained scientific innovation. As international awareness of environmental exposures, nutrition and metabolic health grows, consumer demand for affordable and accessible biomarker and toxicity-screening technologies is increasing rapidly. Without investment in local research, health practitioners and agencies will be unable to address health inequities over the longer term as these technologies become more sophisticated and affordable.

A recent paper, *Personalized Nutrition in Pediatric Chronic Diseases*, reviewed Omics technologies which can increasingly precisely analyse gene–diet interactions, gut microbiome compositions, and metabolic responses. Multi-omics integration combines microbiome, metabolomics, and genomics diagnostics. The data can play an important role in revealing inter-individual variability in nutrient processing.[429] While there are many challenges, which include concerns about privacy, this interdisciplinary area is an exciting field of development that could support health clinicians with future diagnostics.



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Oral Health Opportunity: Correlates with common metabolic conditions.

> *If one looks at teeth as a window to overall systemic health, an absence of both dental caries and gingival bleeding in the absence of oral hygiene could be regarded as a potentially sensitive marker for an overall healthy diet.*[430]

An increasing weight of evidence suggests that that the modern epidemics of dental caries and periodontal disease are correlates of a broader chronic disease spectrum that includes metabolic syndrome and poor mental health.[431] [432] [433] Oral disease does not occur in isolation; it frequently reflects broader metabolic and inflammatory pressures operating across the body.

As such, the modern oral health epidemic may be primarily mediated by diets high in refined carbohydrates. These foods exert cascading effects on the oral microbiome, local immune responses, epithelial integrity, and the mineralisation of bone and teeth. The cluster of risks that undermine oral health (poor diet, smoking, stress, low socioeconomic status, metabolic dysfunction) are the same modifiable determinants that drive most chronic diseases. The cluster of risks that undermine oral health (poor diet, smoking, stress, low socioeconomic status, metabolic dysfunction) are the same modifiable determinants that drive most chronic diseases. In a landmark paper, Sheiham and Watt argued that '*a collaborative approach is more rational than one that is disease specific'*.[434]

Oral health, like mental health, does not exclusively begin above the neck. The state of the mouth is intricately associated with the health of the human body. The most commonly encountered dental diseases and conditions in New Zealand include tooth decay (dental caries), chalky teeth (molar hypomineralisation), gum disease (periodontal disease), and oral cancer.

Acute risk factors, such as high free-sugar intake and frequent consumption of acidic beverages, directly contribute to dental hard-tissue erosion and the development of dental caries. Dental erosion may also result from certain medications or repeated exposure to gastric acid.

In contrast, poor overall dietary quality drives more complex, *chronic* metabolic responses that act systemically to increase the risk of common dental diseases and conditions. Many factors are implicated in oral health decline, and numerous confounders, oral-hygiene practices, stress, smoking, socioeconomic status, genetics have historically made direct attribution difficult. Yet these interacting factors are themselves influenced, up- or down-regulated, by diet quality. Nutrient-dense diets can buffer genetic vulnerabilities and support stress resilience, whereas inferior diets promote a cascading effect across body systems, increasing susceptibility to inherited conditions, inflammation, and stress-related disorders. Before the widespread availability of processed foods, periodontal disease was recognised as a manifestation of scurvy, caused by inadequate vitamin C intake, illustrating how diet and nutrient status directly affect gum and bone health. [435]

Clinicians in hospitals, dental clinics and general practice now routinely encounter patients with multimorbidity, a pattern that includes elevated risk of tooth decay and periodontal disease. The scientific

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[430] Hujoel PP, Lindström P. (2017) Nutrition, dental caries and periodontal disease: a narrative review. *J Clin Periodontol* 2017; 44 (Suppl. 18): S79–S84. DOI: 10.1111/jcpe.12672

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literature demonstrates consistent associations between oral disease and both free sugars (added to processed foods and beverages, and naturally present in syrups, honey, fruit concentrates and juices) and rapidly digestible starchy foods.

Persistent exposure to refined carbohydrates, including high-glycaemic starches and ultra-processed foods, is strongly implicated in the aetiology of dental caries, hypomineralisation, periodontal disease and, to some extent, oral cancer. These risks converge around several mechanisms: diets that sustain an environment conducive to bacterial replication; persistent local or systemic inflammation; deficiencies in essential vitamins and minerals; and reduced immune competence. However, these interrelated drivers of dysbiosis, chronic inflammation[436] [437] and poor mineralisation of bone and teeth are rarely examined in combination in landmark oral-health papers (such as Venturelli *et al* (2019)[438]) or reflected in health-promotion materials.

Dental caries arise primarily from sustained exposure to fermentable carbohydrates. Both simple sugars and rapidly digestible starches possess similar capacities to increase caries risk. Historically, populations consuming minimal refined carbohydrates experienced far lower rates of dental caries. [439] [440]

Frequent consumption of refined carbohydrates across the day, including meals and snacks, creates an oral environment conducive to caries, gingivitis (a reversible early-stage gum inflammation), and periodontal disease. Periodontitis is a progressive, destructive condition involving irreversible damage to gum tissue, the periodontal ligament and the alveolar bone. Progression from gingivitis to periodontitis depends on a range of interacting factors, including metabolic health, immune function, smoking, stress, and genetic susceptibility.

Refined starches and their breakdown products lower plaque pH, shift the oral microbiome, and promote bacterial fermentation. Highly processed starches adhere to the tooth surface and are readily metabolised by oral bacteria. Increased sugar intake promotes plaque accumulation and is associated with heightened gingival inflammation, producing a biofilm-induced inflammatory response. Gingivitis can develop even in the presence of good oral hygiene if dietary pressures are persistent.[441] The persistence of refined starch in the mouth promotes dental decay but also sets the scene for the development of gingivitis and/or periodontal disease.[442] [443] A recent (2025) paper reported that increased intake of sugars in study participants resulted in increased plaque accumulation, and was associated with higher levels of gingival inflammation, producing a biofilm-induced inflammatory response on the gingival tissue.[444]

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[436] Mukherjee, M. S., Han, C. Y., Sukumaran, *et al.* (2023). Effect of anti-inflammatory diets on inflammation markers in adult human populations: A systematic review of randomized controlled trials. *Nutrition Reviews,* 81(1), 55–74.
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[438] Peres MA, Macpherson LMD, Weyant RJ, *et al.* (2019). Oral diseases: a global public health challenge. *Lancet*. 394(10194):249-260. DOI: 10.1016/S0140-6736(19)31146-8. Erratum in: Lancet. 2019 Sep 21;394(10203):1010.
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[443] Nyvad B & Takahashi N. (2020). Integrated hypothesis of dental caries and periodontal diseases, Journal of Oral Microbiology, 12:1, 1710953, DOI:10.1080/20002297.2019.1710953
[444] Salim A, Angelova S, Roussev B, et al. (2025). Association between frequency of sugar and protein intake and severity of plaque-induced gingivitis in children. *Turk Arch Pediatr*. 60(3):319-325. DOI: 10.5152/TurkArchPediatr.2025.24166

Ingredients in ultra-processed foods which include sugars, refined starches and refined seed oils, are associated with increased risk for periodontal disease, and early-life exposure may predispose to earlier onset of disease.[445] [446] [447] [448]

Excess intake of refined carbohydrates and ultra-processed foods not only harms oral tissues directly but also displaces nutrient-dense foods that support epithelial integrity, immune function, bone mineralisation and periodontal health.[449] [450] [451] [452] Key mechanisms include:

- **B-vitamins** for cellular metabolism, repair and proliferation
- **Vitamin A** for epithelial health and antioxidant defence
- **Vitamin C** for collagen formation and oxidative-stress protection
- **Vitamin D** for calcium absorption, bone and tooth mineralisation, and prevention of hypomineralisation
- **Minerals** such as calcium and magnesium for bone and tooth structure; iron for preventing ulceration and oxidative stress; zinc for epithelial repair and immune function

Together, these findings indicate that oral health is a sensitive indicator of broader metabolic and nutritional status, and that contemporary diets dominated by refined and ultra-processed foods create conditions that undermine both.

A recent white paper by the New Zealand Dental Association (NZDA), *Roadmap Towards Better Oral Health*[453] highlighted the poor oral health of the New Zealand population and the barriers to adequate dental treatment. The paper reported that in 2023/2024 an estimated 321,000, or 7.4% of the adult population had one or more teeth removed due to decay, abscess, infection or gum disease, while 31,000, or 3.3% of children had one or more teeth removed over the same period. The report highlighted the progressive and severe implications of chronic progressive dental disease, and emphasised that low-income groups and that Pasifika populations were most at risk of deteriorating oral health.

The report recommended expanding care to young adults and the implementation of dental service models to meet the needs of local communities and high-need population groups. The *Roadmap* continued the advocacy of the NZDA in increasing the affordability of oral health related prescriptions and laboratory screening services:

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[445] Iwasaki M, Taylor G W, Moynihan P, *et al.* (2011) Dietary ratio of n-6 ton-3 polyunsaturated fatty acids and periodontaldisease in community-based older Japanese: a3-year follow-up study. *Prostaglandins, Leuko-trienes and Essential Fatty Acids* 85:107–112. DOI: 10.1016/j.plefa.2011.04.002.

[446] Cassiano LS, Peres MA, Motta JVS, *et al.* (2022). Periodontitis Is Associated with Consumption of Processed and Ultra-Processed Foods: Findings from a Population-Based Study. *Nutrients*, 14(18):3735. DOI: 10.3390/nu14183735

[447] Shanmugasundaram, S., Karmakar, S. (2024) Excess dietary sugar and its impact on periodontal inflammation: a narrative review. *BDJ Open* 10:78. DOI:10.1038/s41405-024-00265-w

[448] Coll I, Vallejos D, Estebala P, López-Safont N. (2025). The Relationship Between Processed Food Consumption and Periodontal Disease: Sex Disparities in the Majorcan Adolescent Population. *Life (Basel).* 15(4):580. DOI: 10.3390/life15040580.

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[450] Albalooshy A. (2024). Vitamin D deficiency and chronological hypoplasia with hypomineralisation: a case report. *J Clin Ped Dent.* 48(3):177-181. DOI:10.22514/jocpd.2024.072.

[451] Uwitonze AM, Rahman S, Ojeh N, *et al.* (2020). Oral manifestations of magnesium and vitamin D inadequacy. *J Steroid Biochemistry and Molecular Biology*, 200:105636. DOI: 1 0.1016/j.jsbmb.2020.105636.

[452] Najeeb S, Zafar MS, Khurshid Z. *et al.* (2016). The Role of Nutrition in Periodontal Health: An Update. *Nutrients*. 8, 530; DOI:10.3390/nu8090530.

[453] NZDA (November 2025). Roadmap Towards Better Oral Health. www.roadmap.nzda.org.nz

- That pharmacy charges to patients for prescriptions issued by a dentist should be the same as those for prescriptions issued by a medical practitioner in primary care.
- That patients attending a dentist should have access to funded laboratory services for histology and routine blood tests on the same basis as primary care.

PSGRNZ supports many of the *Roadmap's* positions. The following paragraphs explain why, in our view, the NZDA missed an important opportunity by downplaying the role of diet and nutrition while giving substantial weight to a compound that may pose risks to children's brain health.

The *Roadmap* identifies several risk factors for poor oral health that are shared with other chronic conditions such as diabetes, cardiovascular disease and respiratory disease. The recognised obstacle of excess sugar consumption, tobacco use and alcohol is addressed, however, the *Roadmap* does not discuss the broader role of refined carbohydrates, i.e. the cumulative carbohydrate burden as an underlying driver of tooth decay (dental caries), chalky teeth (molar hypomineralisation), gum disease (periodontal disease) and oral cancer. The *Roadmap* does not devote attention to nutritional determinants of oral health, particularly the roles of essential vitamins and minerals in supporting epithelial integrity, immune function, bone mineralisation and periodontal tissue health. Instead, six pages are dedicated to community water fluoridation and topical fluoridation.

The *Roadmap* does not disclose the ongoing scientific controversy surrounding community water fluoridation or the ethical considerations relating to fluoride exposure in children under ten years of age, a population exquisitely vulnerable to neurodevelopmental toxicants. Recently, a 2024 Cochrane review found only the low-certainty evidence for the potential for water fluoridation to reduce socioeconomic disparities in dental caries. For clinicians committed to achieving the best health outcomes for children, low-certainty evidence raises a reasonable ethical question about cost–benefit trade-offs, particularly given emerging concerns about neurodevelopmental risks. New Zealand has not conducted a risk assessment examining the combined exposure of fluoridated drinking water and fluoride toothpaste in children under ten. At present, no lower safe threshold has been established for these early developmental age groups.[454] The *Roadmap* cited papers by the Prime Minister's Chief Science Adviser and the Royal Society Te Aparangi. These reviews did not conform to any standards for a methods-based risk assessment which are appropriate for the formation of government policy.

The findings published in the U.S. National Toxicology Program monograph[455], an influential document that has intensified international debate regarding the safety of community water fluoridation were also omitted from the report, and evidence that was disclosed in a U.S. court case (2024) [456] which found that the U.S. Environmental Protection Agency had failed to follow their own risk assessment guidelines. [457]

PSGRNZ recognises that, over time, the NZDA will place greater emphasis on the multifactorial dietary and nutritional drivers of poor oral health, including the substantial overlaps with metabolic and mental health. A stronger focus on micronutrients, central to epithelial integrity, immune function, bone mineralisation and periodontal health, would support a more comprehensive, evidence-informed approach to improving oral health outcomes in New Zealand.

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[454] Iheozor-Ejiofor Z, Walsh T, Lewis SR, Riley P, Boyers D, Clarkson JE, Worthington HV, Glenny A-M, O'Malley L. Water fluoridation for the prevention of dental caries. Cochrane Database of Systematic Reviews 2024, Issue 10. Art. No.: CD010856. DOI: 10.1002/14651858.CD010856.pub3.

[455] NTP Monograph on the State of the Science Concerning Fluoride Exposure and Neurodevelopment and Cognition: A Systematic Review. NTP Monograph 08. National Toxicology Program Public Health Service U.S. Department of Health and Human Services. August 2024.

[456] Food & Water Watch Inc et al. *v.* United States Environmental Protection Agency et al. Findings of Fact and Conclusions of Law. Judge E. Chen. Document 445. August 24, 2024.

[457] PSGRNZ (2024). New Zealand Fluoride Timeline. https://PSGRNZ.org.nz/component/jdownloads/send/1-root/152-fluoride-timeline-2024-oct

PART II. GOVERNMENT AGENCIES 'DRAFT OUT' INDIVIDUAL BIOLOGY & MULTIMORBIDITY

6. NEW ZEALAND'S CARBOHYDRATE-RICH GUIDELINES

Part II reviews Ministry of Health and Health New Zealand policies to identify the extent to which nutrition and diet is prioritised across the policy spectrum. The public health role of primordial and primary prevention is outlined and contrasted against secondary and tertiary prevention interventions.

This rapid review of government health policies and white papers reveals that New Zealand's health policies may mention diet or nutrition but consistently lack substantive content reflecting contemporary scientific knowledge and individual risk factors. Contemporary health policies have not translated into tangible outcomes that have lowered the prevalence of poor, and frequently comorbid mental and metabolic health outcomes.

New Zealand's legislation emphasises the role of officials in the protection of health. Ministry of Health entities and officials are granted powers under the Health Act 1956 and the Pae Ora (Healthy Futures) Act 2022. At all times they are required to 'improve, promote and protect public health'.

New Zealand acknowledges that poor nutrition is a modifiable risk factor for poor health. In the

New Zealand's health budget is considerable, but the percentage dedicated for the research to update existing knowledge bases, to reflect new findings in the scientific literature, to ensure that our policies reflect best scientific evidence, is negligible. Funding to support practitioner and community education for diet and nutrition is negligible. The 2025/2026 budget for the Ministry of Health is $31,052,217,000. Of the $31 billion, $15.7 is budgeted for hospital and specialist services, $9.7 billion is budgeted primary, community, public and population health services and $1.77 billion is targeted for pharmaceutical medicines.[458]

The 2025/2026 budget allocated $6 million for the PHA and the NPHS and $95.3 million to public health and population health leadership.:

> *'This category is limited to providing leadership on policy, strategy, regulatory, intelligence, surveillance and monitoring related to public and population health'.*[459]

Current New Zealand dietary guidelines for adults recommend 6 servings of grain foods a day and two servings of fruit a day.[460] Pregnant women are advised to consume diets low in only unsaturated fat and eat at least eight servings of cereals a day[461]. The guidelines for children and young people urge four servings of breads and cereals for preschoolers, five servings for children and six to seven servings of breads and cereals per day for young people.[462] Older people are advised to 'eat plenty of breads and cereals', 3

[458] Treasury. Vote Health. The Estimates of Appropriations 2025/26 - Health Sector B.5 Vol.5 https://budget.govt.nz/budget/pdfs/estimates/v5/est25-v5-health.pdf
[459] [459] Treasury. Vote Health. The Estimates of Appropriations 2025/26 - Health Sector B.5 Vol.5 Page 4.
[460] Health New Zealand (2020) Healthy eating, active living. Food and Activity Advice for Adults from 19-64 years. HE code HE1518 https://cdn.accentuate.io/5313685192862/11408390422661/HE1518-healthy_eating_active_living-web_0_Sep2020-v1603943673274.pdf
[461] Health New Zealand (2023). Safe and Healthy Eating in Pregnancy. HE code HE1805 https://cdn.accentuate.io/5313672904862/11408390422661/1.0-HE1805-Healthy-healthy-for-pregnancy_Aug-2023-(1)-v1695958003663.pdf
[462] Healthy Eating for Young People - HE1230 https://healthed.govt.nz/products/healthy-eating-for-young-people

servings for women and 5 servings for men a day.[463] Cereals are describe as 'the best source of energy for the body.'[464]

The guidelines describe T2DM as:

> *A condition associated with insulin resistance, leading to a relative insulin deficit. It usually develops in adulthood and is caused by lifestyle factors, including obesity. Treatment includes changes to diet, physical activity, weight loss, tablets and/or insulin injections. Sometimes referred to as adult-onset diabetes mellitus or non insulin-dependent diabetes mellitus.*[465]

The dietary guidelines do not describe the association of elevated blood glucose levels with risk for insulin resistance, a diagnosis of T2DM and the strong association of elevated insulin levels with obesity. The guidelines simply describe insulin resistance as:

> '*The reduced sensitivity of cells to insulin.*'[466]

Government guidelines broadly recommending high carbohydrate dietary intakes, do not take into account individual insulin sensitivity, particularly in genetically or metabolically sensitive (e.g. ageing) individuals.

A 2024 Official Information Act request asked two questions regarding carbohydrates and cardiac risk:[467]

> ***Question:*** *Has the Ministry of Health ever reviewed the evidence that partial substitution of carbohydrate with either protein and fats can lower blood pressure, improve lipid levels, reduce estimated cardiovascular risk and reduce pre-diabetes and diabetes incidence?*
>
> ***Response:*** *The Ministry undertook a review of popular diets in 2017 including paleo and very low-carbohydrate diets. The information in this review has now been transferred to Health New Zealand – Te Whatu Ora and can be found on their website at: https://info.health.nz/keeping healthy/popular-diets-review.*
>
> ***Question:*** *Has the Ministry of Health reviewed evidence that current dietary guidelines relating to current recommended levels of breads and cereals in the diet may have potential to increase serum lipids and contribute to the development of pre-diabetes and diabetes? - Pre-schoolers: at least 4 servings - Children: at least 5 servings - Young people: at least 6 servings.*
>
> ***Response:*** *This question was not directly addressed, with Dr Jones instead referring to the 2020 update of current serving sizes which was based on Australian nutrition data from 2013 and earlier.* [468]

The government has not reviewed the potential for the cumulative burden of recommended carbohydrate intakes to drive risk for prediabetes and diabetes, and the potential for this to be amplified by ultraprocessed food intake. In a similar vein, New Zealand has been disinclined to identify, regulate and tax

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[463] Health New Zealand (2021). Eating for Healthy Older People. https://cdn.accentuate.io/5313672052894/11408390422661/HE1145-HealthyEatingOlderPeople-WEB-Apr-21-v1621897274164.pdf HE1145

[464] Health New Zealand (2023). Eating for healthy children from 2-12 years. Code HE1302 https://cdn.accentuate.io/5313671594142/11408390422661/1.1_he1302_healthy_children_apr_2023_web_0-v1685674014232.pdf

[465] Ministry of Health. 2012. Food and Nutrition Guidelines for Healthy Children and Young People (Aged 2–18 years): A background paper. Partial revision February 2015. Wellington: Ministry of Health. P.181.

[466] Ministry of Health. 2012. Food and Nutrition Guidelines for Healthy Children and Young People. P.177

[467] Ministry of Health Official Information Act Request response. September 4, 2024. H2024048401 https://fyi.org.nz/request/27933/response/107134/attach/9/H2024048401%20Response%20Letter.pdf

[468] Health New Zealand (December 2020). New Serving Size Advice. https://www.tewhatuora.govt.nz/assets/Health-services-and-programmes/Nutrition/new-serving-size-advice-dec20-v3.pdf

ultraprocessed foods and sugar.[469] [470] Ministry of Health guidelines for type 2 diabetes mellitus (T2DM) do not recommend low-carbohydrate approaches.[471] The term 'metabolic syndrome' is not recognised by the Ministry of Health.

PSGRNZ's review of government websites and white pages was unable to identify any budgeted area of policy or policy deployment which were directly concerned with the role of diet and nutrition beyond the production of promotional literature. There are no established bioethics publications specifically addressing whether panels should be ethically required to revisit paediatric carbohydrate guidelines in light of rising prediabetes, T2DM risk and the associated comorbidities. Consensus reviews acknowledge a lack of paediatric carbohydrate alternatives, but they stop short of ethical examination.

As we discuss below in chapter 7-9, neither health policy, health agencies nor science policies build in obligations and explicitly fund ongoing research to keep abreast of the evidence on elevated glucose and unstable insulin levels as a driver of chronic disease, the role of nutrition and toxic exposures to inform policy.

Finally, a series of examples are provided that demonstrate how current policies do not work scientifically or practically, underserving both clinicians and patients.

7. HEALTH TARGETS DECOUPLED FROM POLICIES. NO POWER TO STOP RISING DISEASE RATES

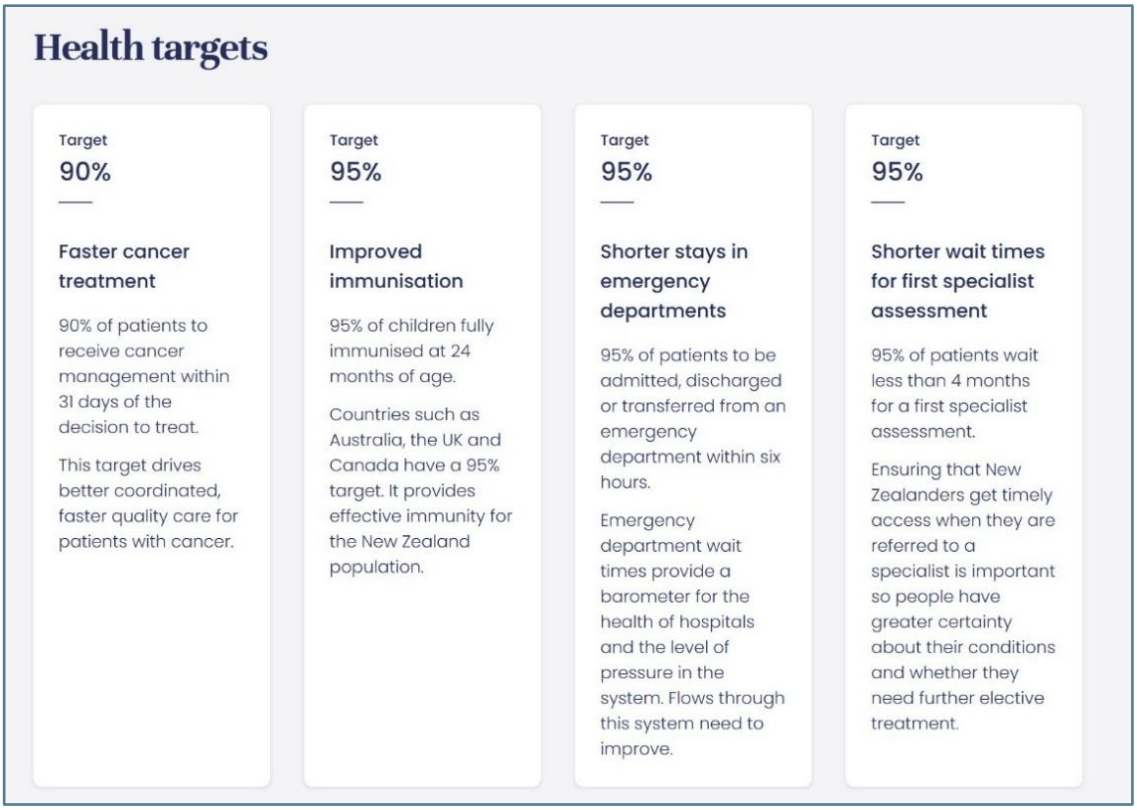

Figure 10. Five Targets for the Health System. https://www.health.govt.nz/statistics-research/system-monitoring/health-targets#Thetargets

[469] Lustig R (2021). Ultraprocessed Food: Addictive, Toxic, and Ready for Regulation. Nutrients 12, 3401; DOI:10.3390/nu12113401

[470] Warhurst L. (October 9, 2019). Jacinda Ardern 'rules out' introduction of sugar tax despite rising numbers of diabetes. *Stuff*. https://www.newshub.co.nz/home/lifestyle/2019/10/jacinda-ardern-rules-out-introduction-of-sugar-tax-despite-rising-numbers-of-diabetes.html

[471] Ministry of Health and New Zealand Society for the Study of Diabetes. Type 2 Diabetes Management Guidelines Healthy eating and weight loss. https://t2dm.nzssd.org.nz/Section-88-Healthy-eating-and-weight-loss

The key *indicators* of New Zealanders' health and wellbeing include self-rated health, life expectancy, mortality rates across the population and maternity measures.[472] In contrast, the *health targets* that are published in the Government Policy Statement on Health 2024-2027 concern Ministry priorities and expectations for service care and delivery in the medical system.[473] No health targets exist for lowering the prevalence and incidence of diabetes, heart disease, the spectrum of problems driven by obesity (including pain and inflammation) and mental illness, in the public health directorate policies.

The targets are decoupled from the indicators, with no chain-of-logic that would support the integrative, health-protective approach that is necessary to sustain health and wellbeing. This policy–indicator misalignment means that targets do little to arrest upstream drivers of symptom clusters that precede diagnosis. Ministry targets instead centre on disease metrics and medical treatment.

This misalignment helps explain why metabolic and mental health burdens continue to rise despite expanded service access. The health targets do nothing to mitigate or stop the upstream drivers of the clusters of symptoms that drive complex illness and that precede a medical diagnosis, and produce declining health, wellbeing and life expectancy. The health targets are unable to address the drivers of chronic illness and multimorbidity and years lost due to illness.[474] [475]

Primordial prevention revolves around building and sustaining a healthy metabolism from the outset to prevent decline. Primordial prevention is absent from the current strategy. Instead, the health targets prioritise medically-focussed secondary and tertiary interventions. This structural misalignment helps explain why metabolic and mental health burdens continue to rise despite expanded service access.

Health targets include shorter stays in emergency departments, and shorter wait times for specialist assessments. Such policies can be presented in parallel with primordial and primary prevention strategies. The health targets focus on faster cancer treatment, yet there are no policies that prevent cancer arising in the first place.

The Ministry of Health maintains a page on multimorbidity, which is drawn from staff presentations at General Practice consultations.[476] The page includes a section on prevention, which includes:

- lifestyle modification (increase physical activity, improve nutrition, smoking cessation, alcohol moderation)
- motivational interviewing
- referrals to community providers for support eg Green Prescriptions.
- smoking cessation, treating hypertension and hypercholesterolemia.

It is not evident that primordial and primary prevention strategies have been clearly distinguished or prioritised by health officials. Current dietary guidelines appear to be treated as the extent of prevention in this domain. The current priority of screening and immunisation may also have, in practice, displaced earlier and more fundamental prevention strategies.

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[472] Ministry of Health. 2024. Health and Independence Report 2023 - Te Pūrongo mō te Hauora me te Tū Motuhake 2023. Wellington: Ministry of Health.
[473] Delivery Plan A summary of the plan to improve healthcare and achieve the Government's priorities. March 2025 – June 2026
[474] Ministry of Health (September 2024). Achieving the Health Targets. High Level Implementation Plans. July 2024 – June 2027. ISBN 978-1-99-106778-4
[475] Minister of Health (July 2024). Government Policy Statement on Health 2024 – 2027. HP9076. ISBN 978-1-991075-77-2 Wellington: Ministry of Health.
[476] Health New Zealand (January 2024). Multimorbidity. https://www.tewhatuora.govt.nz/for-health-professionals/clinical-guidance/diseases-and-conditions/long-term-conditions/management-of-multimorbidity

PUBLIC HEALTH FRAMEWORK

Primordial prevention:

Preventing the upstream social, environmental, cultural and dietary risk factors that disrupt the metabolic function of the individual. Risk factors can include factors that drive low-grade inflammation; dysregulate the endocrine and immune system; overload hepatic–renal clearance; and impair digestive, mitochondrial, cardiovascular, and neurodevelopmental function.

Primary prevention:

Reduce the risk of future disease by identifying and targeting modifiable risk factors in healthy people who are at risk of chronic disease through relevant support programmes. Nutritional and biomarker screening to identify e.g. methylation problems can identify personal vulnerabilities. Programmes may include doctor-patient and clinic-patient support, community support and local government and regional support. Programmes can increase knowledge, skills, and access to healthy food; reduce barriers to exercise and the natural environment; and promote connected communities.

Secondary prevention:

The identification of people with early/asymptomatic disease via the use of screening programmes in order to treat patients early, and ensure health status does not decline further. Programmes currently emphasise medical screening, and nutritional and biomarker screening can be more fully integrated to ensure an integrative approach to support optimum biological health.

Tertiary prevention:

Diagnosis of and treatment of people with the fully developed disease, so as to prevent recurrence and complications. Nutritional frameworks are yet to be adapted into tertiary prevention modalities.

Health Policy: A Prevention Framework That Sidelines Nutrition.

New Zealand health policy documents consistently emphasise central values of wellbeing, equity, and health. ‘Prevention’ in New Zealand health policy refers predominantly to health system interventions such as screening and early treatment. At times prevention refers to the need for a healthy diet. A healthy diet explicitly applies to Ministry of Health dietary guideline information.

‘Health promotion’ in New Zealand draws from the Ottawa Charter, the Bangkok Charter for Health Promotion and Māori models of health.[477] [478] These Charters recognise that a wide range of factors support health: peace, shelter, education, food, income, a stable eco-system, sustainable resources, social justice, and equity.

[477] Health New Zealand. Models of Health. https://www.tewhatuora.govt.nz/health-services-and-programmes/public-health/models-of-health

[478] WHO. The 1st International Conference on Health Promotion, Ottawa, 1986. https://www.who.int/teams/health-promotion/enhanced-wellbeing/first-global-conference

The Charters and corresponding Ministry of Health policy and health promotion literature under-emphasise the outsize role of diet in protecting the gut microbiome and digestive tract, supporting metabolic health to protect people from chronic metabolic diseases and syndromes, including mental illness. Papers outlining central role of nutrition in increasing resilience to the physical and emotional stresses of life, which includes supporting sleep, could not be identified. [479]

The Charters infer that current health information is correct. For example, the Bangkok Charter states:

> *'Health promotion has an established repertoire of proven effective strategies which need to be fully utilised'*.

In a response to an Official Information Act request regarding the Ministry's Eating and Activity Guidelines (EAHs) Dr Nicholas Jones, Director of Public Health at the Public Health Agency confirmed that the primary documents for current guidelines consist of two background papers[480] [481] and the Australian Guide to Healthy Eating.[482] Dr Jones confirmed that the updated advice was 'based on recent large international evidence reviews that: [483]

> *The updated advice in the EAGs is based on recent large international evidence reviews that were used to develop dietary guidelines for Australia, the United States and the Nordic countries. This multi-sourced evidence consistently describes a healthy eating pattern that is high in vegetables and fruit; includes whole grain cereals; low-fat milk products; legumes and nuts; fish and other seafood; and unsaturated oils. This eating pattern is low in processed meats, saturated fat, sodium (salt) and sugar-sweetened foods and drinks. The evidence shows that this way of eating is associated with a lower risk of heart disease, stroke and other health conditions.*
>
> *The EAGs advice is also closely linked to evidence-based recommendations from the World Health Organization and the World Cancer Research Fund. The link between saturated fat consumption, blood cholesterol levels and heart disease are well established, with evidence building over the past 60 years.*

In 2020 Health New Zealand updated its serving size advice, the first adjustment since 1991, to reflect Australian changes.[484] The Australian National Health and Medical Research Council (NHMRC) updated the Australian Guide to Healthy Eating[485], and this was adjusted to reflect data published in the 2006 Nutrient Reference Values for Australia and New Zealand.[486] These reference values as this paper discusses elsewhere, are based on the blood levels of healthy people from the 1980s- to the early 2000s.

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[479] Sierra P. Feeney et al. (2025) Sleep loss is a metabolic disorder. *Sci.Signal.* 18,eadp9358. DOI:10.1126/scisignal.adp9358

[480] Ministry of Health. 2012. Food and Nutrition Guidelines for Healthy Children and Young People (Aged 2–18 years): A background paper. Partial revision February 2015. Wellington: Ministry of Health. HP 5480. https://www.health.govt.nz/system/files/2012-08/food-nutrition-guidelines-healthy-children-young-people-background-paper-feb15-v2.pdf

[481] Ministry of Health. 2013. Food and Nutrition Guidelines for Healthy Older People: A background paper. Wellington: Ministry of Health. HP 5574. https://www.health.govt.nz/system/files/2011-11/food-nutrition-guidelines-healthy-older-people-background-paper-v2.pdf

[482] Ministry of Health Official Information Act Request response. September 4, 2024. H2024048401 https://fyi.org.nz/request/27933/response/107134/attach/9/H2024048401%20Response%20Letter.pdf

[483] Ministry of Health Official Information Act Request response. September 4, 2024. H2024048401

[484] Health New Zealand (December 2020). New Serving Size Advice. https://www.tewhatuora.govt.nz/assets/Health-services-and-programmes/Nutrition/new-serving-size-advice-dec20-v3.pdf

[485] NHMRC. 2013. Australian Dietary Guidelines. Canberra: National Health and Medical Research Council. URL: www.eatforhealth.gov.au/sites/default/files/files/the_guidelines/n55_australian_dietary_guidelines.pdf

[486] NHMRC. 2006. Nutrient Reference Values for Australia and New Zealand including Recommended Dietary Intakes. Canberra: National Health and Medical Research Council; Wellington: Ministry of Health.

The Health Promotion Directorate oversees public health campaigns; health promotion programmes and develops educational resources and publications. Nutrition and dietary information are based on Ministry of Health dietary guidelines which are then translated into promotional flyers and published on the HealthEd Resource database.[487]

Where food is mentioned in policy and health promotion literature, people are encouraged to consume more fruit and vegetables and consume less sugar. The scientific basis that high carbohydrate diets play a substantial role in increasing blood glucose levels and promote insulin resistance and inflammation is well established. However, New Zealand dietary guidelines do not communicate that cumulative dietary carbohydrate intakes elevate blood glucose and triglyceride levels and increase risk for insulin resistance. The government does not recommend paleo, detox or very low (e.g. ketogenic) diets, but does not show evidence of recently having undertaken reviews of the scientific literature to support this position.[488]

As we discussed in chapter 2, official recommendations that revolve around the reduction of saturated fat increasingly appear to be misdirected. The promotional PDFs discuss the benefits of all dietary categories with the exception of fat, and the guidelines recommend low-fat dairy products, and that fat is cut off meat. Although fat is an essential macronutrient, fat is regarded as a high-risk food. Children are advised to only consume foods high in fat, sugar or salt less than once a week.

Increasing evidence suggests that good quality dietary fats support metabolic including cardiac health.[489] Healthy minimally processed dietary fats play an important role in satiety, particularly for people reducing their fat to carbohydrate ratio, aiming to reverse a prediabetic or T2DM diagnosis.[490] [491] [492]

Important nuances in dietary intake are not addressed. This paper focusses on the challenge of refined, ultraprocessed food, yet, for example, official documents do not discern between the health potential of wholefood diets high in saturated foods but low in refined ingredients, versus diets that contain a high proportion of processed and refined products that might also include saturated fats.[493] [494]

There is no advice concerning the role of vitamins and minerals in preventing chronic and infectious disease (including respiratory illnesses). Despite the role of dietary nutrition in epigenetic, hormonal, neurotransmitter and immune regulation, and in moderating inflammation and oxidative stress, dietary nutrition is not prioritised in key health policies. Relatedly, guidance in the promotional flyers for stress, depression and anxiety does not educate or discuss the role of food and nutrition in contributing to improved or impaired mental health.[495]

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[487] Healthed. Healthy Eating. https://healthed.govt.nz/collections/topic-healthy-eating

[488] Health New Zealand (July 2025). Popular diets review. https://info.health.nz/keeping-healthy/eating-well/popular-diets-review

[489] Wu JH, Micha R & Mozaffarian D. Dietary fats and cardiometabolic disease: mechanisms and effects on risk factors and outcomes. *Nat Rev Cardiol* 16:581–601 (2019). DOI: 10.1038/s41569-019-0206-1

[490] Masood W, Annamaraju P, Khan Suheb MZ, et al. (June 2023). Ketogenic Diet. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK499830/

[491] Dashti HM, Mathew TC, Hussein T, *et al* (2004) Long-term effects of a ketogenic diet in obese patients. *Exp Clin Cardiol*. 2004 Fall;9(3):200-5. PMID: 19641727; PMCID: PMC2716748.

[492] Kelly, T, Unwin, D, Finucane, F. (2020). Low-Carbohydrate Diets in the Management of Obesity and Type 2 Diabetes: A Review from Clinicians Using the Approach in Practice. *Int. J. Environ. Res. Public Health* 2020, 17, 2557. DOI: 10.3390/ijerph17072557

[493] Hendriksen RB, van der Gaag EJ (2022). Effect of a dietary intervention including minimal and unprocessed foods, high in natural saturated fats, on the lipid profile of children, pooled evidence from randomized controlled trials and a cohort study. PLOS ONE 17(1): e0261446. https://doi.org/10.1371/journal.pone.0261446

[494] Astrup, A, Magkos, F, Bier, D. *et al*. (2020) Saturated Fats and Health: A Reassessment and Proposal for Food-Based Recommendations: JACC State-of-the-Art Review. *JACC.* 76 (7) 844–857. DOI: 10.1016/j.jacc.2020.05.077

[495] Health New Zealand (2025). There is a way through. A guide for people experiencing stress, depression and anxiety. HE2570. https://healthed.govt.nz/cdn/shop/files/HE2570_There_is_a_way_through_booklet_JAN_25-WEB_d4285d44-ddac-4dc8-af38-2b30d5d3432e.pdf?

Nutrition: The Missing Pillar in New Zealand's Health Policy and Prevention Strategy.

The Government Policy Statement on Health (July 2024)[496]and the New Zealand Health Plan (August 2025)[497] [498] do not carry substantive policy content that provides pathways for health agencies to assess and address dietary inadequacies and nutritional deficiencies. The Statement notes:

> *The Government is particularly focused on accelerating action to address five non communicable diseases: cancer, cardiovascular disease, respiratory disease, diabetes and poor mental health. Together, these conditions account for around 80% of deaths from non-communicable diseases in New Zealand and considerable health loss experienced by New Zealanders.*
>
> *Improved prevention of these non-communicable diseases will be achieved through addressing five modifiable risk factors: alcohol, tobacco, poor nutrition, physical inactivity, and adverse social and environmental factors.* [499]

The action, or target, of 'poor nutrition', which directly relates to nutrition and diet, is the Policy Statement aim to increase the:

> *Percentage of people eating the recommended daily intake of vegetables and fruit (five or more servings of vegetables, and two or more servings of fruit).*[500]

This insubstantial lever cannot address the increasing non-communicable disease burden. This aim drafts out and downplays the role of non-carbohydrate macronutrient groups in supporting health, micronutrients in supporting health and the problem of refined food intakes.

When the policy language consistently fails to prioritise dietary and nutritional drivers, officials are unlikely to launch into related policy development. More opaque outcomes might improve health outcomes in the long term, but direct policy focusing on diet and nutrition might improve health outcomes more swiftly.

> *Social determinants and environmental factors such as education, employment, income, housing, transport and climate account for the majority of health loss, but when strengthened, these same factors also provide a significant opportunity to improve health outcomes.* [501]

The Policy Statement lists seven key health strategies over the next 5-10 years in providing the direction to guide health entities in protecting, promoting, and improving specific health outcomes. As with the overarching document, these key policy papers neglect or downplay the role of diet and nutrition, effectively amplifying the silence around the dietary drivers of New Zealand's burden of disease.:

1. **Pae Tū: Hauora Māori Strategy (2023):** No mention of diet, nutrition or diabetes. Wellbeing mentioned 81 times, equity mentioned 40 times, health mentioned 628 times.
2. **Whakamaua: Māori Health Action Plan 2020–2025 (2020):** No mention of diet or nutrition. Diabetes mentioned 5 times, regarding an action plan to prevent and manage gout and diabetes. Wellbeing mentioned 61 times, equity mentioned 54 times, health mentioned 760 times.

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[496] Minister of Health. 2024. Government Policy Statement on Health 2024 – 2027. https://www.health.govt.nz/system/files/2024-06/government-policy-statement-on-health-2024-2027-v4.pdf
[497] Health New Zealand (August 2025). New Zealand Health Plan | Te Pae Waenga 2024-2027. Wellington: Health New Zealand. https://www.tewhatuora.govt.nz/assets/Publications/New-Zealand-Health-Plan/New-Zealand-Health-Plan-Te-Pae-Waenga.pdf
[498] Health New Zealand (March 2025). Delivery Plan. A summary of the plan to improve healthcare and achieve the Government's priorities March 2025 – June 2026. ISBN 978-1-991139-27-6 (online)
[499] Minister of Health. 2024. Government Policy Statement on Health 2024 – 2027. Page 4.
[500] Minister of Health. 2024. Government Policy Statement on Health 2024 – 2027. Page 48.
[501] Minister of Health. 2024. Government Policy Statement on Health 2024 – 2027. Page 8. https://www.health.govt.nz/system/files/2024-06/government-policy-statement-on-health-2024-2027-v4.pdf

3. **New Zealand Health Strategy (2023):** Dietary risk factors (mentioned once), and notes (once) need for better access to healthy food and nutrition. Refers to the work of Healthy Families NZ. Wellbeing mentioned 104 times and equity mentioned 32 times.
4. **Te Mana Ola: The Pacific Health Strategy (2023):** Notes Pasifika people are three times more likely to have diabetes than European. Mentions dietary risk factors. This policy recommends an expansion of healthy school lunches and a food reformulation target work programme to reduce sodium and sugar in processed food.
5. **Health of Disabled People Strategy (2023):** Report mentions higher rates of heart disease, diabetes, respiratory disease and mental health experienced by disabled people. No mention of diet or nutrition. Wellbeing mentioned 129 times, equity mentioned 12 times, health mentioned 780 times.
6. **Women's Health Strategy (2023):** Mentions poor nutrition and that women spend more of their life in poorer health. Priorities 1-3 focus on health system support and pregnancy care. Priority 4 'living well and ageing well' concerns prevention and early intervention but does not mention diet or nutrition.
7. **Rural Health Strategy (2023):** No mention of diet or nutrition. One case study discusses a poor diet and diabetes. Wellbeing is mentioned 51 times, equity 28 times, and health 770 times.

The policies repeatedly stress prevention, however, consistent with the Policy Statement on Health, these papers predominantly view health promotion as timely access to health services including screening and immunisation services.

The seven health strategies frequently refer to chronic illness being driven by the broader social determinants of disease, and acknowledge that fast-food businesses will cluster geographically near low-income communities.

New Zealand's relatively recent mental wellbeing plan does not address the role of nutrition in supporting brain health, despite the *He Ara Oranga Report of the Government Inquiry into Mental Health and Addiction* (2018) consistently acknowledging associations between poor nutritional status and poor mental health outcomes. The Inquiry was informed by more than 5,200 public submissions and over 400 engagement meetings with communities, clinicians, and sector stakeholders. [502]

'He Ara Oranga translates as Pathways to Wellness'

The Inquiry was therefore explicitly tasked with examining systemic pathways that support mental wellbeing across the life course. However, despite repeated recognition of nutrition as a contributing factor, the Inquiry did not recommend the allocation of public resources to systematically evaluate the role of diet and nutrition in brain health by age, sex, developmental stage, or multimorbidity status.

As a consequence, no policy mandate exists that might address nutrition and embed nutrition and dietary health as a core consideration in subsequent policy development. Neither the ten-year strategy *Kia Manawanui Aotearoa: the Long-term pathway to mental wellbeing'*,[503] nor a recent update[504], includes a coherent policy framework for integrating nutrition into mental health service design, prevention strategies, or therapeutic pathways.

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[502] He Ara Oranga, Report of the Government Inquiry into Mental Health and Addiction. Published in November 2018 by the Government Inquiry into Mental Health and Addiction 978-0-9941245-2-4 (print) https://mentalhealth.inquiry.govt.nz/__data/assets/pdf_file/0024/20868/he-ara-oranga.pdf

[503] Ministry of Health. 2021. Kia Manawanui Aotearoa: Long-term pathway to mental wellbeing. Wellington: Ministry of Health. https://www.health.govt.nz/system/files/2021-08/kia-manawanui-aotearoa-companion-document-sep21.pdf

[504] Ministry of Health. 2023. Kia Manawanui Aotearoa: Update on implementation of a mental wellbeing approach. Wellington: Ministry of Health.

Policies that highlight the problem of insufficiency, suggest ways to identify insufficiency, and which tangibly increase access to nourishing food do not exist in the strategies. Some policies have wish-lists and most policies hope for people to eat healthier diets, however healthy diets are aligned with Ministry of Health guideline recommendations which do not address optimum nutritional status.

PSGRNZ could locate only one only consistently funded programme by the Ministry of Health and Health New Zealand that is directly related to improving dietary nutrition and health in New Zealand communities. Healthy Families NZ commenced in 2014. It has received $10 million annually to provide resources to encourage 'community-up' leadership and collaboration to leverage activities which support health and equity. The programme has worked across ten locations, primarily Māori communities, with the aim of preventing the rise of chronic disease.[505]

Healthy Families NZ adopted a 'six conditions of systems change' approach (aimed at shifting conditions which hold a problem in place[506]) coupled with Kaupapa Māori and mātauranga Māori to drive purpose and impact in local communities. Projects supported by Health Families NZ include the Papatoetoe Food Hub initiative and a Tupu Tahi Whangaroa Growing Together Initiative.[507]

The programme is funded by the Ministry of Health. However, this initiative is not mentioned in Ministry Annual Reports, nor is the allocated funding referred to in the past two Vote Health appropriations. It is not known if funding will extend after 2026.

It is unlikely that other similar projects are funded from the Ministry of Health's $31 billion budget, other than this $10 million per annum project, that increase access in local communities to high quality wholefoods and which support nutrition and dietary education (including cooking education).

8. HEALTH, RESEARCH & ACADEMIC SECTOR: NO PATHWAYS FOR KNOWLEDGE.

Under the Health Act 1956, the Director of Public Health is appointed by the Director-General and is required to advise the Director-General of personal health and regulatory matters relating to public health.[508] The Health Act 1956 also provides powers to the Director of Public Health to (after consulting with the Director-General) advise the Minister on any matter, and report to the Minister on any matter relating to public health.[509]

Three entities are primarily responsible for public health in New Zealand.

A. The Public Health Advisory Committee (PHAC) is an independent expert advisory committee established under section 93 of the Pae Ora (Healthy Futures) Act 2022, to provide public-facing and evidence-based public health advice to Ministers, the Public Health Agency, and Health New Zealand –Te Whatu Ora. Neither the PHAC terms of reference nor the PHAC Position Statement specify any role for nutrition or diet in public health.[510] [511]

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[505] Matheson A, Wehipeihana N, Gray R, *et al*. (2022). Community-up system change for health and wellbeing Healthy Families NZ Summative Evaluation Report 2022. Te Whatu Ora — Health New Zealand. Wellington.
[506] Kania J, Kramer M, & Senge P, 2018. The Water of Systems Change. FSG. https://inspiringcommunities.org.nz/wp-content/uploads/2019/04/The-Water-of-Systems-Change-FSG-2018.pdf
[507] Healthy Families NZ. 10 Years of Impact. Reshaping our Systems for A Healthier Aotearoa New Zealand. https://www.healthyfamiliesnz.org/_files/ugd/44d27c_bb65d9af249949bc9d114e432cc26b0e.pdf
[508] Health Act 1956. S3B. https://www.legislation.govt.nz/act/public/1956/0065/latest/whole.html#DLM306476
[509] Health Act 1956. S3C.
[510] PHAC (May 2024). Terms of Reference. https://www.health.govt.nz/system/files/2024-05/final_tor_for_phac.pdf
[511] PHAC (Sept 2023) Position statement on Equity, Te Tiriti o Waitangi, and Māori Health. https://www.health.govt.nz/system/files/2024-05/phac-te-tiriti-equity-statement-sep23.pdf

B. The Public Health Agency (PHA) is a division of the Ministry of Health established under section 3 of the Pae Ora (Healthy Futures) Act 2022. The PHA *'sits at the heart of the Ministry of Health and leads the Ministry's work on public health and population health'* and is the most important agency for providing advice relating to regulations, to either the Director-General of Health or the Minister. The PHA is required to provide systems leadership across the public health sector and advise the Director-General on matters relating to public health, including –

   (i) personal health matters relating to public health; and
   (ii) regulatory and strategic matters relating to public health.[512]

   The PHAs work-scope includes: policy, strategy, regulation, intelligence, surveillance and monitoring.[513] The PHA's founding document, Pou Whirinaki,[514] mentions 'equity' 29 times, but does not mention diet or nutrition. The document states its key functions:

   'The PHA will lead all public health and population health strategy, policy, regulatory, intelligence, surveillance, knowledge and monitoring functions.'

C. The National Public Health Service (NPHS) is charged with the delivery of public health services, 'the work on the ground'[515], working 'alongside communities to deliver national, regional and local programmes for achieving pae ora'.

'Outside the Work Programme': Dietary Nutrition's Impact on Metabolic and Mental Health.

Dietary nutrition is a low priority issue across the Ministry of Health and within its sub-agencies. This was confirmed by Official Information Act (OIA) requests undertaken in 2024. Sub-agency priorities reflect overarching directives with entities adopting a sit and hold consensus positions, rather than engaging in active enquiry to assess new information.

The Public Health Association of New Zealand lacks policy addressing diet, nutrition and metabolic and mental health, despite being responsible for nutrition strategy and updating standards. Information on diet and nutrition mirrors the legacy content on Ministry of Health websites.[516]

A later OIA request confirmed that neither the head of the Public Health Agency nor the top directors-general of the Ministry of Health were briefed on the relationships between dietary nutrition and mental health, the problem of multimorbidity and metabolic syndrome. Work that could assess the relationship of diet with poor mental health is outside the work programme of the Agency.[517] The OIA request revealed that nutrition, diet and the regulatory levels (including recommended and safe levels) of nutrients, including vitamins and minerals, are issues that were all apparently outside the workstreams of Ministry of Health directorates. The PHA's work programme does not include any undertaking to understand the nutritional or dietary drivers of many common diseases. [518]

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[512] Pae Ora (Healthy Futures) Act 2022. S.3.
[513] Ministry of Health. https://www.health.govt.nz/about-us/organisation-and-leadership/public-health-agency/about-the-public-health-agency
[514] New Zealand Government (2022). Te Pou Hauora Tūmatanui The Public Health Agency. Pou Whirinaki Strategic Intent and High-level Operating Model design. https://www.health.govt.nz/system/files/2024-05/pou-whirinaki-nov22.pdf
[515] Beehive Press Release (July 4, 2022). New Public Health services to improve health for all Kiwis https://www.beehive.govt.nz/release/new-public-health-services-improve-health-all-kiwis
[516] Public Health Association of New Zealand. https://www.pha.org.nz/
[517] Ministry of Health (February 13, 2024). Official Information Act Request. H2023033847 https://fyi.org.nz/request/25086/response/96405/attach/6/H2023033847%20Response.pdf
[518] Bruning JR (March 12, 2024) The Silent Shame of Health Institutions. https://brownstone.org/articles/the-silent-shame-of-health-institutions/

The PHA's work programme reflects the political priorities of sequential governments, and prioritises immunisation, climate change, recognised addictions, fluoridation, and emergency preparedness.

The gap between policy and real-world health risk for the New Zealand population may be observed in the absence of references to metabolic syndrome in official policy. Rates of metabolic syndrome are unprecedented, and metabolic syndrome is framed as a high-risk state, due to the association of the syndrome with increased heart disease, T2DM and mortality risk.[519] [520] [521] [522] Yet in 2024, metabolic syndrome was not formally recognised by the Ministry of Health. Instead, the Ministry of Health viewed the cluster of symptoms characterised by central obesity, dyslipidaemia, hypertension and insulin resistance as either:

> *'considered discretely or as part of a broader cardiovascular disease risk calculation. Cardiovascular risk is calculated based on multiple risk factors, set out in the following guidance: Cardiovascular Disease Risk Assessment and Management for Primary Care."*[523]

Officials are unfamiliar with the term metabolic syndrome, and appear to be unaware of the role of carbohydrate-mediated elevated blood glucose (HbA1c) and elevated triglyceride levels as a precursor to metabolic syndrome and heart disease risk. The Ministry acknowledges that macrovascular (cardiovascular) risk increases 'substantially' with worsening glycaemic control.: [524]

> *Microvascular risk increases exponentially and macrovascular risk increases substantially with worsening glycaemic control. The greatest individual benefit is achieved with a reduction in higher levels of HbA1c.*
>
> *Worsening glycaemic control increases microvascular risk (retinopathy, nephropathy and neuropathy) more substantially than macrovascular risk.*
>
> *Microvascular disease risk increases progressively from HbA1c levels above the threshold for diagnosed diabetes (48–50 mmol/mol).* [525]

However, insulin therapy (a tertiary prevention strategy) is the first line treatment for T2DM. Primordial and primary prevention strategies wherein the Ministry of Health and its associated agencies explicitly recommend a reduction in refined carbohydrates and starchy-foods for the prevention of T2DM and heart health, do not exist. New Zealand's cardiovascular risk management guidelines for clinicians consequently lack a pathway for clinicians to support patients to lower carbohydrate intake to reduce blood glucose and improve glycaemic control for heart health. The current frameworks suggest that if clinicians actively

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[519] Saklayen MG (2018). The Global Epidemic of the Metabolic Syndrome. *Curr Hypertens Rep.* 20(2):12. DOI: 10.1007/s11906-018-0812-z

[520] Leong KSW, Jayasinghe TN, Wilson BC, *et al.* (2020). High prevalence of undiagnosed comorbidities among adolescents with obesity. *Sci Rep.* 10(1):20101. DOI: 10.1038/s41598-020-76921-6.

[521] Barthow C, Pullon S, Weatherall M, Krebs J. (2022) They're sicker than we think: an exploratory study profiling the cardio-metabolic health in a sample of adults with pre-diabetes in Aotearoa New Zealand. *Journal of Primary Health Care* 14:221–228. DOI:10.1071/HC22068

[522] Oh, J.E., Oh, J.A., Demopoulos, M. *et al.* (2023). Impact of the metabolic syndrome on prevalence and survival in motor neuron disease: a retrospective case series. *Metab Brain Dis* 38:2583–2589. DOI:10.1007/s11011-023-01296-2

[523] Ministry of Health (Dec 19, 2023). Official Information Act Request. H2023033845 https://fyi.org.nz/request/25087-moh-evidence-research-and-innovation-deputy-director-general-is-dean-rutherford-briefed-driving-policy-on-metabolic-syndrome-mental-health-ultraprocessed-food-nutritional-deficiency#incoming-94650

[524] Ministry of Health (2018). Cardiovascular Disease Risk Assessment and Management for Primary Care. Wellington: Ministry of Health. Page 18. https://www.tewhatuora.govt.nz/assets/Publications/Cardiovascular-Publications/cardiovascular-disease-risk-assessment-management-primary-care-feb18-v4_0.pdf

[525] Ministry of Health (2018). Cardiovascular Disease Risk Assessment and Management for Primary Care. Wellington: Ministry of Health. https://www.tewhatuora.govt.nz/assets/Publications/Cardiovascular-Publications/cardiovascular-disease-risk-assessment-management-primary-care-feb18-v4_0.pdf

recommended lower carbohydrate intakes than dietary guidelines recommend, this deviation from guideline recommendations could expose clinicians to investigation.

Government agencies have consistently advanced a consensus perspective, known as the diet-heart hypothesis. This posits that saturated fat and cholesterol are a primary driver of heart disease, and underlies the dietary recommendations for low-fat diets high in cereals, fruits and vegetables. Many government-funded trials have failed to provide evidence to support this hypothesis, and government reviews have failed to incorporate and discuss contradictory data, reflecting long-standing bias towards this issue.[526] [527]

'Lifestyle modification' and a 'healthy diet' is referred to in government documents, however, dietary management of lipids appears to exclusively concern reducing dietary saturated fat intake to 'reduce low-density lipoprotein cholesterol (LDL-C) while maintaining or increasing high-density lipoprotein cholesterol (HDL-C)'.[528] Authorities tend to represent lifestyle interventions that induce remission of T2DM as occurring following (downstream) weight reduction, rather than via (upstream) carbohydrate intake reduction.[529]

Insulin was recognised as a fattening substance nearly a century ago. The combination of current carbohydrate-rich dietary guidelines and conventional insulin therapy may act in concert to increase population-level rates of obesity. Historically, naturally produced insulin (from the pancreas) resolved the issue of high blood lipids and enabled fat storage for seasons when food supplies diminished.

Chronically elevated levels of insulin in the blood (hyperinsulinemia), metabolic stress, excessive oxidation and inflammation affect all body systems, from the mitochondria, to the human brain.[530] [531] The common problem of 'brain fog' may be driven by elevated glucose and brain inflammation, arising from the over-consumption of carbohydrates.[532] New Zealand's poor educational performance and poor productivity may be associated with average blood glucose and insulin levels as much as any other factor.

Systemic Neglect of Nutrition and Environmental Health Research.

'Public health' refers to the health of all the people of New Zealand, or a population group, community, or section of people within New Zealand, while personal health means the health of an individual.[533] Individuals form the units of any sub-population, and insights from cellular research and single-case studies can inform policy, particularly when they align with evidence from larger cohorts and population-level research.

Therefore, for public health to reflect the state of scientific knowledge, researchers and policy-makers require adequate resourcing to investigate across the full spectrum of scientific evidence: including cellular and mechanistic studies, case reports, cohort studies, and population-level epidemiological research.

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[526] Teicholz N. (2023) Major NIH Trials Whose Results Not Considered for the US Dietary Guidelines. https://www.scribd.com/document/886754036/Major-NIH-Trials-Whose-Results-Not-Considered-for-the-US-Dietary-Guidelines?

[527] Teicholz N. (2022). A short history of saturated fat: the making and unmaking of a scientific consensus. *Current Opinion in Endocrinology & Diabetes and Obesity* 30(1):65-71, DOI: 10.1097/MED.0000000000000791

[528] BPAC (2018). Cardiovascular disease risk assessment in primary care: managing lipids https://bpac.org.nz/2018/docs/lipids.pdf

[529] BPAC (2021). Type 2 diabetes management toolbox: from lifestyle to insulin. https://bpac.org.nz/2021/diabetes-management.aspx

[530] Pomytkin I, & Pinelis V. (2020). Brain Insulin Resistance: Focus on Insulin Receptor-Mitochondria Interactions. *Life* 11,262. DOI:10.3390/life11030262

[531] Palmer C. (2022). Brain Energy. *Benbella Books*.

[532] Kikkawa Y, Kuwabara S, Misawa S. et al (2005). The acute effects of glycemic control on nerve conduction in human diabetics. *Clinical Neurophysiology.* 116(2)270-7274. DOI: 10.1016/j.clinph.2004.08.011

[533] Pae Ora (Healthy Futures) Act 2022. Interpretation: Public Health, Personal Health.

Position papers that rely predominantly on epidemiological evidence to justify dietary guideline recommendations may be inadequate for contemporary policy needs. Epidemiological studies are often dated, may omit critical biological or environmental determinants, can conflate effects from multiple exposures, and frequently mask differences in vulnerability across sub-populations. Without incorporating new mechanistic findings and insights from emerging scientific fields, policies weighted heavily toward epidemiology risk being anchored in incomplete or obsolete evidence. As a result, the underlying data may be poorly suited to assessing risk or tailoring benefits at the individual level, which is essential for promoting personal health.

The outcomes of interventions undertaken by clinics represent an opportunity that has yet to be fully pursued. Early-adopter clinics can serve as important bellwethers for policy. Findings generated through clinical audits can highlight how the health status of patients with complex conditions changes over the course of an intervention. The U.K.-based Norwood National Health Service (NHS) Surgery serves as a case in point, having published data tracking outcomes from low-carbohydrate interventions in patients with prediabetes and type 2 diabetes mellitus (T2DM). In a 2024 *BMJ* editorial, Unwin urged[534]:

> *I would encourage clinicians working in the field of nutrition to think about publishing audit. In that way we could have vastly more information on interventions that are working well in the 'real world'. Audit is the way to answer interesting questions about your clinic. For example, do you know the average weight loss, blood pressure improvement or other important clinical metrics achieved by your service?*

Outcomes published by the Unwin clinic (discussed below) are yet to be considered by New Zealand health agencies.

Funding for New Zealand–based basic health research falls well short of the scale of the country's metabolic and mental-illness burden and is insufficient to meaningfully confront the growing challenge of multimorbidity. As this paper was being drafted, the Health Research Council of New Zealand (HRC) communicated that the government was reducing the HRC's budget by 10% and that this was being 'repurposed' to a New Zealand Institute for Advanced Technology which is designed to pursue 'breakthrough technologies like AI, quantum computing, and synthetic biology'. The HRC was advised that from July 2026 there will be a reduction of $590,000 in the HRC's annual operating budget, and from July 2028 there will be a reduction of $11,487,000 (approximately 10 percent) in the annual investment budget (i.e. the Health Research Fund).[535]

The Ministry of Business, Innovation and Employment (MBIE) controls science and research policy and funding in New Zealand. There is no dedicated, long-term research stream to support high-quality basic and applied public-good research in dietary nutrition, metabolic health, or mental health. In an earlier paper on science-system reform, PSGRNZ outlined how New Zealand's science system was quietly placed under MBIE's control through secondary legislation. Members of Parliament did not have the opportunity to vote on the shift from an independent science agency to one primarily oriented toward promoting business and economic growth.

Two years later, in 2015, the National Statement of Science Investment 2015–2025 established a policy platform that, for the next decade, prioritised projects that were 'innovative' (with patents treated as a proxy for economic growth) and 'excellent' (favouring narrow, discipline-specific expertise). This approach effectively concentrated decision-making for large research investments within small groups of politically aligned actors. At the same time, multidisciplinary public-good research, particularly in environmental

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[534] Unwin D. (2024). Reducing overweight and obesity; so how are we doing? BMJ Nutrition, *Prevention & Health*.
[535] Reti S, (July 2025). Beehive Press Release. New Advanced Tech Institute backs science sector. https://www.beehive.govt.nz/release/new-advanced-tech-institute-backs-science-sector

science, agriculture, and public health was relegated to short-term, tightly managed, and modestly funded programmes.[536] [537]

Gravida, the institution established to undertake broader research into infant and child health, was defunded and disestablished.[538] The Centre for Public Health Research (CPHR) has long operated with insufficient funding. Limited pathways for environmental and occupational disease research have left CPHR with narrow income streams for essential public-good work.

At first glance, the Public Health Agency (PHA) appears positioned to lead nutrition- and environment-related research, evaluation and education. However, neither its founding documents nor its work programme contain any indication of an intent to review nutrition, investigate toxic chemical exposures, or develop related research capacity.

Since the publication of the National Statement of Science Investment 2015–2025, MBIE has not developed policies or ring-fenced funding to support long-term public-good health research. This gap includes the absence of dedicated funding for dietary, nutritional and environmental-health research or for systems that would allow such findings to be integrated into government policy.

Many research questions that warrant investigation fall outside current funding pathways because they diverge from Ministry of Health guidelines or lack an 'innovation' framing. These include long-term studies examining dietary changes across multimorbid conditions such as cardiovascular disease, T2DM, cancer and inflammatory disorders. Critical questions remain unexplored, such as the metabolic effects of diets high in unprocessed fats and low in carbohydrates (under 30% of daily energy), the comparative risks of chemically refined seed oils versus minimally processed dietary fats, and the roles of fat, protein and fibre in satiety and between-meal hunger regulation.[539] [540]

Given the strong association between metabolic syndrome and psychiatric diagnoses, research into how advances in dietary and metabolic health can improve mental wellbeing and resilience is also needed. Likewise, New Zealand lacks any research stream capable of examining the health risks arising from expanding medical prescribing practices, including the extent, impact and effects of polypharmacy among younger populations.[541] [542] [543]

Despite global advances in understanding nutrition, metabolism and environmental exposures, New Zealand has not built the research infrastructure necessary to detect, characterise or respond to environmental determinants of human health risk.

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[536] PSGRNZ (2025). When powerful agencies hijack democratic systems. Part II: The case of science system reform. Page 5-6. Bruning, J.R.. Physicians & Scientists for Global Responsibility New Zealand. April 2025. ISBN 978-1-0670678-1-6. https://PSGRNZ.org.nz/component/jdownloads/send/1-root/174-science-system-reforms-hijack-democracy

[537] MBIE (2015) National Statement of Science Investment 2015-2025. Ministry of Business, Innovation and Employment. https://www.mbie.govt.nz/science-and-technology/science-and-innovation/funding-information-and opportunities/national-statement-of-science-investment

[538] Gravida. https://gravida.org.nz/

[539] Gharby S. (2022). Refining Vegetable Oils: Chemical and Physical Refining. *The Scientific World Journal*. 2022:6627013, DOI: 10.1155/2022/6627013

[540] Johnstone AM, Horgan GW, Murison SD, *et al.* (2008). Effects of a high-protein ketogenic diet on hunger, appetite, and weight loss in obese men feeding ad libitum. *Am J Clin Nutr.* 87(1):44-55. DOI: 10.1093/ajcn/87.1.44.

[541] Health New Zealand. Understanding Depression https://www.depression.org.nz/understanding-mental-health/understanding-depression

[542] Health New Zealand. Depression. https://info.health.nz/mental-health/mental-health-conditions/understanding-depression

[543] Health New Zealand. Mental Health. https://info.health.nz/mental-health/mental-health-conditions

Barriers to Clinical Testing for Toxicity, Genetic Variants and Nutrient Status.

Globally, research unpicking the environmental, including nutritional drivers of human health and disease has increased at pace.[544] Research on human health, diet, and toxic chemical and heavy metal exposures is short term and rare in New Zealand. This spills into limited access to screening technologies for clinical practitioners.

PSGRNZ have identified five systemic barriers that constrain what can realistically be known in clinical practice, limiting the capacity of clinicians and patients to act together in the patient's best interests. Government commitments to equity ring hollow in this context, as these constraints fall most heavily on low-socioeconomic groups and other population sub-groups who are more likely to encounter toxic exposures, carry particular genetic variants, or live with persistent nutrient insufficiencies.

1. *Public-sector clinicians lack structured pathways to screen for toxic exposures whether dietary, occupational, or urban.* It is uncommon for clinicians to order tests even when a patient reports or documents exposure to a hazardous substance. Individuals and families who believe that acute or chronic symptoms may be environmentally driven are often forced into private testing. Domestic human-health screening services are limited, and it is, in practice, easier to obtain toxicology testing for livestock than for people.

Clinicians have minimal access to publicly funded diagnostic pathways, and the few that exist are narrow in scope. Although toxic stressors contribute to a continuum of health risks, facilities to measure biomarkers of exposure, assess toxin burdens, or support research programmes that would build a national evidence base have not been developed or funded.

2. *Public-sector clinicians also lack access to basic genomic screening for common gene variants that influence nutrient absorption and metabolic pathways*. Routine testing for variations affecting one-carbon metabolism and methylation capacity, such as the well-characterised MTHFR polymorphisms, is unavailable. Impairments in these pathways can reduce nutrient utilisation and contribute to downstream health effects, yet no accessible clinical pathways exist to identify such risks early.

3. *Clinicians may face scrutiny when they attempt to address optimal nutrition*. Routine or repeated testing of nutrient serum levels, e.g. for vitamin D, is sometimes questioned. Moreover, clinicians risk investigation if they recommend intakes above the Ministry of Health's recommended daily intake levels (RDIs), even though RDIs are designed to prevent deficiency, not to identify or achieve optimal physiological levels for health promotion or disease prevention.

4. *Finally, clinicians must use caution when their prescribing or deprescribing practices diverge from government guidelines.* This is the case even when clinical judgement or emerging evidence would support a more individualised approach, as guideline levels focus on deficiency rather than nutrient sufficiency.

Vitamin D is one such example where the health system overstates risk for a basic nutrient while downplaying new scientific evidence. When an OIA request asked for specific nutritional advice for clinicians to support patients on use and dosage of B group vitamins, vitamin C, D, selenium and zinc, the only information that could be supplied were statements on vitamin D and sun exposure.[545]

The Ministry of Health's 33-page Companion Statement on Vitamin D in pregnancy and infancy[546] understates the problem and extent of deficiency in New Zealand, providing a new recommendation that was not

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[544] Petit, P., Vuillerme, N. (2025). Global research trends on the human exposome: a bibliometric analysis.
[545] Ministry of Health (February 13, 2025). Official Information Act request. H2025059327. https://fyi.org.nz/request/29768/response/117956/attach/7/H2025059327%20Response.pdf
[546] Ministry of Health (2024). Companion Statement on Vitamin D and Sun Exposure in Pregnancy & Infancy. https://www.tewhatuora.govt.nz/assets/Publications/Environmental-health/companion-statement-vitamin-d-sun-exposure-pregnancy-infancy-nz.pdf

based on evidence in the scientific literature but on levels recommended by North American and European governments.[547]

The Statement does not engage with the wider biological mechanisms that depend on adequate vitamin D status, instead maintaining a narrow focus on bone-related symptoms. It offers no assessment of the levels required to achieve or correct deficiency. It also overlooks the substantial body of evidence linking subclinical insufficiency with increased risks of cancer, impaired immune function, cardiovascular disease, and metabolic syndrome.[548] The paper recommends a much lower daily intake than is expressed in multiple studies, and neglects to review studies recommending higher intake.

The safety of vitamin D is well established, yet the Statement does not review evidence that higher doses of vitamin D are safe, and did not suggest that the upper limits of 100 µg/4000 IU per day were associated with any detrimental health risk. The paper does not recommend that people are tested for vitamin D insufficiency (unless they experience the symptoms relating to skeletal/bone health).

Current legislation may be obstructing progress in this area. In October 2025, Medsafe released its decision on Professor Julia Rucklidge's application to raise permitted daily dose limit of vitamin D from 25 micrograms to 75 micrograms (3000 IU), and to allow naturally occurring lithium (at levels commonly present in food ingredients) to be included in nutrient supplements.[549] The Committee argued that her application did not sufficiently demonstrate the benefits of these changes while the risks were not clearly categorised (as these are safe levels of nutrients well-recognised by the body, risks which would be applicable to a medical drug, do not exist).

When PSGRNZ sought clarification, Rucklidge explained that she had been caught in a regulatory Catch-22 within the Medicines Act 1981: the moment any therapeutic benefit is described, the nutrient is automatically reclassified as a medicine, triggering full pharmaceutical regulation and making it impossible to justify nutritional use without simultaneously invoking regulatory medicine controls.

Vitamin D is one case. It highlights the challenges medical doctors face in advising patients on optimum nutrition levels that are required to prevent chronic deficiency. Implicit in this is sufficient information to ensure that the patient has a tangible choice and that the patient, is *fully informed*. The example of vitamin D levels suggests that chronic deficiency and broader nutritional benefits remain broadly unrecognised by government officials, or not regarded as inside their work programme. These knowledge-gaps bend policy frameworks to favour the prescribing of medical treatments.

Public Health Blindspot: The Addictive Potential of Industrial Ultraprocessed Foods.

In September 2024 the Public Health Communication Centre (PHCC) released an info-paper on ultraprocessed foods. The paper downplayed the health harms and did not discuss the issue that ultraprocessed foods are consistently higher in insulin-spiking carbohydrates, designed for addictive potential, higher in industrially refined chemically synthesised ingredients and associated with food addiction (dependency behaviours relating to sugar and processed foods). [550] [551] The 2024 paper by the

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[547] NHMRC (2017) Nutrient Reference Values for Australia and New Zealand. Vitamin D page 122

[548] Bouillon R and Carmeliet G (2018). Vitamin D insufficiency: Definition, diagnosis and management. *Best Practice & Research Clinical Endocrinology & Metabolism*. *32*(5)669-684. DOI: 10.1016/j.beem.2018.09.014

[549] Medsafe (October 30, 2025). Minutes for the 74th meeting of the Medicines Classification Committee held at 133 Molesworth Street, Wellington on 23 July 2025. https://www.medsafe.govt.nz/profs/class/Minutes/2021-2025/74mccMin23July2025.htm

[550] Unwin J, Delon C, Giæver H, *et al*. (2022) Low-carbohydrate and psychoeducational programs show promise for the treatment of ultra-processed food addiction. *Front. Psychiatry.*

[551] Cleghorn C, Egli V, Shields E. *et al* (Sept 2024). Debate on ultra-processed foods shouldn't derail good dietary advice. Public Health Expert Briefing (ISSN 2816-1203) https://www.phcc.org.nz/briefing/debate-ultra-processed-foods-shouldnt-derail-good-dietary-advice

PHCC may have been the first time the health sector mentioned ultraprocessed foods on a government-associated website.

Health Coalition Aotearoa's (HCA) Food Policy Expert Group is an umbrella group which includes public health scientists from New Zealand universities, is yet to confront these issues, focussing more on free sugars and 'healthy food'.[552] [553] The food and nutrition strategy recommended by the HCA is based on the recommendations of the Public Health Advisory Committee (PHAC) and their work harmonises with the PHCC. The PHAC's recommendations, documented in the *Rebalancing our food system* (2024) report drew attention to the 'out of balance food system' and discussed the need for a 20% levy on sugary drinks, the challenge of unhealthy food environments and food insecurity. The report did discuss dietary guideline recommendation, nor discuss the addictive potential of junk and ultraprocessed foods.[554]

Approximately 70% of packaged food sold in New Zealand supermarkets is ultraprocessed.[555] Yet, despite the weight of evidence linking these products to adverse health outcomes, neither the Ministry of Health nor Health New Zealand (Te Whatu Ora) meaningfully address ultraprocessed foods in their public material, and the current nutrition guidelines do not mention them at all. Limited research pathways, combined with structural barriers to undertaking independent nutrition research, further constrain knowledge development and narrow the scope for informed policy-making. Without contemporary scientific literacy within the Ministry of Health, and a fluency with the potential of these foods to promote a spectrum of symptom (multimorbidity) it is difficult to see how complex policy approaches could emerge that would shift current burden-of-disease trajectories.

It is similarly unlikely that policy will restrict the advertising of ultraprocessed foods, advertising that disproportionately influences vulnerable groups[556] or support territorial and local authorities to address 'food swamps' (areas with a high-density of establishments selling high-calorie fast food and junk food, relative to healthier food options)[557], or the uneven distribution of 'food islands' where poorer suburbs have lower levels of choice, less vital food aesthetics, and less healthy foods than in wealthier suburbs.[558]

Data presented in Chapter 4 indicate that many people struggle to limit their consumption of ultraprocessed foods, consistent with evidence that these products are engineered to be hyper-palatable and behaviourally reinforcing. Standard supermarket bread and many common cereals, which contain industrially synthesised ingredients and added sugars, fall squarely within this category.

Food addiction remains unaddressed by the Ministry of Health, and it is improbable that policy leadership on this issue will emerge from within the Ministry. Clinicians who recommend low-carbohydrate diets, that contradict existing guidelines or who advise higher-than-RDI micronutrient intakes risk being challenged by

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[552] Health Coalition Aotearoa (2024). Unhealthy Food. https://www.healthcoalition.org.nz/unhealthy-food/
[553] HCA (November 2023) Briefing to the Incoming Parliament Health Coalition Aotearoa Policy Briefing 2023 – 2026. https://www.healthcoalition.org.nz/wp-content/uploads/2024/04/HCA-BIP-2023-2026-FINAL-1.pdf
[554] Public Health Advisory Committee. 2024. Rebalancing our food system. Wellington: Ministry of Health.
[555] University of Auckland (2019). Fallen stars: most of our packaged food is ultra-processed, unhealthy. https://www.auckland.ac.nz/en/news/2019/08/17/fallen-stars-most-packaged-food-is-ultra-processed-unhealthy.html
[556] Gearhardt AN, Yokum S, Harris JL, Epstein LH & Lumeng JC. (2020). Neural response to fast food commercials in adolescents predicts intake. *The American journal of clinical nutrition,* 111(3):493-502.
[557] Cooksey-Stowers K, Schwartz MB, Brownell KD (2017). Food Swamps Predict Obesity Rates Better Than Food Deserts in the United States. *Int. J. Environ. Res. Public Health* 2017, 14:1366; DOI:10.3390/ijerph14111366
[558] McLellan G. (2018). Auckland's Uneven Food Landscapes. A dissertation submitted in partial fulfilment of the requirement for the degree of BSc(Honours) in Geography 2018. University of Auckland.

health authorities, even when doing so aligns with emerging evidence or the clinical needs of individual patients. [559] [560]

Global Blindspot: When Health Guidance Focusses more on Climate than on Nutrient Sufficiency.

In 2019 the EAT–Lancet Commission defined food-group ranges for a 'healthy' diet and proposed the share of planetary boundaries attributable to food systems as part of a broader agenda for 'food systems transformation'. [561] The Commission's claims about what constitutes a healthy and sustainable diet were immediately met with criticism, particularly regarding nutrient sufficiency for specific population groups, including children, young people, and women of child-bearing age. In 2025 the original analysis was updated to 'expand its scope and strengthen its evidence base', with the authors asserting that the revised framework 'positions justice as both a goal and a driving force for a food-systems transformation'. [562]

Although widely celebrated, the EAT–Lancet dietary proposal does not meaningfully examine the micronutrient and neurobiological requirements of children and young adults. [563] The brain is the most nutrient-intensive organ in childhood and adolescence, yet neither the 2019 nor the 2025 paper assesses age-specific dietary needs to support optimal neurodevelopment and mental health. The recommended reduction in animal-source protein is not accompanied by an evaluation of developmental requirements for essential amino acids, omega-3 fatty acids, or B vitamins. These nutrients play well-established roles in neurodevelopment: B-vitamin-dependent methylation pathways shape brain architecture; amino acids underpin neurotransmitter synthesis; omega-3 fatty acids influence neuronal membrane function; and iron, zinc and iodine are critical for cognitive performance.

A substantial group of scholars has highlighted flaws in the EAT–Lancet model and cautioned that issues of nutrient adequacy, cultural acceptability and affordability are yet to be integrated. [564] [565] [566] Reported shortfalls include:

- Women of reproductive age: deficient in vitamin B12, calcium, iron, and zinc. [567]
- All people aged 6-65 years: deficient in Vitamin A, Vitamin D, Calcium, Iodine, and Selenium. [568] [569]
- Underestimating the more complex benefits of nutrient dense whole foods.

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[559] Dyńka D, Rodzeń Ł, Rodzeń M, Pacholak-Klimas A. *et al.* (2025) Ketogenic Diets for Body Weight Loss: A Comparison with Other Diets. *Nutrients* 2025, 17:965. DOI:10.3390/nu17060965

[560] Neuman V, Plachy L, Drnkova L. *et al.* (2024) Low-carbohydrate diet in children and young people with type 1 diabetes: A randomized controlled trial with cross-over design. *Diabetes Research and Clinical Practice*, 217:111844. DOI: 10.1016/j.diabres.2024.111844.

[561] Willett W, Rockström J, Loken B, et al. Food in the Anthropocene: the EAT–Lancet Commission on healthy diets from sustainable food systems. Lancet 2019; 393: 447–92.

[562] Rockström J, Thilsted SH, Willett WC *et al. (2025).* The EAT–Lancet Commission on healthy, sustainable, and just food systems. *The Lancet*, 5 DOI: 10.1016/S0140-6736(25)01201-2

[563] Venegas Hargous C, Orellana L, Strugnell C. *et al*. (2023). Adapting the Planetary Health Diet Index for children and adolescents. *Int J Behav Nutr Phys Act* 20:146. DOI:10.1186/s12966-023-01516-z

[564] Ortenzi F, McAuliffe GA, Leroy F *et al.* (2023). Can we estimate the impact of small targeted dietary changes on human health and environmental sustainability? *Environmental Impact Assessment Review*, 102:107222. DOI: 10.1016/j.eiar.2023.107222

[565] Zagmutt, FJ. Pouzou JG, Ortenzi F *et al.* (2025). The EAT-Lancet 2025 Report: Half A Decade Later, Key Methodological Issues Remain (November 03, 2025). Preprint published. http://dx.doi.org/10.2139/ssrn.5718962

[566] Leroy F, Ederer P, Lee MRF, Pulina G. (2025). The Systemantics of Meat in Dietary Policy Making, or How to Professionally Fail at Understanding the Complexities of Nourishment. *Meat and Muscle Biology* 9(1): 20155, 1–25. DOI:10.22175/mmb.20155

[567] Beal T, Ortenzi F, Fanzo J. (2023). Estimated micronutrient shortfalls of the EAT–Lancet planetary health diet. *The Lancet Planetary Health*. 7: e233–7.

[568] Lassen AD, Christensen LM, Trolle E. (2020). Development of a Danish adapted healthy plant-based diet based on the EAT-Lancet reference diet. *Nutrients,* 12(3):738. DOI:10.3390/nu12030738.

[569] Nicol K, Nugent AP, Woodside JV. et al. Eating within planetary boundaries - a cross-country analysis of iodine provision from the EAT-Lancet diet. *npj Sci Food* 9, 252. DOI: 10.1038/s41538-025-00612-7

The Commission recommended iron supplementation to meet the needs of adolescent girls but did not address the broader supplementation that would be required to ensure optimal brain health should children or young adults adopt the proposed diet.

The Commission also does not address the historical and cultural role of animal products in providing efficient access to key nutrients that support mental health, nor the practical pressures faced by families in which caregivers work full-time and may have limited capacity to prepare nutrient-dense vegetarian meals. Substituting meat with highly refined ultra-processed protein products, often containing additives designed to enhance palatability, may not represent an optimal dietary strategy.

PSGRNZ adopts a more nuanced perspective, recognising that conventional climate-focused sustainability literature seldom addresses the increased agrichemical use associated with horticultural use and large-scale cropping, including heavy herbicide use of glyphosate on Roundup Ready crops.[570] [571]. Sustainability can be more broadly interpreted to include the beneficial impact of well managed (free-range) livestock production for human and planetary health, including an appreciation of the role animals play in sustaining soil health through the fertilisation of soil, and a traditional key role of animals in terrain and/or climatic environments that are unsuitable for cropping.[572] [573]

A recent paper by Leroy et al. (2025) proposes a shift in language from 'healthy diets' to an emphasis on adequate nourishment. Many foods considered 'healthy' may, in practice, be low in nutritional density. Leroy et al. present a perspective that integrates animal-sourced foods without negating the value of plant-based foods, and they emphasise that certain forms of food processing can support health. The authors highlight the distinction between nutrient density and energy density, noting that ultra-processed foods may be high in calories yet typically *'do not provide the nutrient richness and biochemical complexity of less processed foods, even when fortified with micronutrients.'*[574]

Leroy et al. weave the priority of nutrient density into a flexible framework that recognises cultural context and the regional realities of agricultural systems.

> *While consciously refraining from formulating more specific guidance than what is provided by cultural and physiological 'nutritional wisdom', we contend that self-selection of dominantly nutrient-dense and satiating foods, which are ideally of a mostly minimally and moderately processed nature … maximizes dietary flexibility according to personal needs and preferences within a broad yet optimal domain for adequate nourishment.* [575]

As this paper and our forthcoming work demonstrate, nutrients essential for biological health and with long histories of safe use appear increasingly over-regulated. Officials remain silent on the micronutrient levels required for optimal health, particularly during vulnerable developmental periods. They have declined to examine evidence that challenges the status quo, placing the burden entirely on applicants to persuade regulators that change is warranted. This default approach is a legacy framework designed for corporate

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[570] Navarro J, Hadjikakou M Ridoutt B. *et al* (2021). Pesticide Toxicity Hazard of Agriculture: Regional and Commodity Hotspots in Australia. 55:2;1290-1300 DOI: 10.1021/acs.est.0c05717

[571] Myers, JP, Antoniou MN, Blumberg B. *et al*. (2016). Concerns over use of glyphosate-based herbicides and risks associated with exposures: a consensus statement. *Environ Health* 15,19. DOI:10.1186/s12940-016-0117-0

[572] Macheka L, Kanter R, Lawrence M, Dernini S, Naja F, Oenema S. (2025). Sustainable diets: where from and where to? *J Nutr Sci.* 14:e78. DOI: 10.1017/jns.2025.10049.

[573] Fleming A, Provenza FD, Leroy F, *et al.* (2025). Connecting plant, animal, and human health using untargeted metabolomics, Journal of Animal Science, 103:skaf254, DOI:10.1093/jas/skaf254

[574] Leroy L, Beal T, de Mûelenaere N, *et al.* (2025). A framework for adequate nourishment: balancing nutrient density and food processing levels within the context of culturally and regionally appropriate diets. *Animal Frontiers.* 15(1): 10–23, DOI:10.1093/af/vfae032

[575] Leroy L, Beal T, de Mûelenaere N, *et al.* (2025). A framework for adequate nourishment.

applications applying for regulatory approvals for new drugs and medical devices; it is ill-suited to the stewardship of nutrients fundamental to human physiology.

Barriers to knowledge and to affordable access to multi-nutrients disproportionately affect under-resourced communities. As evidence for the role of micronutrients in mental health continues to advance, existing legislation, and the persistent reluctance of the Ministry of Health and Medsafe, may ultimately cause more harm than good for people living with common conditions including depression, anxiety, and ADHD.

> *Multimorbidity, the co-occurrence of three or more chronic conditions[576], occurs a decade earlier in deprived communities.[577] [578] The societal cost of multimorbidity is super-additive.[579]*

Official health literature and communications consistently neglect to address the super-additive cost of multimorbidity, which consistently includes poor mental health. The burden of suffering, disproportionately impact poor communities is antithetical to the requirement in the Health Act 1956 to protect health.

Slightly higher levels of a generic vitamin or mineral intended to restore depleted levels, can be conflated as having the same potential toxic risks as a synthetically developed, patented pharmaceutical drug. The nutrient is essential for biological health; the drug mitigates a symptom that has been diagnosed by a clinician. The institutional and regulatory health system architecture, in addition to legislation that privileges medical drug and device authorisations perpetuates a status quo approach. This approach has set aside the contradictions expressed in the scientific literature on diet and nutrition.

The inability to stem the drivers of chronic illness and multimorbidity is good business for drug companies. A recent survey found a maximum of 53 drugs prescribed to one person. [580] This is costly to the NZ public both in expenditure and health outcomes. The Ministry of Health directorates do not evaluate the cost of multimorbidity by age and gender, together with the increasing prevalence of polypharmacy by age and gender. There is little resourcing directed to understanding the extent of adverse effects experienced by age, gender and by drug category, particularly when it comes to psychiatric drugs. The data are effectively buried.

The absence of information, outdated information and a lack of expertise can lead to mistaken beliefs which implicitly and explicitly benefit drug manufacturers. As the case of vitamin D shows, the New Zealand government overstates risk for nutrients with a strong safety profile. In comparison, psychiatric drug risk is rarely addressed in Ministry of Health literature. For example, there are no 33-page government papers the risk of one psychiatric drug and the risks in pregnancy. A search could locate one paper, *The Psychotropic Medications for Mothers and Babies Guidelines*, a 24-page document discussing dozens of drugs and their effects.[581] There were no links to the scientific literature to which would enable the reader to understand relevant issues including the study design and the provenance and age of the trial. The extraordinary attention given to vitamin D, and the relative neglect where a review of dozens of psychiatric drugs was condensed into one 24-page paper, highlights how risks from micronutrients tend to be overstated while risks from drugs tend to be understated in Ministry of Health literature.

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[576] Skou ST, Mair FS, Fortin M. *et al*. (2022). Multimorbidity. *Nat Rev Dis Primers* 8,48.
[577] Head A, Fleming K, Kypridemos C, et al. (2021). Multimorbidity: the case for prevention. *J Epidemiol Community Health* 2021;75:242–244. DOI:10.1136/jech-2020-214301
[578] Skou ST, Mair FS, Fortin M. *et al*. (2022). Multimorbidity. *Nat Rev Dis Primers* 8,48.
[579] Blakely T, Kvizhinadze G, Atkinson J, Dieleman J, Clarke P. (2019). Health system costs for individual and comorbid noncommunicable diseases: An analysis of publicly funded health events from New Zealand. *PLoS Med*.
[580] Leitch S, Dovey SM, Cunningham WK, *et al* (2021). Medication-related harm in New Zealand general practice: a retrospective records review *Br J Gen Pract*. 71 (709): e626-e633. DOI: 10.3399/BJGP.2020.1126
[581] Psychotropic Medications for Mothers and Babies Guidelines. Ref: 2407235 Waitaha Canterbury Specialist Mental Health Service, Guidelines. https://edu.cdhb.health.nz/Hospitals-Services/Health-Professionals/maternity-care-guidelines/Documents/Psychotropic-Medications-for-Mothers-and-Babies.pdf

Patents and trademarks, and the commercial advantages they confer, shape an important part of the wider context. Pharmaceutical patents allow companies to build in margins that cover research, development, and marketing. Officials within Pharmac and Medsafe generally recognise that drug companies conduct controlled trials to generate the data needed to justify an application for entry into the New Zealand market. Yet these trials are typically short, often lasting only weeks to months, while the resulting medications may be prescribed to patients for many years.

In contrast, small and medium sized micronutrient supplement manufacturers struggle to build trademarked protections because of the generic nature of vitamins and minerals and pay the application fees for access to drug markets. Current government frameworks, by failing to address the advantage conferred by the capacity of pharmaceutical and biologic drug manufacturers to patent a medication with margins built in for marketing and drug applications, effectively marginalise and penalise nutrition-based policy.

Dietary approaches: A Survey of the Food/Diet Health NGO Landscape.

Alongside the government health sector, non-government organisations and clinical approaches tend to fall into two distinct categories: one aligns with current dietary guidelines that favour a generous carbohydrate-based intake with reduced intake of fat and animal protein; the other advocates therapeutic carbohydrate reduction and a correspondingly greater emphasis on fat and protein. Both categories strongly support reducing sugar, junk food consumption and consuming whole foods.

Category 1: *Alignment with dietary guidelines, low-fat diets and restricted animal protein:*

Cancer Society: Harmonises with the New Zealand eating and activity guidelines.[582] Emphasises limits of sugar, 'fast foods', red and processed meat and to consume a 'diet rich in wholegrains, fruit, vegetables, nuts and beans (lentils and legumes)'. Protein and fat consumption is downplayed.

Heart Foundation: Emphasises vegetables and fruit. Recommends that sugar, salt, trans fat and junk food are cut back on. Focusses on reducing fat and limiting egg consumption for a healthy heart. Carbohydrate burdens are not discussed.[583]

Diabetes NZ: Diabetes NZ recognises the direct association of starchy and sweet food with high blood glucose. The organisation adopts the conventional approach that a heart attack is most directly affected by the 'amount and type of fat and salt you eat'.[584] Carbohydrate intake recommendations revolve around limiting plate portion size to a quarter of the plate, with daily numbers of servings per day of carbohydrate-based foods in the under 50 group recommended at 6 servings of grains, 3 servings of fruit, one cup of milk/yoghurt. Portions of proteins are limited to 2-3 servings, and fat is severely restricted.

The Helen Clarke Foundation: Recommends sugar taxes and restrictions of advertising of products high in fat, salt and sugar, increased restrictions on unhealthy food outlets near schools, increased funding for and improvements in nutrition access for schools, hospitals, the defence force and prisons. Increased investment in weight loss drugs. [585]

Evidence Based Eating NZ: Promotes Whole Food Plant-Based (WFPB) which is vegetarian and does not include added oil. A search could not identify reviews of the scientific literature to identify nutrient requirements by age and gender, including for deficient populations.

Category 2: *Low-carbohydrate and ketogenic approaches, support for increased focus on fats & proteins:*

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[582] Cancer Society. Healthy Eating. https://www.cancer.org.nz/cancer/reduce-your-risk-of-cancer/healthy-eating/
[583] Heart Foundation. https://www.heartfoundation.org.nz/wellbeing/healthy-eating/eating-for-a-healthy-heart
[584] Diabetes NZ: Diabetes and healthy food choices.
[585] The Helen Clark Foundation. Junk Food and Poor Policy? How weak rules undermine health and economic growth in New Zealand and how to fix it

WholeNZ: Representative group of individuals and organisations supporting a carbohydrate-reduction approach to reversing pre-diabetes and T2diabetes across New Zealand communities.[586]

The Holistic Performance Institute: Online nutrition and health coach training which considers individual physiology and carbohydrate tolerance. Courses include ketogenic and low-carbohydrate approaches.[587]

Reversal NZ: Founded by Dr Glenn Davies, Reversal promotes a carbohydrate reduction approach to reverse and encourages consumption of low-carbohydrate and ketogenic diets as a dietary approach to reverse, and reduce risk for prediabetes and common metabolic syndrome associated conditions.[588]

Aronui/Turuki Healthcare: NZ-based clinics supporting patients with Lifestyle health coaching/ Health Coaching offering a low-carb approach to support the reversal and remission of prediabetes and T2DM.

Prekure: Health Coach programme training where coaches gain fluency across metabolic and mental health, where low carbohydrate diets and behavioural change strategies are emphasised.[589]

Real Healthy Me: Health improvement programme via a packaged set of appointments at 1-2 weekly intervals, with commitment for a total of 6 months. [590]

The Australasian Metabolic Health Society (AMHS): Provides education, training, and support of evidence-based nutritional approaches, including carbohydrate restriction & ketogenic therapies, as a valid therapeutic option or intervention. Its primary purpose is to develop consensus guidelines, provide education and training for health practitioners in Australia, New Zealand and the Asia Pacific region. [591]

The Heart Research Institute: Healthy Eating Hub, a team of university qualified nutritionists and dieticians discuss the implementation of a low-carbohydrate diet.[592]

ADHD New Zealand: Recognises the combinatory role of starchy foods to add to the carbohydrate burden and elevate blood glucose. Emphasises the importance of protein in the diet.[593]

An increasing group of websites with clinician leadership (that could be identified) that discuss low carbohydrate approaches are online. These include the *Low Carb Doctor New Zealand* (Facebook group), *Liz Ford*, *Reverse T2 Diabetes Christchurch Keto New Zealand*, *Low Carb Taranaki, Ketogenic Diet Therapy NZ,* and *Fearless Nutrition*. Other consumer/researcher-developed websites promote low-carbohydrate and ketogenic approaches, including *Deborah Murtagh* and *Keto New Zealand*.

When Cumulative Daily Carbohydrate Intakes are Ignored: The $3 School Lunch Programme.

The historic downplaying of the importance of healthy macronutrient fat and protein groups may have been a factor that enabled the National Government to shift quickly to carbohydrate-rich school lunches, such as sandwiches and wraps, mirroring the typical contents of children's and adolescents' lunch boxes. While many children have a carbohydrate rich lunch, and then return home to a more nourishing vegetable and protein dinner, the targeting of the lunch programme was explicitly designed to mitigate dietary insufficiency in children and adolescents who are much less likely to return home to a nutrient rich meal, and more likely to return to a calorie-dense but low nutrient meal.

In August 2019, the Labour Government announced that 30 primary and intermediate schools would begin receiving free lunches at the start of the 2020 school year. The trial expanded in 2020 to become Ka Ora, Ka

[586] WholeNZ. https://www.wholenz.org/about-us
[587] Holistic Performance Institute. www.holisticperformance.institute
[588] Reversal NZ. https://reversalnz.co.nz/resources/
[589] Prekure. https://prekure.com/
[590] Real Healthy Me. https://www.realhealthyme.com/
[591] Australasian Metabolic Health Society. https://amhs.org.au/about-us/
[592] Heart Research Institute: Going Low Carb. https://www.hri.org.nz/health/nutrition/going-low-carb
[593] ADHD New Zealand. https://www.adhd.org.nz/info/extras/7-nutritional-tips-to-manage-your-adhd

Ako, targeted at the 25% of students living in the lowest socio-economic communities. The plan was to feed 21,000 children in 120 schools by early 2021 as part of the Child and Youth Wellbeing Strategy.[594] By June 2025 this had expanded to feed 242,000 children nationally.[595]

The programme required local providers to supply nourishing meals with a healthy macronutrient ratio, which in practice limited refined carbohydrates and starchy vegetables by ensuring adequate protein and non-starchy vegetable intake.[596]

> *In October 2024, Cabinet announced major budget cuts to Ka Ora, Ka Ako with the funding for Years 7-13 lunches reduced from $6.99 (Years 4-8) and $8.90 (Years 9+) to $3.2 Schools could continue to use internal model providers (where the meals are made at the school or by a local school on the programme) or remain on the iwi/hapu model if the providers could supply the meals at a reduced price of $4. Lunches remained at status quo for schools with Years 0-6 students. The School Lunch Collective, a consortium formed at the time between Compass Group NZ, Gilmours and Libelle Group, were contracted to take over lunch supply to the remaining 124,941 students on the programme.* [597]

In an assessment of lunches provided by the Collective, compared against the national nutrient reference values, the Public Health Communication Centre (PHCC) concluded that:

- *None of the meals met all the Nutrition Standards for all age groups.'*
- *Meals are very low in energy, providing approximately half of the energy expected for a school lunch and 30-40% less than lunches provided under the previous model.*

The $3 cost-effective meals reflect the minimum standards and are predominantly carbohydrate-based.[598] While meat and vegetarian protein is included, the minimum standards are low, 45-75 grams, based on age. Saturated fat is 'red', all meat must have fat trimmed or lean, and vegetable oils are 'green'. Very small quantities (around half a cup) of non-starchy vegetables reflect the established guideline standards.[599]

Where earlier versions of the programme had a broader focus on nourishment and nutrition, the $3 lunch minimum standards are likely to be the supply target for suppliers due to budget restrictions. This shift sidelines a more complex set of aims associated with early food exposure: promoting gastronomy (complex, nutrient-rich foods, including secondary compounds), encouraging seasonal appreciation of foods, fostering dietary diversity, and protecting children from ultra-processed foods and unhealthy eating habits. These aims are central to addressing inequities, as children from wealthier households typically encounter a wider range of foods and develop more varied taste preferences.

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[594] Beehive (August 29, 2019. School lunch programme launched. https://www.beehive.govt.nz/release/school-lunch-programme-launched

[595] Ministry of Education (June 4, 2025). Ka Ora, Ka Ako Pānui. https://www.education.govt.nz/bulletins/ka-ora-ka-ako-panui/27-05-25?

[596] Ministry of Education (March 6, 2025). Nutrition standards for Ka Ora, Ka Ako https://www.education.govt.nz/education-professionals/schools-year-0-13/healthy-school-lunches/nutrition-standards-ka-ora-ka-ako

[597] De Seymour J, Swinburn B, Mackay S. (March 28, 2025). Aotearoa's government-funded school lunches failing nutrition standards. https://www.phcc.org.nz/briefing/aotearoas-government-funded-school-lunches-failing-nutrition-standards

[598] Ministry of Education (Oct 2022). Nutrition Standards for Ka Ora, Ka Ako, Health School Lunch Programme. https://web-assets.education.govt.nz/s3fs-public/2025-03/Nutrition%2520Standards%25202022%5B1%5D.pdf?

[599] Ka Ora Ka Ako (Nov 6, 2025). Cost effective menu and recipes. https://kaorakaako.education.govt.nz/working-together/internal-model-schools--iwi-and-hapu-providers/cost-effective-menu-recipes?

European food policies reflect this broader purpose. French school lunches, for example, are designed not merely to 'feed' children but to provide a public health and educational intervention that helps establish long-term dietary norms and habits that lower barriers to wider ranges of nourishing whole-foods.[600]

One way nutrient-rich school lunches reduce inequities is by mitigating the impact of economic constraints on the development of children's taste preferences. Children often reject unfamiliar foods 8 to 15 times before accepting them; expanding dietary variety increases their openness to new tastes and textures. Low-income parents cannot afford to purchase foods that may be discarded if their child refuses them, and children's taste aversion exerts a strong influence on food purchasing decisions. While children in low-income families may prefer energy-dense, nutrient-poor items, this can reflect intergenerational constraints, parents may have experienced similarly narrow food environments, as well as rational risk-aversion related to food waste.[601]

The default to hyperpalatable, energy dense, nutrient poor foods is a component of the limited control exercised by low-income parents, in making food choices when shopping. This does not necessarily reflect a 'culture of poverty', but rather, the financial limitations of food provisioning.[602]

Victoria University research fellow Kahurangi Dey's evaluation of the initial phase of the Ka Ora, Ka Ako programme (before the budget was reduced to $3) aligns with established literature on the development of taste preferences. Dey noted that children did not immediately accept the healthier lunches containing unfamiliar foods, but that acceptance increased over time.

> *the transition to nutrition resulted in uneaten lunches. Initially some unfamiliar healthy foods are rejected, but over time, children's food preferences change as they become accustomed to healthier food. Within six months, the transition to nutrition for children is typically well under way, with previously unfamiliar foods, like tuna or fresh vegetables, becoming accepted. Uneaten lunches are then no longer the issue they were at the beginning of the programme. The graduated approach, with providers slowly adapting from familiar but less healthy options (red and orange light foods) to healthier but initially unfamiliar items (green light foods) that meet nutrition standards, helps improve uptake and promote Ka Ora Ka Ako as reliable and trustworthy*.[603]

Studies have since noted that children adapted to new and unfamiliar tastes and flavours and broadened children's palate acceptances. Reviews have found that the original *Ka Ora Ka Ako* programme improved mental wellbeing, improved emotional, learning and social functioning, concentration and energy.[604] [605]

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[600] European Commission (2011). School Food Policy Country FactSheets. https://joint-research-centre.ec.europa.eu/system/files/2017-07/jrc-school-food-policy-factsheet-france_en.pdf?

[601] Daniel, C. (2016). Economic constraints on taste formation and the true cost of healthy eating. *Social Science & Medicine* 148:34e41, /10.1016/j.socscimed.2015.11.025.

[602] Fielding-Singh, Priya (2017). A Taste of Inequality: Food's Symbolic Value across the Socioeconomic Spectrum. *Sociological Science,* 4: 424-448. DOI: 10.15195/v4.a17

[603] Dey, KJ. (2025). Health by Stealth, Lifting the Load, and Just Food: The influence of Ka Ora Ka Ako on the lives of whānau. A thesis submitted to the Victoria University of Wellington in partial fulfilment of the requirements for the degree of Doctor of Philosophy.

[604] Vermillion Peirce, P., Jarvis-Child, B., Chu, L., & Lennox, K. (2022). New Zealand healthy school lunches programme: Impact evaluation. Ministry of Education https://apo.org.au/node/321482

[605] Aikman, P. J., & Yates-Pahulu, R. (2023). He kai kei aku ringa. Evaluation of the iwi and hapū social procurement and partnership model, under Ka Ora, Ka Ako | The healthy school lunches programme. Ministry of Education https://web-assets.education.govt.nz/s3fs-public/2024-11/He-Kai-Kei-Ringa_Evaluation-Report-FINAL-.pdf

It must be noted that since 2011, European policies have been adjusted to reflect climate prerogatives following a 2019 EAT-Lancet Commission on Food, Planet and Health that reduce meat protein consumption[606] (updated in 2025[607]).

Health System Architecture Bias to Consistently Favour Medical Interventions.

Government policies and actions can shape the capacity for public-health research in direct, indirect and often subtle ways. These influences affect what politicians, policy-makers, agency officials, practitioners and patients come to know, and shape the cultural and professional attitudes that produce normative perspectives. In doing so, they can implicitly or explicitly steer attention away from certain topic areas. Such influence can occur at any stage of the research process, from priority-setting and funding decisions to publication pathways and guideline development.

These dynamics help explain why many public-health officials in New Zealand remain primarily focused on meeting screening and immunisation targets, and why the broader health system, from government officials to frontline clinicians, lacks a shared language for metabolic illness, mental illness and multimorbidity.

Social science research draws attention to the human and institutional forces that shape how knowledge is produced, recognised and disseminated, revealing how certain forms of evidence gain visibility while others remain marginalised. A recent U.S. review shed light on the positive and negative influence of governments on scientific research trajectories.[608] Senior et al (2025) noted:

> *Importantly, these findings highlight that although it may be intuitive to assume that governments seek evidence that facilitates decisions to improve public health outcomes, other motivations beyond beneficial health outcomes may be present.*

The conclusions of this U.S. review are consistent with findings from a comparable New Zealand study.[609]

MODE OF INFLUENCE	NATURE OF SPECIFIC INFLUENCE REPORTED [610]
Direct	➢ Preventing or delaying dissemination of findings ➢ Changing information availability ➢ Prioritizing most desirable work ➢ Displaying preferences for output(s) above scientific rigour ➢ Framing of findings ➢ Changing staff involved in the project ➢ Diverting focus away from the project ➢ Controlling access to data, resources, funding, contracts, etc.

[606] Willett, W. et al. (2019). Food in the Anthropocene: the EAT-Lancet Commission on healthy diets from sustainable food systems. *Lancet* 393:447–492.
[607] Klapp AL, Wyma N, Alessandrini R, *et al.* (2025). Recommendations to address the shortfalls of the EAT-Lancet planetary health diet from a plant-forward perspective. *Lancet Planet Health*. 9:e23–33. DOI: 10.1016/S2542-5196(24)00305-X
[608] Senior HJ, Teimouri N, Waller M, Capewell S, Cullerton K (2025). How governments influence public health research: a scoping review. *Health Promotion International*, 40:daaf097. DOI: 10.1093/heapro/daaf097
[609] Bruning J (2021) Innovation and Ignorance: How Innovation Funding Cultures Disincentivise Endocrine Disruption Research. Thesis. University of Auckland. Master of Arts, Sociology (research). https://researchspace.auckland.ac.nz/handle/2292/57929
[610] Senior HJ, *et al* (2025). How governments influence public health research. Page. 7

Indirect

- Using bribery
- Threatening job/contract loss
- Promoting the concept of research impact
- Creating fear of reporting undesired findings
- Encouraging research to reach wider policy audiences
- Encouraging the use of specific methodologies, approaches, language, dissemination channels, etc.
- Using IP as a tool to influence others
- Using the credibility of researchers as a divisive tool
- Creating relationships with the researchers to allow more influence on the research

Subtle

- Promoting societal impact through idealism
- Creating an environment where government ideology is shared
- Creating job/career insecurity
- Promoting a research environment where fear is present

The exploration of policy content and research capacity serves an important purpose in elucidating the very human influence on New Zealand's political, legal and regulatory health sector – public health. This section demonstrates how brand-new health targets and mental health policy effectively creates barriers to nutritional advice, effectively smooth access to drug-dispensing, effectively corrupting the principle of informed consent. When clinicians and patients lack an adequate understanding of the role of nutrition in supporting mental health, the range of viable options for decision-making narrows. When nutritional psychiatry remains out of the policy equation, care pathways default prematurely to psychiatric medications which are Pharmac funded, effectively dismissing non-medical interventions (which may not be Pharmac funded and/or not included in training programmes).

Without routine assessment of micronutrient status, metabolic health or dietary patterns, potentially reversible contributors to mental distress remain unidentified. For example, improvements may begin with basic measures, optimising nutrition to support sleep, cortisol regulation or energy balance, tailored by age and gender. When these factors are invisible in the clinical encounter, care pathways may default prematurely to psychiatric medications, not necessarily because they are the most clinically appropriate choice, but because alternative explanations and interventions have not been made available.

Informed consent, as defined in health law and ethical practice, requires that patients receive sufficient information to ensure they are fully informed about their condition, the material risks and expected benefits of proposed treatments, and any reasonable alternatives. It also requires that patients are given an opportunity to consider this information and make a voluntary choice. When nutritional and metabolic factors are neither evaluated nor disclosed, the requirements for informed consent cannot be met in substance. Patients cannot provide meaningful consent to psychiatric medication if they have not been informed that modifiable nutritional deficiencies or dietary interventions may constitute viable, lower-risk alternatives or adjuncts. In such circumstances, informed consent is reduced to a procedural formality: the documentation may be completed, but the underlying ethical obligation to ensure decisions are made on the basis of full and balanced information is compromised.

The informational and research pathways that might alter the status quo, have been actively suppressed through MBIE's science policies that favour research agendas that prioritise innovation-based research with technological outcomes. As a consequence, public good, basic research to elucidate the role of nutrition in the protection of health and prevention of disease across the medical, agricultural and health sciences have dwindled.

Funding incentives and triggers ensure that researchers modify science and research funding proposals to favour 'innovation' research and away from research that focuses on a population's needs. Research can be narrowed around specific topics and methodologies, preventing less 'desirable' research from receiving attention. Large research groups and individual researchers will not risk contradicting the policy goal and losing their funding source. Government officials and contracted agencies, then produce policy and health promotion material that is consistent with the preformed agendas handed down by senior staff and officials. Policy agencies commission research that harmonises with preformed policy goals. [611]

The structure and operating practices of these agencies leave little room for internal challenge to long-standing approaches. Moreover, there are no dedicated policies or funding mechanisms to ensure that government agencies undertake transparent, rigorous, and regularly scheduled reviews of the scientific literature, conducted at arm's length from the agencies responsible for implementing policy advice and interventions. The harmonisation of position statements used elsewhere, in countries with similar disease profiles, is not adequate for government officials who are charged with administering legislation to protect health.

The Medicines Act 1981, procedural norms, guideline rigidity and the absence of research capacity play an important role in perpetuating existing orthodoxies and keeping new evidence at arm's length. Nutrition-education forms only a relatively small element of medical education while medical prescribing forms a much larger part. [612] In the past, doctors who have deviated from guideline advice have historically been sanctioned by the Medical Council of New Zealand (MCNZ). Following the COVID-19 event, the MCNZ increased sanctioning actions against doctors who failed to follow medical guidelines and who advocated too strongly for dietary and nutritional solutions for certain conditions. This has continued into 2025.

The notion of a single, isolated disease cause is increasingly recognised as the exception rather than the rule. Today, when scientists explore psychiatric, neurodevelopmental, and neurodegenerative conditions they show metabolic *and* immune system impairment. The question may arise, is it uncontroversial that drug-related research can evaluate the role of, for example, the immune system to understand brain-immune relationships, but it is controversial to highlight the underlying role of nutrition in mediating the immune system and the associative role of the neurological system? For example, scientists exploring drug development recently identified 29 biomarkers that suggest an underlying role of the immune system in a range of seven neuropsychiatric conditions.[613]

It is well established that nutrition is poorly incorporated in medical education 'regardless of country, setting, or year of medical education'.[614] U.S.-based doctors for example, receive on average, 19 hours of nutritional training. [615] [616] A 2024 survey found that fewer than 22% of surveyed medical schools meet the minimum recommended 25 hours of nutrition education for medical students.[617] A review of medical students and doctors examining why nutrition knowledge is lacking, identified a range of complex drivers, including insufficient curriculum time dedicated to nutrition education, perceptions and confidence,

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[611] Senior HJ, *et al* (2025). How governments influence public health research.
[612] King RE, Palermo C, Wilson AN. (2023) Mapping nutrition within medical curricula in Australia and New Zealand: a cross-sectional content analysis. *BMJ Nutr Prev Health*. 6(2):196-202. DOI: 10.1136/bmjnph-2022-000522.
[613] Dardani C, Robinson JW, Jones HJ. *et al*. (2025) Immunological drivers and potential novel drug targets for major psychiatric, neurodevelopmental & neurodegenerative conditions. *Mol Psychiatry*. DOI: 10.1038/s41380-025-03032-x
[614] Crowley J, Ball L, Hiddink GJ. (2019). Nutrition in medical education: a systematic review. *The Lancet Planetary Health*, 3(9):e379 - e389
[615] Adams KM, Kohlmeier M, Zeisel SH.(2010). Nutrition education in U.S. medical schools: latest update of a national survey. *Acad Med*. 85(9):1537-42. DOI: 10.1097/ACM.0b013e3181eab71b. PMID: 20736683; PMCID: PMC4042309.
[616] Devries S, Dalen JE, Eisenberg DM. *et al*. (2014). A Deficiency of Nutrition Education in Medical Training. *The American Journal of Medicine*, 127(9):804 – 806, DOI: 10.1016/j.amjmed.2014.04.003
[617] Eldin MM, Huynh RA, Khan S. *et al*. (2024). The Current State of Nutrition Education in Medical Schools in the United States: An Analysis of Curriculum, Faculty Perspectives and Resources. bmjnph 2024;7(Suppl 1):A1–A8. https://nutrition.bmj.com/content/bmjnph/7/Suppl_1/A4.1.full.pdf

stigmas and health habits, and challenges in clinical practice.[618] The effect is that doctors are unlikely to view nutrition as a therapeutic option for patients and translate the benefits of nutritional research, advice and interventions into medical training and practice.[619]

Avenues for doctors to engage in post-graduate, and continuing, or professional development education are limited. U.K. royal colleges have been criticised for failing to encourage and develop pathways for doctors to study nutrition as an ongoing professional development.[620] Organisations such as the Australasian Society of Lifestyle Medicine (ASLM), the Australasian College of Nutritional and Environmental Medicine (ACNEM) and others, run nutrition-based courses through which medical practitioners can accrue continuing professional development (CPD) or continuing medical education (CME) points, that are approved by the Royal Australian College of General Practitioners (RACGP), Royal New Zealand College of General Practitioners (RNZCGP) and other professional bodies.

The challenge facing New Zealand's health system is not a simple one. Cultural, educational, structural and informational barriers intersect in complex ways, undermining the ability of Members of Parliament, health-sector agencies, independent practitioners and the public to accurately evaluate and address the drivers of poor metabolic and mental health, and to implement strategies that directly tackle inadequate food quality, nutritional insufficiency and harmful dietary patterns, including those shaped by addiction as well as social, psychological, educational and economic factors.

Is Governance Failing?

A democratic government is responsible for serving the public's best interests. The World Health Organisation, in the preamble to its constitution, sets out this selection of principles about health, which recognises health as having a central role in the public interest:

> *Health is a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity.*
>
> - *The enjoyment of the highest attainable standard of health is one of the fundamental rights of every human being without distinction of race, religion, political belief, economic or social condition.*
> - *The extension to all peoples of the benefits of medical, psychological and related knowledge is essential to the fullest attainment of health.*
> - *Informed opinion and active co-operation on the part of the public are of the utmost importance in the improvement of the health of the people.*
> - *Governments have a responsibility for the health of their people, which can be fulfilled only by the provision of adequate health and social measures.*[621]

From a public and constitutional law perspective, health lies at the core of the public interest. Vote Health is the Government's single largest area of expenditure, and virtually all aspects of government activity, economic, environmental, regulatory, and social, have direct or indirect effects on population health. In constitutional terms, function should follow form. This paper demonstrates that New Zealand's current governance arrangements are not fulfilling their intended public function. While the present approach to health policy formulation and implementation is inadequate, it is not immutable; like metabolic dysfunction, institutional failure is capable of correction.

As documented in this paper, New Zealand's governance structures have, through both omission and commission, increasingly prioritised the interests of the medical and food industries over the public

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[618] Khiri N, Howells K. (2025). Nutritional Education in Medical Curricula and Clinical Practice: A Scoping Review on the Knowledge Deficit Amongst Medical Students and Doctors. *J Hum Nutr Diet*. 38(2):e70031. DOI: 10.1111/jhn.70031.
[619] Devries S, Willett W, Bonow RO. (2019). Nutrition Education in Medical School, Residency Training, and Practice. *JAMA*. 321(14):1351–1352. DOI:10.1001/jama.2019.1581
[620] Khiri N, Howells K. (2025). Nutritional Education in Medical Curricula and Clinical Practice.
[621] World Health Organization. Preamble. https://www.who.int/about/governance/constitution

interest. The resulting costs are borne by the public through reduced quality and length of life, alongside the escalating fiscal burden of largely preventable and reversible disease. In this context, New Zealand's poor population health outcomes and the disproportionate growth in health expenditure may reasonably be viewed as bellwether indicators of wider governance failure.

PSGRNZ advances a set of recommendations aimed at addressing, to a significant degree, the proximate drivers of metabolic and mental illness. However, a genuinely root-cause approach requires more than sector-specific reform. Addressing the entrenched malaise within New Zealand's health system, where conventional approaches persist despite demonstrable ineffectiveness, necessitates systemic reform of governance structures themselves. Constitutional scholars have long identified structural weaknesses within New Zealand's governance arrangements and have increasingly emphasised the need for democratic renewal to restore institutional legitimacy and effectiveness.[622]

Governance structures must be capable of delivering on the public interest across all domains, with health recognised as an overarching and cross-cutting priority, rather than a siloed policy outcome. For a democratic system to function effectively, it must incorporate mechanisms that ensure transparency, accountability, and meaningful public participation. This requires institutional arrangements that safeguard sovereignty, reinforce genuine separation of powers, resist excessive centralisation, and limit opportunities for private or commercial interests to compromise public decision-making.

Equally essential is the availability of effective legal accountability mechanisms. Members of the public must have meaningful access to justice, including avenues for scrutiny and, where appropriate, inquisitorial inquiry into proposed policies and into policies or systems that demonstrably fail to serve the public interest. Administrative law, potentially supported by the evolution of a written constitutional framework, offers a coherent legal pathway through which much of the necessary structural and cultural reform could be advanced. [623]

[622] Palmer G. & Butler A. 2018. Towards Democratic Renewal. Victoria University Press

[623] Joseph, P. (2021). Joseph on Constitutional and Administrative Law, 5th Ed. Thomson Reuters

PART III. REFORM. FOCUS ON HUMAN BIOLOGY

9. TYPE 2 DIABETES: REMISSION IS REAL

Type two diabetes mellitus (T2DM) has traditionally been characterised as a chronic, progressive condition not amenable to reversal. It was believed that pancreatic beta cell function was permanently lost by all people who were diagnosed with T2DM. However, clinicians working with patients now understand that the permanent loss of beta cell function is rare. Clinicians report that the sooner the patient adopts a low-carbohydrate diet, after a diagnosis of prediabetes or T2DM, the more likely natural insulin metabolism can resume. [624] Reversal and/or remission of T2DM is well documented in 2025.

T2DM remission, defined as normal blood glucose levels for 3 months or more in the absence of pharmacological therapy is dependent on factors including the length of time with T2DM, personal capacity and support networks.[625]

Physicians adopting a low-carbohydrate approach with patients have demonstrated that long-term improvements in blood glucose control (lower HbA1c levels),[626] can lead to remission of prediabetes and T2DM[627] [628] and reduce or eliminate dependence on diabetes medication use. [629] [630] [631].[632]

A battery of papers has been published in recent years which demonstrate broad improvement across metabolic disease parameters beyond T2DM. Trials consistently demonstrate that ketogenic diets reduce seizure risk and improve neurologic and cardiometabolic outcomes.[633] [634] [635] [636] Studies report:

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[624] Taylor R, Al-Mrabeh A, Zhyzhneuskaya S, *et al.* (2018). Remission of human type 2 diabetes requires decrease in liver and pancreas fat content but is dependent upon capacity for β cell recovery. *Cell Metabolism*, 28(4), 547–556.e3. DOI: 10.1016/j.cmet.2018.07.003.

[625] Hallberg SJ, McKenzie AL, Williams PT, *et al.* (2018). Effectiveness and Safety of a Novel Care Model for the Management of Type 2 Diabetes at 1 Year: An Open-Label, Non-Randomized, Controlled Study. *Diabetes Ther.* 9(2):583-612. doi: 10.1007/s13300-018-0373-9. Epub 2018 Feb 7. Erratum in: Diabetes Ther. 2018 Apr;9(2):613-621. doi: 10.1007/s13300-018-0386-4.

[626] Yuan, X., Wang, J., Yang, S. *et al*. (2020). Effect of the ketogenic diet on glycemic control, insulin resistance, and lipid metabolism in patients with T2DM: a systematic review and meta-analysis. *Nutr. Diabetes* 10, 38 (2020). DOI: 10.1038/s41387-020-00142-z

[627] Unwin D, Delon C, Unwin J, et al. (2023) What predicts drug-free type 2 diabetes remission? Insights from an 8- year general practice service evaluation of a lower carbohydrate diet with weight loss. *BMJ Nutrition, Prevention & Health*.

[628] Brown A, McArdle P, Taplin J, Unwin D, Unwin, J, *et al.* (2022). Dietary strategies for remission of type 2 diabetes: A narrative review. *J Hum Nutr Diet.* 35:165–178. DOI: 10.1111/jhn.12938.

[629] Unwin D, Khalid AA, Unwin J, *et al*. (2020). Insights from a general practice service evaluation supporting a lower carbohydrate diet in patients with type 2 diabetes mellitus and prediabetes: a secondary analysis of routine clinic data including HbA1c, weight and prescribing over 6 years. BMJ Nutr Prev Health.2020;3:285–94 bmjnph-2020-000072.

[630] MacKay D, Chan C, Dasgupta K *et al.* (2022). Remission of Type 2 Diabetes. Diabetes Canada Clinical Practice Guidelines Expert Working Group. *Can J Diabetes*, 46:753-761. DOI: DOI: 10.1016/j.jcjd.2022.10.004.

[631] Lingvay I, Sumithran P, Cohen RV, le Roux CW. (2022) Obesity management as a primary treatment goal for type 2 diabetes: time to reframe the conversation. *Lancet*;399:394–405

[632] Xin Y, Davies A, Briggs A, McCombie L, Messow CM, Grieve E, et al. (2020). Type 2 diabetes remission: 2 year within-trial and lifetime-horizon cost-effectiveness of the Diabetes Remission Clinical Trial (DiRECT)/counterweight-plus weight management programme. *Diabetologia*.63(10):2112–22. DOI: 10.1007/s00125-020-05224-2.

[633] Dyńka D, Kowalcze K, Paziewska A. (2022). The Role of Ketogenic Diet in the Treatment of Neurological Diseases. *Nutrients.* 14(23):5003. DOI: 10.3390/nu14235003.

[634] Patikorn C, Saidoung P, Pham T. *et al.* (2023). Effects of ketogenic diet on health outcomes: an umbrella review of meta-analyses of randomized clinical trials. *BMC Med* 21:196. DOI:10.1186/s12916-023-02874-y

[635] Nojek P, Zawół M, Zimonczyk M, *et al.* (2024) Ketogenic diet and metabolic health.

[636] Baylie T, Ayelgn T, Tiruneh M, Tefsa KH (2024). Effect of Ketogenic Diet on Obesity and Other Metabolic Disorders.

- Improvements in kidney function, [637]
- Improvements in neurodegenerative conditions. E.g. Parkinson's and Alzheimer's disease.[638] [639] [640]
- Reduced risk for cancer.[641] [642] [643]
- Lower blood pressure.[644]
- Improved mitochondrial function.[645]
- Improvements in biomarkers for cardiovascular disease.[646] [647] [648] [649]

Type 1 diabetes mellitus: Carb Control Sees Improvements in Health Biomarkers & Quality of Life.

Type 1 diabetics (T1DM) who adopt a long-term low carb or ketogenic diet can experience improvements in quality of life and reduce risk for chronic conditions that are commonly associated with T1DM.[650] People with T1DM have a ten-fold higher risk for cardiovascular disease risk compared to the general population. Case studies assessing the metabolic parameters of T1DM individuals on ketogenic diets to manage glycemia and lower insulin requirements, have demonstrated improvements in biomarkers which suggest

[637] Athinarayanan SJ, Roberts CGP, Phinney SD, *et al.* (2025). Effects of a continuous remote care intervention including nutritional ketosis on kidney function and inflammation in adults with type 2 diabetes: a post-hoc latent class trajectory analysis. *Front Nutr.* Jun 6(12):1609737. doi: 10.3389/fnut.2025.1609737. PMID: 40547366

[638] Phillips, M.C.L., Deprez, L.M., Mortimer, G.M.N. et al. Randomized crossover trial of a modified ketogenic diet in Alzheimer's disease. Alz Res Therapy 13, 51 (2021). https://doi.org/10.1186/s13195-021-00783-x

[639] Phillips, M.C.L., Picard, M. Neurodegenerative disorders, metabolic icebergs, and mitohormesis. *Transl Neurodegener* **13**, 46 (2024). https://doi.org/10.1186/s40035-024-00435-8

[640] Rong L, Peng Y, Shen Q. *et al.* (2024). Effects of ketogenic diet on cognitive function of patients with Alzheimer's disease: a systematic review and meta-analysis. *J nutrition, health and aging*. 28(8):100306. DOI: 10.1016/j.jnha.2024.100306

[641] Duraj, T., Kalamian, M., Zuccoli, G. et al. Clinical research framework proposal for ketogenic metabolic therapy in glioblastoma. BMC Med 22, 578 (2024). https://doi.org/10.1186/s12916-024-03775-4

[642] Phillips MC, Thotathil Z, Hari Dass P, Ziad F and Moon BG: Ketogenic metabolic therapy in conjunction with standard treatment for glioblastoma: A case report. Oncol Lett 27: 230, 2024.

[643] Klement, R. J. (2025). Is the ketogenic diet still controversial in cancer treatment? *Expert Review of Anticancer Therapy*, 1–5. DOI:10.1080/14737140.2025.2522936

[644] Unwin DJ, Tobin SD, Murray SW, *et al.* (2019) Substantial and Sustained Improvements in Blood Pressure, Weight and Lipid Profiles from a Carbohydrate Restricted Diet: An Observational Study of Insulin Resistant Patients in Primary Care. *Int J Environ Res Public Health,* 16(15):2680. doi: 10.3390/ijerph16152680.

[645] Miller VJ, LaFountain RA, Barnhart E. et al. (2020) A ketogenic diet combined with exercise alters mitochondrial function in human skeletal muscle while improving metabolic health. *Am J Physiol Endocrinol Metab*. 1;319(6):E995-E1007. DOI: 10.1152/ajpendo.00305.2020.

[646] Bhanpuri NH, Hallberg SJ, Williams PT, *et al*. (2018). Cardiovascular disease risk factor responses to a type 2 diabetes care model including nutritional ketosis induced by sustained carbohydrate restriction at 1 year: an open label, non-randomized, controlled study. *Cardiovasc Diabetol.*;17:56. DOI: 10.1186/s12933-018-0698-8

[647] Hu T, Mills KT, YaoL ,*et al* (2012) Effects of Low-Carbohydrate Diets Versus Low-Fat Diets on Metabolic Risk Factors: A Meta-Analysis of Randomized Controlled Clinical Trials, *American Journal of Epidemiology*, 176(7): S44–S54, DOI:10.1093/aje/kws264

[648] Unwin DJ, Tobin SD, Murray SW, Delon C, Brady AJ.(2019) Substantial and Sustained Improvements in Blood Pressure, Weight and Lipid Profiles from a Carbohydrate Restricted Diet: An Observational Study of Insulin Resistant Patients in Primary Care. *Int J Environ Res Public Health.* 16(15):2680. DOI: 10.3390/ijerph16152680

[649] Saslow, L.R., Daubenmier, J.J., Moskowitz, J.T. *et al.* (2017) Twelve-month outcomes of a randomized trial of a moderate-carbohydrate versus very low-carbohydrate diet in overweight adults with type 2 diabetes mellitus or prediabetes. *Nutr & Diabetes* 7, 304 (2017). DOI:10.1038/s41387-017-0006-9

[650] Turton JL, Brinkworth GD, Parker HM, et al. (2023) Effects of a low-carbohydrate diet in adults with type 1 diabetes: A single arm non-randomised clinical trial. *PLoS ONE* 18(7): e0288440. DOI:10.1371/journal.pone.0288440

that such individuals, if this diet is maintained over time, can reduce risk for cardiovascular disease and other complications, compared to individuals that follow a conventional T1DM approach.[651] [652] [653] [654]

A protocol was developed in 2021 to support parents to adopt and monitor a low-carbohydrate/ketogenic approach in children and adolescents.[655] Diabetic ketoacidosis (where the body lacks sufficient insulin) may not be a major risk for T1DM individuals who pursue ketogenic diet.[656] Research is still in its infancy and this approach 'may be cautiously considered in highly motivated, well-supported adult patients with structured education and access to continuous glucose and ketone monitoring'.[657]

10. WHOLE OF SYSTEM REFORM: HEALTH COACHING CENTRAL TO REVERSAL & REMISSION OF METABOLIC & MENTAL ILLNESS

New Zealand is an early adopter of integrative health coaching, involving goal setting with values and a sense of purpose. This is distinct from a medical model.[658] Long-term dietary changes are not conventionally viewed as clinical treatment, in the way that pharmaceutical drugs are regarded. An increasing volume of scientific data challenges this perspective.

Health coaching has evolved to predominantly support patients with T2DM and/or other metabolic and brain-related conditions to change food habits and behaviours to reduce carbohydrate intake and increase healthy fat and protein intake. The use of continuous glucose monitoring (CGM) in glycaemic and weight control (discussed later in this section) in addition to health coaching, may potentiate patient improvements.[659]

Coaching involves food and nutrition education, and the attainment of skills to support individuals to navigate daily challenges. Individuals can be assisted to make small and large changes, often revolving around attainment of long-term goals, that gradually become healthy habits and which become embedded as praxis. Coaching and support groups often focus on the management of environmental triggers and behaviour associated with food addiction and recognising the role of wrap-around, complimentary services. [660] [661]

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[651] Watso JC, Robinson AT, Singar SAB et al. (2024). Advanced cardiovascular physiology in an individual with type 1 diabetes after10-year ketogenic diet. *Am J Physiol Cell Physiol* 327: C446–C461. DOI:10.1152/ajpcell.00694.2023
[652] Gardemann C, Knowles S, Marquardt T (2023). Managing type 1 diabetes mellitus with a ketogenic diet. *Endocrinology, Diabetes & Metabolism,* 23:008 DOI:10.1530/EDM-23-0008
[653] Koutnik AP, Klein K, Robinson AT, Watso JC. (2024). Efficacy and Safety of Long-term Ketogenic Diet Therapy in a Patient With Type 1 Diabetes, *JCEM Case Reports*, 2(7):luae102, DOI: 10.1210/jcemcr/luae102
[654] Tóth C, Clemens Z. (2014). Type 1 diabetes mellitus successfully managed with the paleolithic ketogenic diet. Int J Case Rep Images 2014;5(10):699–703. DOI: 10.5348/ijcri-2014124-CR-10435
[655] Rydin AA, Spiegel G, Frohnert BI, *et al.* (2021). Medical management of children with type 1 diabetes on low-carbohydrate or ketogenic diets. *Pediatric Diabetes,* DOI: 10.1111/pedi.13179.
[656] Ozoran H, Matheou M, Dyson P *et al.* (2023). Type 1 diabetes and low-carbohydrate diets—Defining the degree of nutritional ketosis. *Diabetic Medicine,* 40(10);15178. DOI: 10.1111/dme.15178
[657] Korakas E, Kountouri A, Petrovski G, Lambadiari, V. (2025). Low-Carb and Ketogenic Diets in Type 1 Diabetes: Efficacy and Safety Concerns. *Nutrients,* 17:2001. DOI:10.3390/nu17122001
[658] Wolever RQ, Caldwell KL, Wakefield JP *et al.* (2011) Integrative Health Coaching: An Organizational Case Study. *Explore,* 7(1):30-36. DOI: 10.1016/j.explore.2010.10.003
[659] Taylor PJ, Thompson CH, Brinkworth GD. (2018). Effectiveness and acceptability of continuous glucose monitoring for type 2 diabetes management: A narrative review. *J Diabetes Investig.* 9(4):713-725. DOI: 10.1111/jdi.12807.
[660] Zinn C, Campbell JL, Po M. *et al.* (2024) Redefining Diabetes Care: Evaluating the Impact of a Carbohydrate-Reduction, Health Coach Approach Model in New Zealand. Journal of Diabetes Research. 2024:4843889, DOI:10.1155/jdr/4843889
[661] Zinn C, Campbell JL, Fraser L. *et al*. (2025) Carbohydrate Reduction and a Holistic Model of Care in Diabetes Management: Insights from a Retrospective Multi-Year Audit in New Zealand. *Nutrients*.17(24):3953.

New Zealand Primary Health Organisations (PHOs) offer community-based services under an integrated primary mental health and addiction services (IPMHA) that has the aim of building 'people's motivation and capability to better understand and actively manage their physical and emotional wellbeing needs'.[662] In 2025 PHO *Health Improvement Practitioners* and Health Coaches were more likely to be predominantly directed toward general social or life-coaching' functions. People working within the IPMHA framework may support skills development, but they are not specifically tasked with addressing diet and nutrition in ways that would reduce risk for prediabetes, diabetes, cardiovascular disease and improve brain health.[663]

The Te Whatu Ora documentation defining the scope and requirements of Health Coaching, including training standards and learning outcomes, does not include reference to diet or nutrition.[664] [665]

However, in January 2026, Health New Zealand appointed Collaborative Aotearoa, a membership-based network of primary and community health organisations and partners, in partnership with Prekure, a New Zealand-based health coach training provider, as a provider for the Integrated Primary Mental Health and Addiction (IPMHA) Health Coach Training programme.[666] Prekure's core focus is nutritional science and behaviour change to support health. As a result, the content and orientation of IPMHA health coach training may shift towards a stronger emphasis on dietary nutrition.

A recent study, *Redefining Diabetes Care* in New Zealand reported significant health improvements, including weight loss, reduced medication burden, and increased energy with a health coach model.:[667]

> *An increasing number of GPs are now able to refer patients to health coaches and wellbeing advisors, called Health Improvement Practitioners. These healthcare providers are now employed either within a GP clinic or in a Primary Health Organisation (a cluster of clinics which work together to care for patients who are registered with them).*[668]

[662] Te Whatu Ora (Feb 2024). Integrated Primary Mental Health & Addictions HCES. Updated practice profile. February 2024. https://d2ew8vb2gktr0m.cloudfront.net/files/Updated-IPMHA-Practice-Profile-Te-Whatu-Ora-Feb-24.pdf
[663] Te Pou (2026). Integrated primary mental health and addiction. https://www.tepou.co.nz/initiatives/integrated-primary-mental-health-and-addiction
[664] Te Whatu Ora (Feb 2024). Integrated Primary Mental Health & Addictions HCES. Updated practice profile.
[665] Te Pou. Health Coaching. https://www.tepou.co.nz/initiatives/integrated-primary-mental-health-and-addiction/health-coaching
[666] Scoop (January 20, 2026). PREKURE And Collaborative Aotearoa Appointed To Build Aotearoa's "Primary Care-Ready" Health Coach Workforce. https://www.scoop.co.nz/stories/GE2601/S00026/prekure-and-collaborative-aotearoa-appointed-to-build-aotearoas-primary-care-ready-health-coach-workforce.htm?
[667] Zinn C, Campbell JL, Po M. *et al.* (2024) Redefining Diabetes Care: Evaluating the Impact of a Carbohydrate-Reduction, Health Coach Approach Model in New Zealand. Journal of Diabetes Research. 2024:4843889, DOI:10.1155/jdr/4843889
[668] Zinn C, Campbell JL, Po M. *et al.* (2024) Redefining Diabetes Care.

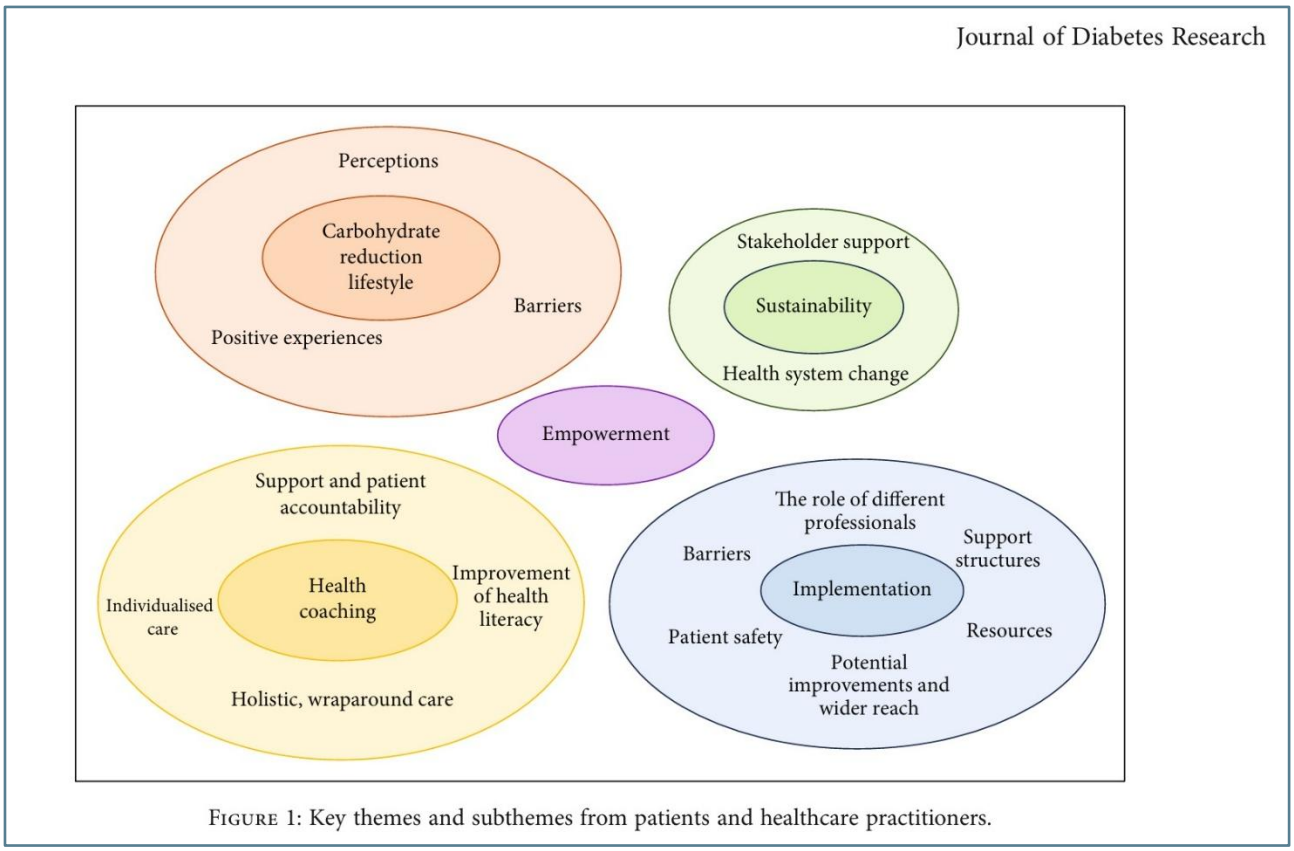

Figure 11. Zinn C, Campbell JL, Po M. et al. (2024) Redefining Diabetes Care: Evaluating the Impact of a Carbohydrate-Reduction, Health Coach Approach Model in New Zealand. *Journal of Diabetes Research*.

Low-Carb Approaches in New Zealand: 2025 Audit of Three Primary Care Practices.

Audits of clinical data can provide real-world insight into the outcomes of clinical interventions. U.K.-based Dr David Unwin has argued that clinical audits should not be a poor cousin to other forms of research:

> *They both start with a question, both expect the answer to inform, change or influence clinical practice, both require formal data collection on patients and both depend on using an appropriate method and design to reach sound conclusions.*[669] [670]

Lead author Caryn Zinn of the *Redefining Diabetes Care* paper, together with colleagues specialising in low-carbohydrate interventions, subsequently conducted a retrospective, observational, real-world clinical audit across three New Zealand-based primary care practices, as a service evaluation, to assess models of care and clinical outcomes. The audit aimed to:

1. *Describe changes in glycosylated haemoglobin (HbA1c) and diabetes status;*
2. *Identify factors associated with HbA1c improvement; and*
3. *Examine changes in related cardiometabolic outcomes.*[671]

[669] Wade DT. (2005). Ethics, audit, and research: all shades of grey. *BMJ* 330:468–71.
[670] Unwin D. (2024) Reducing overweight and obesity; so how are we doing? BMJ Nutrition, *Prevention & Health* 0:e000836. DOI:10.1136/bmjnph-2023-000836
[671] Zinn C, Campbell JL, Fraser L. *et al*. (2025) Carbohydrate Reduction and a Holistic Model of Care in Diabetes Management: Insights from a Retrospective Multi-Year Audit in New Zealand. *Nutrients*.17(24):3953. Page 3.

The three primary care practices utilise a three-pronged approach as a model of care that seeks to manage and potentially reverse prediabetes and T2DM which integrates (a) whole-food, carbohydrate-reduction, (b) a health-coach approach, and (c) supportive community initiatives.

While the methods were consistent (GP oversight, carbohydrate-reduction guidance, and access to health coaching), approaches differed, such as session frequency, content, mode of delivery, and cultural tailoring. These differences were associated with funding models, community-based education opportunities, and the extent of group or peer-support initiatives available at each site which were also a function of local demographics and cultural context. [672]

While the authors acknowledged limitations, including its retrospective design and lack of a control group, the study provided important insight into the consistency of outcomes across diverse settings that flexibly accounted for local culture, practice variation, and community resourcing. Importantly, the study included substantial Māori and Pasifika representation, key groups that experience elevated risk of prediabetes and T2DM. One practice that serves a high Māori and low socio-economic population multiple patients experienced substantial improvements in HbA1c levels, including to under the T2DM range.

> *44.4% of patients with PD achieved normal HbA1C levels at follow-up, 32.1% of those with T2D at baseline were able to reverse their condition*.

The paper noted the importance of long-term support:

> *Anecdotal clinician feedback suggests that adherence often fluctuates, with patients cycling between engagement and lapses before re-committing. This aligns with findings from Unwin et al., where longer time on a low-carbohydrate programme correlated weakly with smaller HbA1c improvements, likely reflecting reduced adherence rather than loss of intervention efficacy.*

The study provides evidence of the importance of support in communities that experience high risk of prediabetes and T2DM due to ethnicity and/or low-socioeconomic status.

> *These findings highlight the importance of culturally and systemically aligned models of care in populations facing longstanding inequities in access to effective T2D management. Such diversity and pragmatic design enhance generalisability compared with tightly controlled trials.*

The Zinn *et al* 2025 paper joins an increasing group of case studies which demonstrate that the health coach model can be rolled out in general practice clinics, in the private sector and in communities, and that the resultant dietary changes produce improvements in metabolic and mental health and reduce dependency on medication.[673] [674] [675]

Proof of the health coaching for remission of T2DM concept had been earlier demonstrated by a UK-based medical (general practitioner) Norwood NHS Surgery, spearheaded by Drs David and Jen Unwin. There is now sufficient published evidence that: [676]

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[672] Zinn C, Campbell JL, Fraser L. *et al*. (2025) Carbohydrate Reduction and a Holistic Model of Care. Page 3.
[673] Saner E, Kalayjian T, Buchanan L et al. *(2025)* TOWARD: a metabolic health intervention that improves food addiction and binge eating symptoms. *Front. Psychiatry.* Vol.16. DOI: 10.3389/fpsyt.2025.1612551
[674] Zinn C, Campbell JL, Po M. *et al.* (2024) Redefining Diabetes Care: Evaluating the Impact of a Carbohydrate-Reduction, Health Coach Approach Model in New Zealand. Journal of Diabetes Research. 2024:4843889, DOI:10.1155/jdr/4843889
[675] Unwin J, Delon C, Giæver H, *et al.* (2022) Low-carbohydrate and psychoeducational programs show promise for the treatment of ultra-processed food addiction. *Front. Psychiatry* 13:1005523.
[676] Unwin D, Delon C, Unwin J, et al. (2022) What predicts drug-free type 2 diabetes remission?

Type 2 diabetes remission (defined as an HbA1c < 48 mmol mol) should be considered as a treatment goal for people living with T2DM (especially for those within 6 years from being diagnosed). The ability to achieve this may be influenced by duration of diabetes, weight loss and gender.

Based on the evidence from clinical trials weight loss (typically 15 kg or greater) is the main driver and predictor of remission.

Drug expenditure can be expected to decline as risk indicators improve. UK case study: average Norwood surgery spend was £4.94 per patient per year on drugs for diabetes compared with £11.30 for local practices. In the year ending January 2022, Norwood surgery spent £68 353 per year less than the area average.

The UK-based Unwin clinic commenced low-carbohydrate health coaching in 2013 after Dr David Unwin recognised that without intervention, the 27% diabetes practice population rate would continue to expand. The goal was to achieve T2DM remission and address food addiction. Resistance in the practice was so severe that the dietician left, and the other doctors wanted to have nothing to do with it. By 2024, the entire practice was on board with the programme. The clinic has become a global case study for the reversal and remission of T2DM with clinicians across the UK have since adopted the principles initiated by the Unwin clinic. [677]

Patients could elect to adopt the programme, which included dietary advice, food addiction counselling and a weekly support group meeting. After a decade people in the initial group remain, to support new entrants. Dr Jen Unwin, a former NHS clinical psychologist, joined the team to address food addiction challenges.

The initial programme was controversial. The Unwin team recorded a wide range of data points, publishing their findings in a series of papers in scientific journals to document progress at the 8-month[678], six[679] and eight-year stages to ensure that key biomarkers were tracked to ensure maximum transparency. Colleagues had expressed concern about the impact of increased protein on kidney function. The clinic tracked renal function, recording that serum creatine markers improved significantly.[680]

Findings included a decrease in health system costs. This included a decrease in medication expenditure, reduction in multimorbidity, and the potential decreased pressure on the public health system with fewer patient visits over time.[681] [682] The intervention:

'delivered significant improvements in HbA1c with 20% of the practice's population achieving drug-free T2D remission. There have also been a range of important cardiovascular risk factor improvements.

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[677] Kelly, T, Unwin, D, Finucane, F. (2020). Low-Carbohydrate Diets in the Management of Obesity and Type 2 Diabetes: A Review from Clinicians Using the Approach in Practice. *Int. J. Environ. Res. Public Health* 2020, 17, 2557. DOI: 10.3390/ijerph17072557

[678] Unwin D and Unwin J. (2014). Low-carbohydrate diet to achieve weight loss and improve HbA1c in type 2 diabetes and pre-diabetes: experience from one general practice. *Practical Diabetes* 2014:31;2:76-79. DOI: 10.1002/pdi.1835

[679] Unwin D, Khalid AA, Unwin J, *et al.* (2020). Insights from a general practice service evaluation supporting a lower carbohydrate diet in patients with type 2 diabetes mellitus and prediabetes: a secondary analysis of routine clinic data including HbA1c, weight and prescribing over 6 years. BMJ Nutrition, Prevention & Health 2020;3:e000072. doi:10.1136/bmjnph-2020-000072

[680] Unwin D, Unwin J, Crocombe D *et al*, (2021). Renal function in patients following a low-carbohydrate diet for type 2 diabetes: a review of the literature and analysis of routine clinical data from a primary care service over 7 years. *Current Opinion in Endocrinology & Diabetes and Obesity* 28(5):469-479, DOI: 10.1097/MED.0000000000000658

[681] Xin Y, Davies A, Briggs A, McCombie L, Messow CM, Grieve E, *et al.* (2020). Type 2 diabetes remission: 2 year within-trial and lifetime-horizon cost-effectiveness of the Diabetes Remission Clinical Trial (DiRECT)/counterweight-plus weight management programme. *Diabetologia*.63(10):2112–22

[682] Brown A, McArdle P, Taplin J, Unwin D, Unwin, J, *et al.* (2022). Dietary strategies for remission of type 2 diabetes: A narrative review. J Hum Nutr Diet. 35:165–178.

Diabetes drug savings are £68 353 per year compared with the local average. These savings are likely to be dwarfed by cost savings from reduced complications of T2D and days lost from work.[683]

The low-carbohydrate approach has been adapted for New Zealand by local organisations such as Reversal NZ and Prekure. To help patients and clients take dietary steps to reduce their ultraprocessed food and carbohydrate intake, Reversal and Prekure have released a nutrition ladder to highlight different and graduated dietary approaches, and their potential impact on health status.[684]

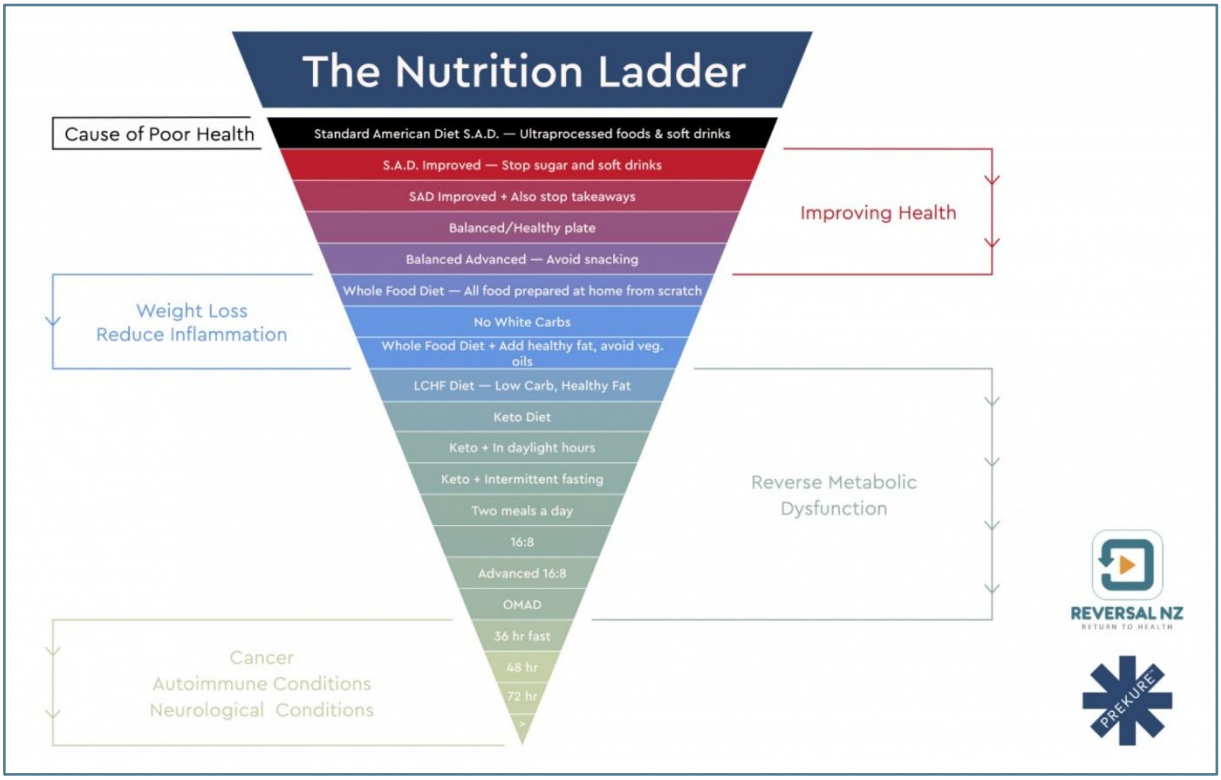

Figure 12. Reversal NZ. Prekure. (August 2022). The Nutrition Ladder. HTTPS://REVERSALNZ.CO.NZ/2022/08/21/THE-NUTRITION-LADDER/

In Australia, anaesthetist Rod Tayler and fitness professional Jamie Taylor founded the Low Carb Downunder website[685] as an information gateway on the subject of restricting carbohydrate intake for long-term health benefits.

A dietary shift to a low-carbohydrate, higher fat diet in New Zealand may be affordable for the majority of people. A 2019 review concluded that total daily costs were $43.42 (national guidelines) and $51.67 (LCHF) representing an $8.25 difference, or $2.06 per person, with the LCHF meal plan being the costlier option. [686]

[683] Unwin D, Delon C, Unwin J, *et al*. (2023). What predicts drug-free type 2 diabetes remission?
[684] Reversal NZ. Prekure (Aug. 2022). The Nutrition Ladder. https://reversalnz.co.nz/2022/08/21/the-nutrition-ladder/
[685] Low Carb Down Under. lowcarbdownunder.com.au
[686] Zinn C, North S, Donovan, Muir C, Henderson G. (2019). Low-carbohydrate, healthy-fat eating: A cost comparison with national dietary guidelines. *Nutrition & Dietetics,* 22(2):283-291. DOI: 10.1111/1747-0080.12534

Deprescribing Following Improvements in Blood Pressure, Insulin, Weight and Lipid Profile.

Low-carbohydrate diets can result in decreases and changes in blood pressure, lipid profiles and other biomarkers of inflammation. As these markers change, doctors can adjust or deprescribe medication as patient parameters change.[687] Deprescribing is the:

> *systematic process of identifying and discontinuing drugs when existing or potential harms outweigh existing or potential benefits within the context of an individual patient's care goals, functional status, life expectancy, values, and preferences.*[688]

A Consensus Report (2022) by the American Diabetes Association (ADA) and the European Association for the Study of Diabetes (EASD) emphasised the individual's role in T2DM including undertaking glucose lowering interventions, which could incorporate behavioural shifts, low-carbohydrate approaches and shared decision-making, along with traditional medical measures. The Consensus Report recognised the potential for remission, but that an individualised approach for T2DM would reflect personal capacity.

The Consensus Report emphasised the importance of long-term glycaemic management to stem the often-microvascular systemic risks associated with a failure to control glucose, including cardiovascular risk factors, organ degeneration (e.g. kidney) and cognitive decline.[689]

Programmes can support patient-centred change.[690] [691] A Lancet (2025) paper emphasised the need for long-term individualised approaches and cautioned against remission being viewed as a 'static endpoint'.[692] Author Professor Kamlesh Khunti noted that:[693]

> *By re-evaluating current definitions, acknowledging the progressive nature of type 2 diabetes, and embracing individualised approaches to glycaemic control, remission can be redefined as a dynamic continuum rather than a static endpoint.*
>
> *Type 2 diabetes remission could be viewed as a spectrum, from medication-supported to drug-free states. This more flexible and inclusive approach would better reflect real-world care and make remission a more relevant outcome in both clinical practice and research.*
>
> *Sustained control of hyperglycaemia, along with reducing adiposity [body fat] – whether through surgical, medical, or lifestyle interventions – could be more relevant than remission itself.*

Deprescribing can be difficult due to the fear of negative consequences, lack of knowledge relating to choices on how and the timing of deprescribing, and the systemic barriers which encourage prescribing but

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[687] Murdoch C, Unwin D, Cavan D *et al* (2019). Adapting diabetes medication for low-carbohydrate management of type 2 diabetes: a practical guide. *British Journal of General Practice* 69(684): 360-361. DOI: 10.3399/bjgp19X704525
[688] Scott IA, Hilmer SN, Reeve E, *et al.* (2015) Reducing inappropriate polypharmacy: the process of deprescribing. *JAMA Intern Med.* 175:827-34. DOI:10.1001/jamainternmed.2015.0324 pmid:25798731
[689] Davies MJ, Aroda VR, Collins BS, *et al.* (2022) Management of Hyperglycemia in Type 2 Diabetes, 2022. A Consensus Report by the American Diabetes Association (ADA) and the European Association for the Study of Diabetes (EASD). *Diabetes Care*. 2022 Nov 1;45(11):2753-2786. DOI: 10.2337/dci22-0034.
[690] Wheatley SA, Deakin TA, Arjomandkhah NC, *et al* (2021) Low-carbohydrate Dietary Approaches for People With Type 2 Diabetes—A Narrative Review. *Frontiers in Nutrition* vol: 8 year: 2021 doi: 10.3389/fnut.2021.687658
[691] Walker L, Smith N, Delon C (2021). Weight loss, hypertension and mental well-being improvements during COVID-19 with a multicomponent health promotion programme on Zoom: a service evaluation in primary care: *BMJ Nutrition,* Prevention & Health 2021;4. DOI: 10.1136/bmjnph-2020-000219
[692] Khunti K, Papamargaritis D, Aroda VR *et al*. (2025) Re-evaluating the concept of remission in type 2 diabetes: a call for patient-centric approaches. *The Lancet Diabetes & Endocrinology*, 13(7): 615 - 634
[693] University of Leicester News (June 18 2025). Experts urge caution against overemphasis on type 2 diabetes remission. https://le.ac.uk/news/2025/june/diabetes-remission-patients-leicester

fail to support deprescribing or acknowledge the problem of multimorbidity and inappropriate polypharmacy. [694]

11. WHOLE OF SYSTEM REFORM - KEYS TO SUCCESS:

(a) Respecting Individual Sensitivity to Dietary Interventions.

Current New Zealand guidelines recommend a cumulative intake of 5-8 servings of cereal a day, 2 servings of carbohydrate-based vegetables a day (e.g. potatoes), 2-4 servings of fruit, while acknowledging that people will normally have one sweet-dessert serving a day.

The Unwin team recognised that an emphasis on dietary carbohydrates initially created confusion, as both health professionals and patients found it difficult to judge the extent to which non-refined carbohydrate foods could raise blood glucose. In response, David Unwin and colleagues subsequently adapted existing glycaemic index values [695] [696] into 'teaspoon-of-sugar equivalents' to illustrate the likely post-prandial glucose impact of common foods.[697] This approach was piloted on patients and a cohort of twenty doctors.

The individual glycaemic response, and Individual insulin sensitivity (the gradual loss of insulin sensitivity in muscle, liver, and fat tissue) or stress on pancreatic insulin-producing cells (β-cell stress) is an important consideration to support patient health. The potential for food addiction that is associated with repetitive exposures to carbohydrates, and which is associated with many psychiatric conditions, is another factor which shapes how clinicians and health coaches address patient needs.

Explanatory charts have since been released to guide people to understand the sugar equivalent of one slice of brown bread or a serving of white rice.[698]

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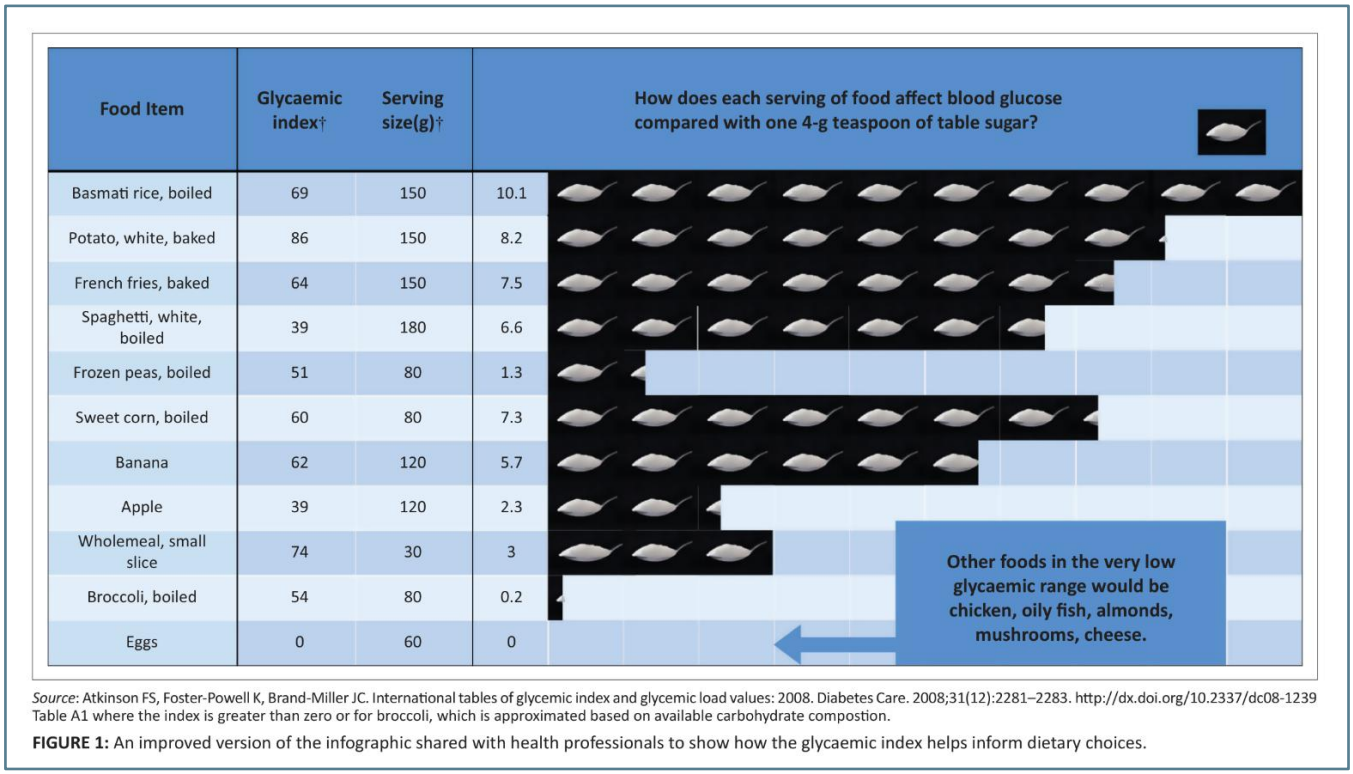
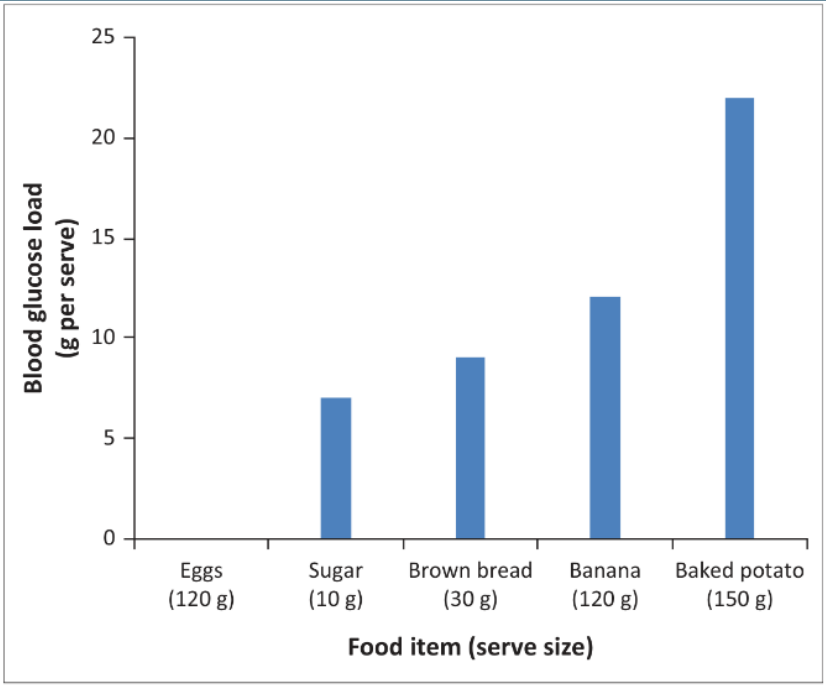
[694] Hung A, Kim Y H, Pavon J M. (2024) Deprescribing in older adults with polypharmacy *BMJ*.
[695] Atkinson FS, Foster-Powell K, Brand-Miller JC. (2008) International tables of glycemic index and glycemic load values. *Diabetes Care*. 2008;31(12):2281–2283. DOI:10.2337/dc08-1239
[696] Jenkins DJ, Wolever TM, Taylor RH, *et al*. 1981. Glycemic index of foods: A physiological basis for carbohydrate exchange. *Am J Clin Nutr*. 34(3):362–366.
[697] Unwin D, Haslam D, Livesey G (2016) It is the glycaemic response to, not the carbohydrate content of food that matters in diabetes and obesity: The glycaemic index revisited. *Journal of Metabolic Health | Journal of Insulin* Resistance: 1 (1)a8, DOI: 10.4102/jir.v1i1.8
[698] Public Health Collaboration UK. Dr David Unwin's Sugar Infographics. https://phcuk.org/sugar/



Source: Atkinson FS, Foster-Powell K, Brand-Miller JC. International tables of glycemic index and glycemic load values: 2008. Diabetes Care. 2008;31(12):2281–2283. http://dx.doi.org/10.2337/dc08-1239 Table A1 where the index is greater than zero or for broccoli, which is approximated based on available carbohydrate compostion.

FIGURE 1: An improved version of the infographic shared with health professionals to show how the glycaemic index helps inform dietary choices.

Figure 13. Unwin D, Haslam D, Livesey G (2016) It is the glycaemic response to, not the carbohydrate content of food that matters in diabetes and obesity: The glycaemic index revisited. *Journal of Insulin Resistance*.



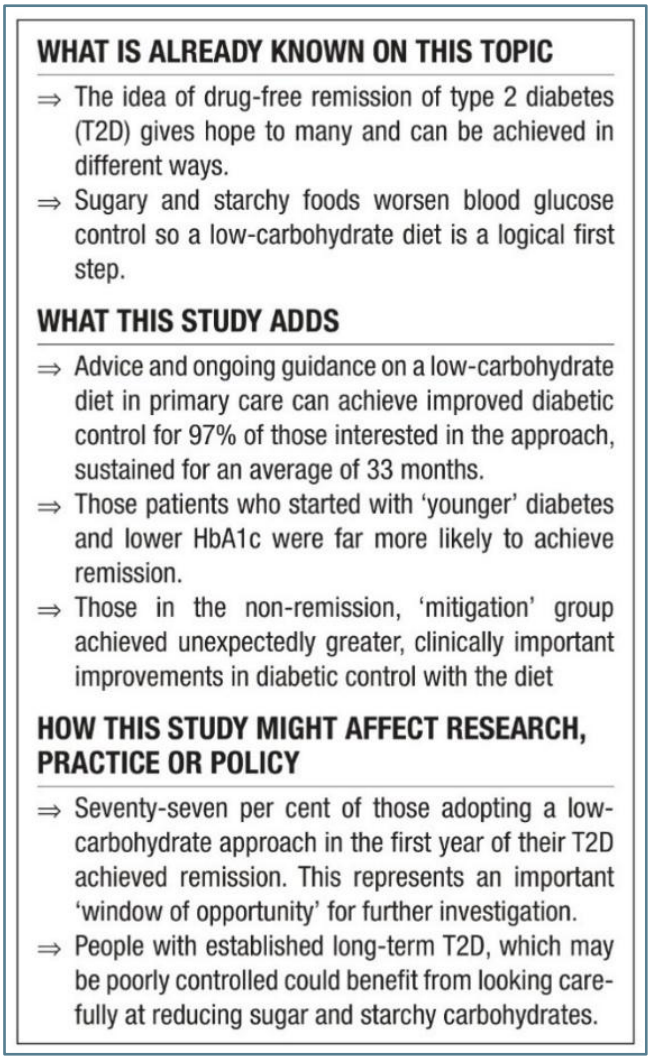
Source: Atkinson FS, Foster-Powell K, Brand-Miller JC. International tables of glycemic index and glycemic load values: 2008. Diabetes Care. 2008;31(12):2281–2283. http://dx.doi.org/10.2337/dc08-1239

Data are from the International tables of glycaemic index and glycaemic load.

FIGURE 2: Blood glucose load* per serve size of food item (*glycaemic load = GI × carbohydrate amount g per serve/100). Sugar is table sugar consumed in water as a drink.

Figure 14. Unwin D, Haslam D, Livesey G (2016) It is the glycaemic response to, not the carbohydrate content of food that matters in diabetes and obesity: The glycaemic index revisited. *Journal of Insulin Resistance*.

A follow-up 2023 paper by David Unwin and colleagues emphasised the importance of an individualised approach:[699]



WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ The idea of drug-free remission of type 2 diabetes (T2D) gives hope to many and can be achieved in different ways.

⇒ Sugary and starchy foods worsen blood glucose control so a low-carbohydrate diet is a logical first step.

WHAT THIS STUDY ADDS

⇒ Advice and ongoing guidance on a low-carbohydrate diet in primary care can achieve improved diabetic control for 97% of those interested in the approach, sustained for an average of 33 months.

⇒ Those patients who started with 'younger' diabetes and lower HbA1c were far more likely to achieve remission.

⇒ Those in the non-remission, 'mitigation' group achieved unexpectedly greater, clinically important improvements in diabetic control with the diet

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ Seventy-seven per cent of those adopting a low-carbohydrate approach in the first year of their T2D achieved remission. This represents an important 'window of opportunity' for further investigation.

⇒ People with established long-term T2D, which may be poorly controlled could benefit from looking carefully at reducing sugar and starchy carbohydrates.

'Similar to many healthcare interventions, one size does not fit all, and individualisation needs to be considered. This may mean that a mixture of different levels of follow-up intensity and mode of delivery (virtual compared to face to face) is likely to be necessary to maximise remission rates, with a more blended approach being taken.

...patients who would like to achieve T2DM remission should be offered a 'menu' of options with respect to educational and dietary approaches if they wish to attempt to achieve remission. This may be key to driving forward remission in a primary care setting.'

Figure 15. Unwin D, Delon C, Unwin J, et al. What predicts drug-free type 2 diabetes remission? Insights from an 8- year general practice service evaluation of a lower carbohydrate diet with weight loss. *BMJ Nutrition, Prevention & Health*

Further studies assessing the safety of a higher protein and low-carbohydrate ketogenic approaches have added to the findings of the Unwin model and demonstrated the safety of increasing dietary fats and proteins relative to carbohydrates.[700] [701]

As people have vastly different temperaments, digestive tracts and genetic and epigenetic factors, dietary tolerances, clinicians and health coaches increasingly recommend a graduated approach to removing refined products from the diet.

Considerations for patients, doctors and health practitioners when reducing dietary carbohydrates relate to concurrent inflammatory risks, including gastrointestinal conditions and food allergies. Individuals may have difficulty digesting particular proteins such as lectins or cereal gluten, and these can aggravate existing digestive symptoms.

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[699] Brown A, McArdle P, Taplin J, Unwin D, Unwin, J, *et al.* (2022). Dietary strategies for remission of type 2 diabetes.

[700] Lin, S.-P.; Chen, C.-M.; Chiu, S.-H. *et al* (2025) Associations of Dietary Protein Intake and Amino Acid Patterns with the Risk of Diabetic Kidney Disease in Adults with Type 2 Diabetes: A Cross-Sectional Study. *Nutrients* 17:2168. DOI: 10.3390/nu17132168

[701] Harvey CJDC, Schofield GM, Zinn C, Thornley S. (2019). Effects of differing levels of carbohydrate restriction on mood achievement of nutritional ketosis, and symptoms of carbohydrate withdrawal in healthy adults: A randomized clinical trial. *Nutrition.* 2019;67-68S:100005. DOI: 10.1016/j.nutx.2019.100005.

For some patients, rapid shifts in macronutrient balance may also unmask underlying gut-microbiome disturbances, exacerbate irritable-bowel–type symptoms, or interact with pre-existing intolerances (e.g., lactose, FODMAPs, or histamine sensitivity). Clinicians therefore need to assess digestive tolerance, inflammatory history, and individual variability when recommending lower-carbohydrate approaches. [702] [703] [704] [705] [706]

Harvard-trained psychiatrist Dr Georgia Ede has found this to be the case for many years, by adopting a graduated dietary nutrition approach with the dual goal of optimising brain health and reducing inflammatory responses, in her U.S. based clinical psychiatric practice. [707] Ede refers to her graduated approach as a 'quiet' approach, which involves reducing dietary ingredients understood to be inflammatory, watching for improvements, and continuing to withdraw ingredients while maintaining a nutrient dense diet, to increase the potential for remission of psychiatric conditions. [708] Ede has reported that healthy fats and grass-fed (rather than feed-lot fed) meat may form an underestimated dietary component of healthy wholefood diets, having found that fewer patients tend to experience adverse inflammatory reactions on these macronutrients.[709]

Doctors and health care professionals emphasise that dietary changes to manage gut-based inflammation are not necessarily permanent, but may be part of a flexible, stepped process to identify inflammatory drivers and support healing in the digestive tract.

(b) Food Addiction Counselling to address Ultraprocessed Food Addiction.

A major, largely unaddressed factor in understanding why many individuals struggle to adopt healthier diets is the role of high-carbohydrate, ultra-processed food addiction. Over the past decade, researchers have characterised 'food addiction' as a measurable construct, developed validated methodologies to assess it, and advanced coherent physiological and behavioural models explaining its drivers. This body of work highlights how highly processed, rapidly absorbed carbohydrate-rich foods can dysregulate appetite, reward pathways and metabolic control, making sustained dietary change considerably more difficult for affected individuals. The scientific basis supporting food addiction as a facet of was discussed in chapter 4.

The financial and health gains from integrating addiction-informed counselling into food and nutrition coaching, whether delivered in medical clinics, community settings or educational environments, may be substantial. Such an approach has the potential to improve long-term adherence, reduce preventable healthcare costs, and better support individuals whose eating patterns are shaped by addictive drivers. A September 2025 Unicef report[710] stated that:

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[702] Cosme-Blanco W, Arroyo-Flores E, Hanadays A. (2020). Food Allergies. *Pediatr Rev* 41(8):403–415. https://doi.org/10.1542/pir.2019-0037https://renaissance.stonybrookmedicine.edu/system/files/Food_Allergies.pdf

[703] Vojdani A, Afar D, and Vojdani E. (2020). Reaction of Lectin-Specific Antibody with Human Tissue: Possible Contributions to Autoimmunity. *J Immun Res.,* DOI: 10.1155/2020/1438957

[704] Bellini M, Tonarelli S, Nagy AG, Pancetti A, Costa F, Ricchiuti A, de Bortoli N, Mosca M, Marchi S, Rossi A. (2020) Low FODMAP Diet: Evidence, Doubts, and Hopes. *Nutrients,* 12(1):148. DOI:10.3390/nu12010148

[705] Roster K, Xie L, Nguyen T *et al.* (2024). Impact of Ketogenic and Low-Glycemic Diets on Inflammatory Skin Conditions. 113:2

[706] Albers J, Kraja G, Eller D, Eck K, McBrian D Main JM. (2022). Assessing the feasibility of using the ketogenic diet in autism spectrum disorder. *JHND,* 36(4):1303-1315. DOI: 10.1111/jhn.13115

[707] Ede G (2024). Change Your Diet, Change Your Mind: A Powerful Plan to Improve Mood, Overcome Anxiety, and Protect Memory for a Lifetime of Optimal Mental Health. *Dimensions*.

[708] Ede G (2024). Change Your Diet, Change Your Mind

[709] Dhakal S, Hossain M, Parajuli S. (2025). The Inclusion of Red Meat in Higher-Quality Diets Supports Nutritional Adequacy, Microbial Diversity, and Mental Health With No Observed Adverse Effects. *Current Developments in Nutrition*. 9(2)106040. DOI: 10.1016/j.cdnut.2025.106040

[710] United Nations Children's Fund (UNICEF), Feeding Profit. How food environments are failing children. Child Nutrition Report 2025, UNICEF, New York, September 2025

The cost of inaction for children, adolescents, families, societies and economies is immense. Unhealthy diets increase the risk of overweight, obesity and other cardiometabolic conditions in children and adolescents, including high blood pressure, elevated blood glucose and abnormal blood lipid levels. These health problems can persist into adult life, increasing the risk of non-communicable diseases, including type 2 diabetes, cardiovascular disease and some cancers. Overweight and obesity are also associated with low self-esteem, anxiety and depression among children and adolescents. Parents bear the emotional toll of their children's mental health challenges and the financial strain of higher medical expenses and lost income to care for them. Economies throughout the world are already struggling with escalating health care costs and reduced workforce productivity because of rising overweight and obesity.

The relevance for coaching programmes that address high-carbohydrate ultraprocessed food addiction was recently highlighted in a small trial which followed the outcomes of clinics in North America, Sweden and the U.K. The programs consisted of 10–14 weeks of 90–120-min sessions in groups of 11–40 participants.

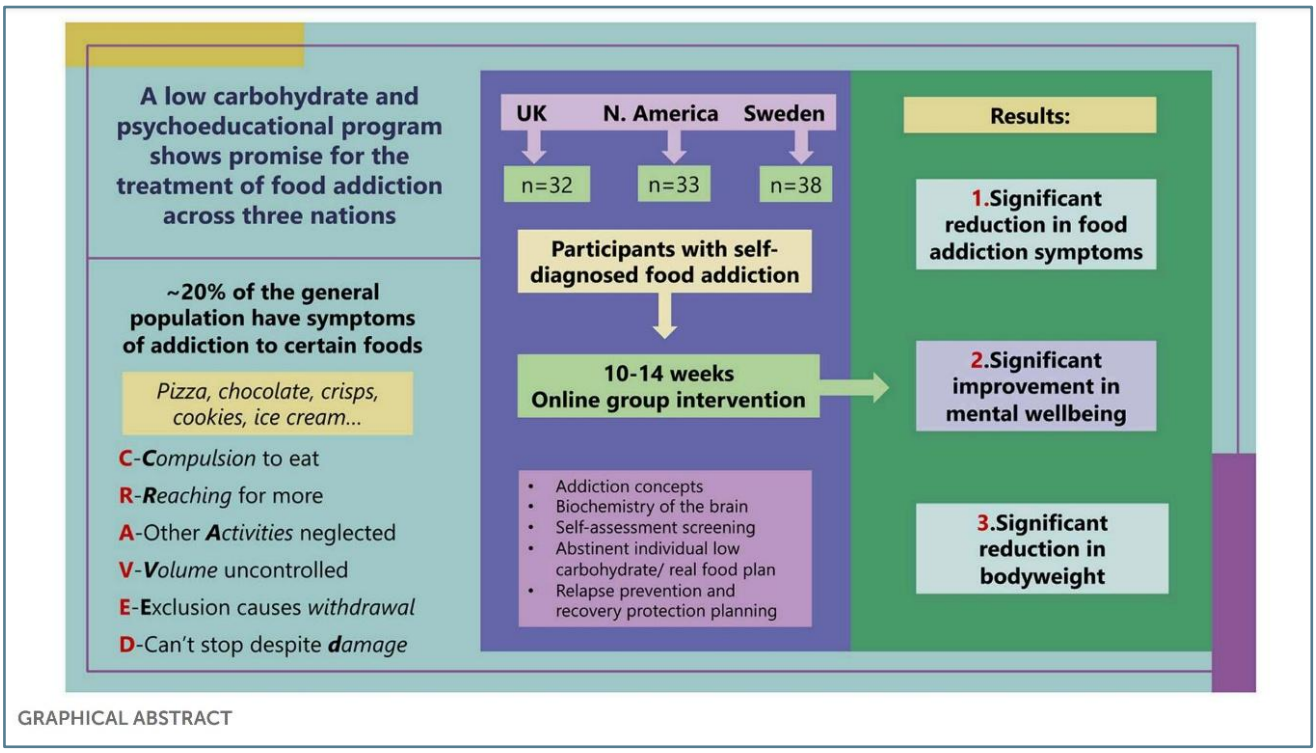

Figure 16. Unwin J, Delon C, Giæver H, Kennedy C, et al. (2022) Low-carbohydrate and psychoeducational programs show promise for the treatment of ultra-processed food addiction. *Front. Psychiatry*

The pre- and post- program outcomes assessed food addiction symptoms measured by the modified Yale Food Addiction Scale 2.0, the ICD-10 symptoms of food related substance use disorder (CRAVED), while mental wellbeing as measured by the short version of the Warwick Edinburgh Mental Wellbeing Scale, [711] and body weight. The ICD-10 symptoms were adapted as the CRAVED screening tool. [712] The researchers

[711] Warwick-Edinburgh Mental Well-being Scale (WEMWBS). User Guide Version 1 (2008). http://www.mentalhealthpromotion.net/resources/user-guide.pdf

[712] Unwin J, Delon C, Giæver H, *et al.* (2022). Low-carbohydrate and psychoeducational programs show promise for the treatment of ultra-processed food addiction. *Front. Psychiatry* 13:1005523. Supplementary materials.

identified a significant reduction in food addiction symptoms, significant improvement in mental wellbeing and a significant reduction in body weight. [713]

Follow-up data was assessed for the North America/Sweden/U.K. trial at 6 and 12 months. The researchers reported:

> *The 12-month follow-up data show significant, sustained improvement in ultra-processed food addiction symptoms and mental well-being. These data are the first long-term follow-up results to be published for a food addiction program.*[714]

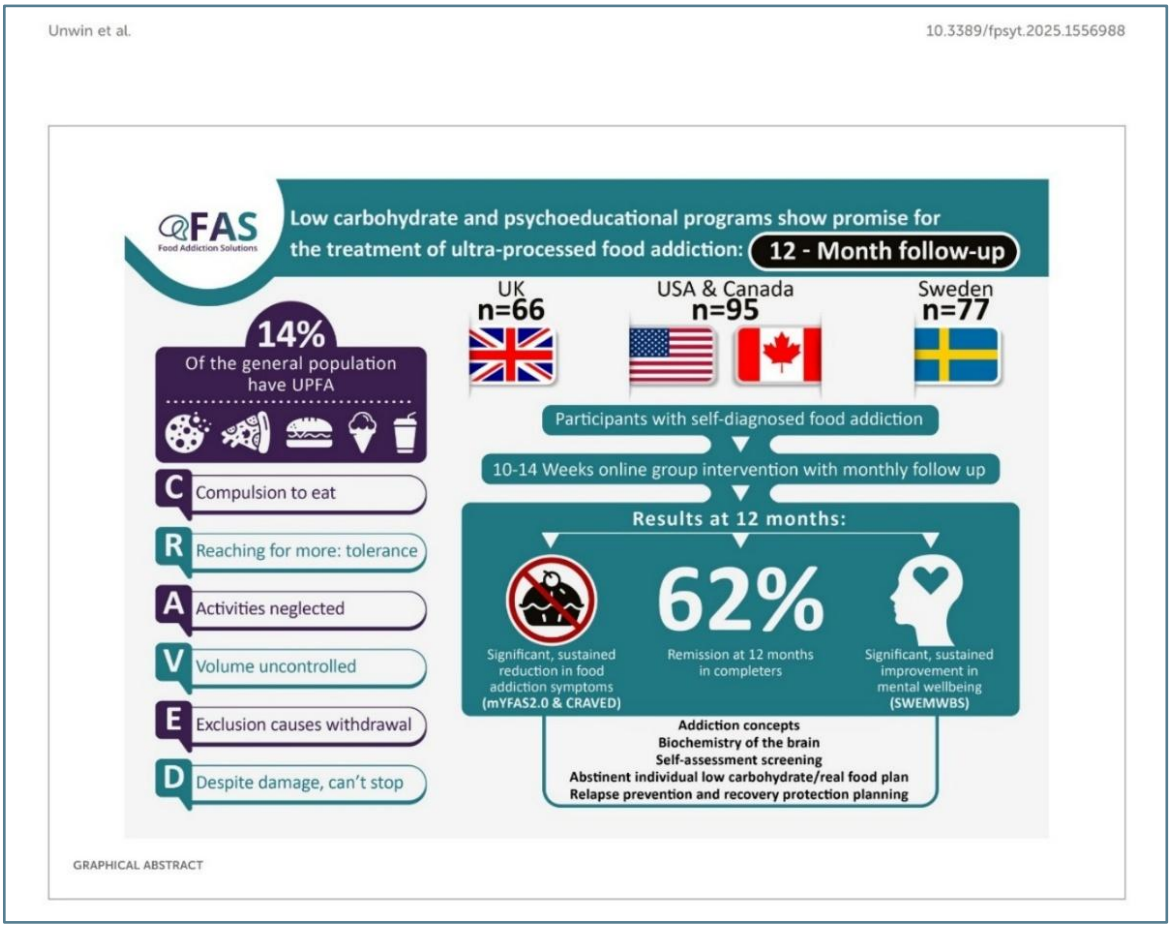

Figure 17. Unwin J, Delon C, Giæver H, Kennedy C,Painschab M, Sandin F, Poulsen CS and Wiss DA (2025) Low-carbohydrate and psychoeducational programs show promise for the treatment of ultra-processed food addiction: 12-month follow-up. *Front. Psychiatry.*

Models to support the integration of programmes that can support patients with ultraprocessed food addiction into general practice have been established. In a 2025 conference presentation[715], Dr Jen Unwin outlined suggestions for treatment of ultraprocessed food addiction:

- Screen using CRAVED then education.

[713] Unwin J, *et al.* (2022). Low-carbohydrate and psychoeducational programs show promise.
[714] Unwin J, Delon C, Giæver H, *et al.* (2025). Low carbohydrate and psychoeducational programs show promise for the treatment of ultra-processed food addiction: 12-month follow-up. *Front. Psychiatry* 16:1556988. DOI: 10.3389/fpsyt.2025.1556988.
[715] Unwin J. The Growing Epidemic of Ultra Processed Food Addiction. Primary Healthcare Lifestyle Conference September 2025. Birmingham UK.

- Working towards abstinence from sugar, flour and processed foods.
- Real food focus. Adequate protein and fat.
- No cheat days and caution with fasting (don't alternate access and restriction).
- No sweeteners (only use in transition if necessary).
- Focus isn't weight loss but stable nutritious eating and neurotransmitter regulation.
- Educate re the addicted brain (+stress management, other activities to replace food rewards).
- Beware alcohol, nicotine, caffeine, (one disease, many outlets).
- Nuts, cheese/dairy with caution and eliminate if cravings persist.
- Ongoing peer support via online groups.

(c) Technologies to support patient knowledge: Continuous glucose monitoring (CGM) devices.

Continuous glucose monitoring (CGM) devices, or sensors are worn on the abdomen or the back of the arm and continuously measure glucose levels, usually in interstitial fluid (the clear fluid that surrounds cells). Glucose diffuses from blood, into the interstitial space where the sensor sits. Real-time glucose monitoring using CGM sensors enable the user to immediately see the response in blood glucose following food consumption, acting as a feed-back mechanism to support patient change. CGM's may be a powerful, low-cost tool for use in patient support and health coaching (contact and non-contact) to assist people with glycaemic control and shift dietary habits over the longer term.[716] [717] [718]

Pharmac provides CGMs to people with T1DM, permanent neonatal diabetes, some types of 'monogenic diabetes with insulin deficiency, type 3c diabetes and some atypical inherited forms of diabetes. Pharmac provides funding for the Dexcom One and FreeStyle Libre 2 or 2 Plus standalone CGMs.[719] People with T2DM do not have access to a Pharmac funded CGM. Diabetes organisations in Australia, the UK and Canada have campaigned for expanded access to subsidised CGM sensors.[720] The U.K. based National Institute of Clinical Excellence (NICE) has recommended CGM technology for children and young people living with T2DM.[721]

Some people who would benefit from a CGM device choose not to use one because of the associated clinical oversight. As a long-term T1DM individual observed to PSGRNZ, patients are acutely aware that a range of health professionals, including their general practitioner, clinical nurse specialists (CNS) and/or endocrinologist, can view their glucose data. This creates the possibility of being asked, 'What did you eat that day?' and of feeling subject to continual surveillance of dietary habits. The implication of misdemeanour or non-compliance is neither welcome nor benign; many experience it as intrusive.

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[716] Ahn YC, Kim YS, Kim B, *et al.* (2023). Effectiveness of Non-Contact Dietary Coaching in Adults with Diabetes or Prediabetes Using a Continuous Glucose Monitoring Device: A Randomized Controlled Trial. *Healthcare* (Basel). 11(2):252. DOI: 10.3390/healthcare11020252.

[717] Richardson KM, Jospe MR, Bohlen LC, *et al.* (2024). The efficacy of using continuous glucose monitoring as a behaviour change tool in populations with and without diabetes: a systematic review and meta-analysis of randomised controlled trials. *Int J Behav Nutr Phys Act.* 21(1):145. DOI: 10.1186/s12966-024-01692-6.

[718] Kumbara AB, Iyer AK, Green CR. (2023). Impact of a Combined Continuous Glucose Monitoring–Digital Health Solution on Glucose Metrics and Self-Management Behavior for Adults With Type 2 Diabetes: Real-World, Observational Study. *JMIR Diabetes* 8:e47638, DOI: 10.2196/47638.

[719] Pharmac. Continuous glucose monitors (CGMs). https://www.pharmac.govt.nz/news-and-resources/cgms-and-insulin-pumps/continuous-glucose-monitors-cgms

[720] Diabetes Australia (2024). Position Statement. Equitable Access to Diabetes Technology.

[721] NICE (May 2023) Diabetes (type 1 and type 2) in children and young people: diagnosis and management. NICE guideline. Reference number:NG18 Published: 01 August 2015 Last updated: 11 May 2023.

However, glucose spikes can occur independently of carbohydrate intake, yet a remote clinician monitoring the data cannot easily distinguish between physiological and dietary causes.[722] [723] Clinical inferences may be inaccurate, and it is plausible that a patient could be viewed as non-compliant when they have done nothing wrong.

Low-cost, over-the-counter CGM sensors are becoming more widely available, but privacy concerns remain. At present, there is no inexpensive CGM that guarantees data is never transmitted to a company or healthcare system. Users who wish to keep their information entirely private may be able to do so only if the device supports a standalone reader and they avoid connecting it to the internet or disable all wireless functions.

The increasing body of case studies using CGM sensors in the scientific literature, provides an evidence base to incentivise adoption by groups and individuals.[724] [725] [726] Large language models (artificial intelligence) are then able to use this data to improve diabetes care[727] however issues relating to surveillance, ethics and oversight, and public use of data will remain.

Current evidence suggests that the use of CGM sensors results in superior outcomes, as compared to standard care, in improving glucose control in patients.[728] [729] Health coaching in combination with the use of CGM devices for patients with sub-optimally controlled T2DM has been demonstrated to improve glycaemic control.[730] Studies show that for people who opt out of standard care, non-insulin treated groups have superior gains in glycaemic control with the use of CGM sensors.[731]

People have the opportunity to become more discerning in their dietary intake when they can directly associate their current carbohydrate intake with their corresponding blood glucose levels. CGM sensors can be used across all diabetes populations, including for people with type 1 diabetes, type 2 diabetes (T2D), and gestational diabetes and to assess neonatal risk.[732] Recommendations for the clinical use of CGM devices have been published. Education is required to ensure that CGM is undertaken correctly and

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[722] Avner S, Robbins T. (2025). A Scoping Review of Glucose Spikes in People Without Diabetes: Comparing Insights from Grey Literature and Medical Research. *Clinical Medicine Insights: Endocrinology and Diabetes.* 2025;18. DOI:10.1177/11795514251381409.

[723] Hantzidiamantis PJ, Awosika AO, Lappin SL. (2025) Physiology, Glucose. [Updated 2024 Apr 30]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing.

[724] Unger J and Franco DR. (2023). Practical Application of Continuous Glucose Monitoring in Clinical Practice: Case Studies. *Diabetes Technology & Therapeutics. 25:S3.* DOI: 10.1089/dia.2023.0080.

[725] Charleer S, Mathieu C, Nobels F, *et al.* (2018). RESCUE Trial Investigators, Effect of Continuous Glucose Monitoring on Glycemic Control, Acute Admissions, and Quality of Life: A Real-World Study, *The Journal of Clinical Endocrinology & Metabolism,* 103(3):1224–1232, DOI: 10.1210/jc.2017-02498.

[726] Rodbard D (2017). Continuous Glucose Monitoring: A Review of Recent Studies Demonstrating Improved Glycemic Outcomes. *Diabetes Technology & Therapeutics*. 19:S3. DOI: 10.1089/dia.2017.003

[727] Healey, E., Tan, A.L.M., Flint, K.L. *et al.* (2025). A case study on using a large language model to analyze continuous glucose monitoring data. *Sci Rep* 15, 1143 DOI: 10.1038/s41598-024-84003-0.

[728] Di Molfetta S, Caruso I, Cignarelli A. *et al.*(2023). Professional continuous glucose monitoring in patients with diabetes mellitus: A systematic review and meta-analysis. *Diabetes obesity and Metbolism*.25(5):1301-1310.

[729] Jancev, M., Vissers, T.A.C.M., Visseren, F.L.J. *et al.* (2024). Continuous glucose monitoring in adults with type 2 diabetes: a systematic review and meta-analysis. *Diabetologia* 67:798–810. DOI: 10.1007/s00125-024-06107-6.

[730] Griauzde DH, Ling G, Wray D, *et al*. (2022). Continuous Glucose Monitoring With Low-Carbohydrate Nutritional Coaching to Improve Type 2 Diabetes Control: Randomized Quality Improvement Program. *J Med Internet Res.* 24(2):e31184. DOI: 10.2196/31184.

[731] Ferreira ROM, Trevisan T, Pasqualotto E, *et al.* (2024). Continuous Glucose Monitoring Systems in Noninsulin-Treated People with Type 2 Diabetes: A Systematic Review and Meta-Analysis of Randomized Controlled Trials. *Diabetes Technology & Therapeutics*. 26:4. DOI: 10.1089/dia.2023.0390.

[732] Chisnoiu, T., Balasa, A. L., Mihai, L., *et al.* (2023). Continuous Glucose Monitoring in Transient Neonatal Diabetes Mellitus—2 Case Reports and Literature Review. *Diagnostics*, *13*(13), 2271. DOI: 10.3390/diagnostics13132271.

that data interpretation is accurate, and devices must be checked to ensure that they are correctly calibrated.[733]

(d) Technologies to support patient knowledge: Breath Ketone Sensors.

Ketogenic diets are a subset of low-carbohydrate diets. By markedly reducing carbohydrate intake, these diets induce a metabolic state of nutritional ketosis, in which the body shifts its primary energy source from glucose to fatty acids. The liver then produces ketone bodies. Typically, this is achieved by restricting carbohydrate intake to below approximately 50 g per day, consuming protein in moderate amounts to avoid inhibiting ketosis, and allowing fat intake ad libitum to satiety.[734] [735] [736]

The primary ketone bodies produced by the liver comprise acetoacetate, β-hydroxybutyrate (BHB) and acetone. Acetone is slightly volatile, it can turn into a gas and leave the body through the lungs and be recorded on the breath. Breath acetone roughly correlates with circulating ketone bodies (predominantly BHB). Breath acetone is being used to monitor trends (when used consistently) but may struggle to deal with acute changes. These devices are not a substitute for blood tests, which remain required for clinical ketogenic use (such as for treatment of epilepsy).[737] [738]

Breath-ketone monitors can non-invasively detect and quantify ketone levels via exhaled breath. Commercial development of breath-based ketone-sensing technologies is ongoing, with several prototype and early-market devices already available. As sensor sensitivity and calibration improve, these tools may eventually become accurate enough to support clinical applications, including the diagnosis or monitoring of diabetes mellitus.[739]

(e) Technologies to support patient knowledge: Digital Apps.

Digital health applications that adhere to current dietary guideline recommendations may support improvements in glycaemic control, cardiovascular risk factors, and diabetes remission, with secondary benefits including weight loss, increased physical activity, and improved mental wellbeing. The GroAus/Gro Health app has demonstrated clinically relevant reductions in body weight and blood glucose, including reductions in, or discontinuation of, diabetes medications and instances of diabetes remission. [740] However, the app does not present itself as a carbohydrate-reduction programme in its outward-facing materials and is framed as general lifestyle coaching aligned with national dietary guidance. In the absence of explicit attention to cumulative carbohydrate intake and the relative balance of fat and protein

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[733] Grunberger G, Sherr J, Allende M, *et al.* (2021). American Association of Clinical Endocrinology Clinical Practice Guideline: The use of advanced technology in the management of persons with diabetes mellitus. *Endocr Pract* 27(6):505–537. DOI: 10.1016/j.eprac.2021.04.008.

[734] Phillips MC. (2022). Metabolic strategies in healthcare: a new era. *Aging Dis.*13:655–72. DOI: 10.14336/AD.2021.1018.

[735] Athinarayanan SJ, Hallberg SJ, McKenzie AL, *et al*. (2020). Impact of a 2-year trial of nutritional ketosis on indices of cardiovascular disease risk in patients with type 2 diabetes. *Cardiovasc Diabetol*. 19:208. DOI: 10.1186/s12933-020-01178-2

[736] Volek JS, LaFountain RA, Dituro P. (2019). Extended ketogenic diet and physical training intervention in military personnel. *Mil Med.* 184:199–200. DOI: 10.1093/milmed/usz184.

[737] Marfatia K, Ni J Preda V, & Nasiri N. (2025). Is Breath Best? A Systematic Review on the Accuracy and Utility of Nanotechnology Based Breath Analysis of Ketones in Type 1 Diabetes. *Biosensors,* 15(1):62. DOI:10.3390/bios15010062

[738] Bastide GMGBH, Remund AL, Oosthuizen DN, *et al.* (2023). Handheld device quantifies breath acetone for real-life metabolic health monitoring. *Sens Diagn.* 2(4):918-928. DOI: 10.1039/d3sd00079f.

[739] Wang W, Zhou W, Wang S, *et al*. (2021). Accuracy of breath test for diabetes mellitus diagnosis: a systematic review and meta-analysis. *BMJ Open Diabetes Research & Care.*;9:e002174. DOI: 10.1136/bmjdrc-2021-002174

[740] Shahidi M, deCourten B, Glennan J, *et al*. (2025) Implementing a Scalable, personalised, behaviour Change digitAL hEalth programme in primary care for type 2 diabetes treatment: the SCALE cluster- randomised study protocol. BMJ Open 15:e101531. DOI:10.1136/bmjopen-2025-101531.

macronutrient groups, such digital interventions may offer utility for some individuals but remain relatively underpowered to address broader population-level metabolic needs.

(f) The Question of Protein Choice.

Ministry of Health health-promotion materials tend to under-emphasise the role of dietary protein. Public messaging typically centres on vegetables and fruit, with comparatively little attention to the macronutrients protein and fat. Current health-promotion literature makes no substantive link between protein quality and mental health, despite protein quality referring to a food's capacity to meet human requirements for essential amino acids (EAAs) and nitrogen. Adequacy is determined by whether intake supports key metabolic endpoints, including nitrogen balance, amino-acid balance and isotope-oxidation measures.[741]

In both New Zealand and Australia, NHMRC macronutrient reference values for protein are derived largely from data identifying levels that prevent frank deficiency, rather than levels that optimise function across age, developmental stage or sex. Much of the evidence informing these reference values for adults, pregnant women, adolescents and children predates 2002, and upper intake limits primarily reflect population-distribution data rather than physiological benchmarks for optimal metabolic performance.

Protein requirements for pregnant women, children and adolescents are framed around growth, weight gain and skeletal maintenance, rather than the broader metabolic and stress-related demands characteristic of these life stages. This leaves important questions regarding optimal protein intake, particularly the role of EAAs in neurocognitive development and mental health, largely unaddressed in current policy guidance. [742]

The optimum macronutrient balance for health and prevention of obesity, T2DM and mental illness is not discussed by the NHMRC and the threshold for 'sufficiency' may be set far below what may be optimal for neurotransmitter homeostasis and mental health resilience. Estimated average requirements reflect approximations based on the NHMRC data:

> *Diets as low as 10% of energy from protein will provide the protein required for maintenance and replacement of body tissues and for the necessary functional and structural proteins required by the body, intakes at or above 15% protein appear to be required for ensuring that the EARs for micronutrients are met, particularly for people with energy requirements below about 15,000 kJ/day.*

The HRMRC discussion does not highlight the critical role of dietary protein in neurological health. The brain is heavily dependent on substances that are disproportionately high in meat proteins, including iron, B vitamins, and amino acids.[743] Iron and B vitamins are indispensable to neurotransmitter synthesis[744], and knowledge concerning the role of amino acids in the immune system, in gut-brain axis signalling, and neurotransmitter synthesis has accelerated. Amino acids are not simply required to build neurotransmitters but are required by the enzymes needed to build the neurotransmitters and the receptors that receive their messages.[745] [746]

- **Essential amino acids:** histidine, isoleucine, leucine, lysine, methionine, phenylalanine, threonine, tryptophan and valine.

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[741] Matthews JJ, Arentson-Lantz EJ, Moughan PJ *et al.* (2025). Understanding Dietary Protein Quality: Digestible Indispensable Amino Acid Score and Beyond. *J Nutrition,* DOI: 10.1016/j.tjnut.2025.07.005

[742] NHMRC (2017). Nutrient Reference Values for Australia and New Zealand. Pages 26-30

[743] NHMRC (2017). Nutrient Reference Values for Australia and New Zealand. Pages 26

[744] Kumar, Sumit, et al. (2022) Nutrition, Neurotransmitters, and Behavior. Nutrition and Psychiatric Disorders. Singapore. Springer Nature Singapore, 2022. 89-108.

[745] Ede G (2024). Change Your Diet, Change Your Mind. See discussion Chapter 4.

[746] Dalangin R, Kim A, Campbell RE. (2020). The Role of Amino Acids in Neurotransmission and Fluorescent Tools for Their Detection. International Journal of Molecular Sciences. 21(17):6197. DOI:10.3390/ijms21176197

- **Conditionally essential:** arginine, cysteine, glutamine, glycine, proline, selenocysteine, tyrosine, taurine.
- **Nonessential:** Alanine, aspartic acid, asparagine, glutamate and serine.

Nearly all animal proteins contain all amino acids, whereas plant proteins tend to be lower in amino acids.

> *Animal-derived proteins generally contain sufficient amounts of each EAA (relative to daily EAA requirements), making them complete protein sources. In contrast, plant-derived proteins often lack sufficient amounts of 1 or more EAAs, making them incomplete protein sources. Cereals, grains, and seeds tend to be proportionally low in lysine, whereas legumes and vegetables are proportionally low in the sulfuric amino acid, methionine. Most plant-derived proteins also contain limited amounts of valine and isoleucine.* [747]

Data used to recommend macronutrient ratios is derived from studies showing population averages where intake tails off in the upper range. The U.S. and Canada established an acceptable macronutrient range in 2002, recommending an:

> *upper limit of 35% energy from protein. However, there is very limited information about the longer-term effects of diets in which protein provides >25% energy. Average usual intakes within the range 25-35% energy from protein are not reported in western populations, even in athletes.* [748]

The NHMRC has not established a formal upper limit for protein intake due to insufficient evidence. Instead, it notes a recommended upper boundary of approximately 25% of total energy intake from protein, with the justification located in the 'Chronic Disease' section of the guidelines.[749] That section frames its reasoning around an increasingly sedentary population engaged in less physically demanding work. However, it does not address the protein requirements needed to prevent fatigue, support exercise capacity, or meet the differing metabolic demands associated with age, developmental stage or sex. These considerations remain largely absent from the current guidance.

Vegetables provide relatively low EEAs per portion size. Complementary proteins, such as chickpea and lentil dishes can increase the metabolic availability of individual amino acids, while natto, tempeh, mycoprotein, and soy-based meat alternative (SBMA) products provide ~6–7 g EAAs per 100 g. This is similar to equivalent portions of whole eggs. [750] [751]

People who favour vegetarian or vegan diets must be competent cooks in order to ensure that their levels of protein, B vitamins, iron and essential amino acids do not become depleted, either rapidly during times of high physiological stress or over the longer term. High quality protein is not only reliant on the EAA profile but on the range, digestibility and bioavailability potential of that food. The role of selection of plant protein, processing (including soaking and fermenting) and cooking. [752]

Health coaching may play an important role in supporting vegetarians and vegans to acquire the knowledge and practical skills needed for adequate meal planning, food preparation and cooking, particularly among younger people and women of childbearing age, whose nutritional requirements are more demanding.

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[747] Matthews JJ, Arentson-Lantz EJ, Moughan PJ *et al.* (2025). Understanding Dietary Protein Quality: Digestible Indispensable Amino Acid Score and Beyond. *J Nutrition,* DOI: 10.1016/j.tjnut.2025.07.005
[748] NHMRC (2017). Nutrient Reference Values for Australia and New Zealand. Pages 240
[749] NHMRC (2017). Nutrient Reference Values for Australia and New Zealand. Pages 29
[750] Matthews JJ, Arentson-Lantz EJ, Moughan PJ *et al.* (2025). Understanding Dietary Protein Quality: Digestible Indispensable Amino Acid Score and Beyond. *J Nutrition,* DOI: 10.1016/j.tjnut.2025.07.005
[751] Hertzler SR, Lieblein-Boff JC, Weiler M, Allgeier C. (2020) Plant Proteins: Assessing Their Nutritional Quality and Effects on Health and Physical Function. *Nutrients.* 12(12):3704. DOI: 10.3390/nu12123704
[752] Matthews JJ, Arentson-Lantz EJ, Moughan PJ *et al.* (2025). Understanding Dietary Protein Quality: Digestible Indispensable Amino Acid Score and Beyond. *J Nutrition,* DOI: 10.1016/j.tjnut.2025.07.005

Achieving optimal intakes of protein, B-group vitamins, iron and essential amino acids is generally more straightforward on an omnivorous diet, whereas plant-based diets require more deliberate planning to ensure nutritional adequacy.

From a practical standpoint, animal-source proteins often require minimal preparation to deliver complete essential amino acid profiles. In contrast, many convenient plant-based protein options are either industrially formulated (with preservatives or flavourings) or derived from crops that may routinely be treated with agricultural chemicals under current farming conventions. These factors do not preclude healthy vegetarian or vegan eating, but they do reinforce the value of tailored health coaching to help individuals meet nutrient needs safely and consistently.

People may also face challenges if amino-acid availability is compromised. Deficits in essential amino acids can impair synaptic signalling, and these effects may be amplified by inflammation, metabolic stress or genetic variations that reduce enzyme efficiency. Trauma, chronic stress and psychiatric illness can shift certain amino acids from being 'non-essential' to functionally essential, while high carbohydrate burdens may further reduce circulating amino-acid diversity.

Digestive tract dysfunction can additionally impair protein breakdown and absorption. Whole-food proteins are generally more efficiently digested and utilised in younger adults than in older adults. [753] Bioavailability is influenced by multiple factors, including prior dietary exposure to specific proteins (and therefore gut adaptation), the extent of food processing, and the composition and integrity of the gut microbiota and gastrointestinal lining.[754]

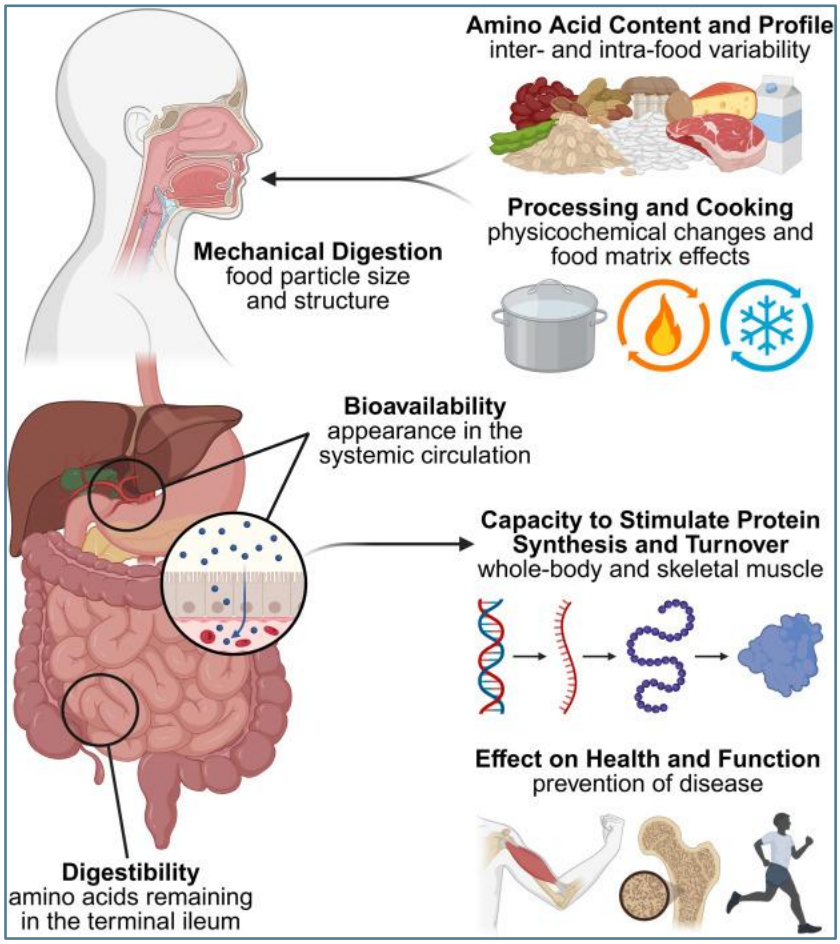

Figure 18. Matthews JJ, Arentson-Lantz EJ, Moughan PJ et al. (2025). Understanding Dietary Protein Quality: Digestible Indispensable Amino Acid Score and Beyond. *J Nutrition*.

[753] Matthews JJ, Arentson-Lantz EJ, Moughan PJ *et al.* (2025). Understanding Dietary Protein Quality: Digestible Indispensable Amino Acid Score and Beyond. *J Nutrition,* DOI: 10.1016/j.tjnut.2025.07.005
[754] Wu S, Bhat ZF, Gounder RS, *et al.* (2022). Effect of Dietary Protein and Processing on Gut Microbiota—A Systematic Review. Nutrients. 14(3):453. DOI: 10.3390/nu14030453

12. WHOLE OF SYSTEM REFORM: *IN BRIEF*

PSGRNZ's proposals for reform involves the implementation of high-level strategic science, regulatory and science system shifts, in addition to community and practitioner led wrap-around policies that directly support people in the short-term to pivot long-term to dietary habits that support metabolic health.

PSGRNZ broadly supports Professor Grant Schofield's proposal:[755]

- Investing in prevention: A minimum 15% of the health budget will be allocated to chronic disease prevention and 5% to mental health services.
- Reforming food policy: Stronger nutrition labelling, reduced unhealthy food marketing, and ultraprocessed food tax measures.
- Reducing medication reliance: Encouraging 'social prescribing' so GPs can refer patients to exercise, nutrition support, and mental health therapy before medication.
- Expanding public health workforce: Training more health coaches and lifestyle medicine experts to support behaviour change.
- Ensuring accountability: A National Health Reform Taskforce with executive powers will monitor progress, report on key health indicators, and adjust strategies as needed

In addition to the Schofield Proposal, PSGRNZ propose the following:

[I] DIET FIRST APPROACHES IN LOCAL COMMUNITIES.

Implement practitioner and community-led dietary approaches that recognise individual susceptibility to hyperglycaemia and hyperinsulinemia in response to high-glycaemic and refined carbohydrate intake (cumulative carbohydrate burden), and that address the challenge of food addiction, which may co-occur with and reinforce chronic high refined carbohydrate intakes.

1. **Formal recognition that so-called prediabetes (HbA1c 39–46 mmol/mol; 5.7–6.4%) is more accurately described as early type 2 diabetes mellitus** (Zinn, 2025). HbA1c values in this range reflect impaired blood glucose regulation and represent a precursor state to metabolic syndrome, conferring increased long-term health risk.

2. **The right to information on the dietary carbohydrate and consequent blood glucose burden for that individual.** From childhood onwards, New Zealanders have the right to be informed of the combinatory role of free sugars and dietary carbohydrates in creating the metabolic conditions which underlie prediabetes, diabetes and which are associated with common chronic metabolic and brain-related conditions. That individual has the right to regular testing to assess that individual's unique predisposition to the risk of unstable blood glucose, elevated triglycerides and elevated insulin.

3. **Right to information and informed consent:** Patients must be provided with clear, comprehensive information about the likely progression of common medication pathways associated with metabolic syndrome, inclusive of diabetes. Informed consent should be explicitly strengthened to ensure that patients understand the potential for a progressive cascade into multimorbidity following diagnoses such as prediabetes and diabetes. This requires that patients are fully briefed on the side effects of medications that are likely to be co-prescribed over the course of treatment for metabolic and psychiatric conditions, including the risks of drug–drug interactions.

4. **Expand health coaching across general practice, integrating a three-pronged approach** (Zinn et al. 2025[756])**:** Whole food, carbohydrate reduction; a health coach, behaviour-change-based delivery

[755] Schofield, G. (March 2025). Health Reform in New Zealand. https://prekure.com/petition/#proposal
[756] See also discussion Part III, above.

approach; and community- or peer-based initiatives to reduce hyperglycaemia and hyperinsulinemia. Health coaches combine holistic and flexible individual- and community-based nutrition education to support patient dietary transitions away from patterns that provoke hyperglycaemia and hyperinsulinemia. Health coaches incorporate food addiction education and counselling to support patients to adopt behavioural and psychological strategies to optimise nutrient intake and health outcomes.

    a. Recent New Zealand findings corroborate with international evidence that the three-pronged health coach model results in meaningful patient outcomes, improves health equity, and reduces medical prescribing. A small number of early-adopter New Zealand primary care practices have integrated qualified health coaches, a model that can be expanded.[757] [758]

    b. Expand PHO health coach services to integrate the three-pronged approach.

    c. Refer all patients with HbA1c 39–46 mmol/mol+ for health coaching to support long-term reduction in chronic elevated blood glucose levels.

5. **Offer subsidised, Pharmac funded continuous glucose monitors (and training) for young people under 25 after diagnosis of prediabetes or diabetes, including T2DM.** Automatic provision for the under 25 age group with the choice of access to a CGM device for an initial six-month period.

6. **Expand care of dental and general practitioner services to young people under 25** (this aligns with the NZDA's call to increase affordability of access to dental care). PSGRNZ echo select proposals by the New Zealand Dental Association (NZDA), *Roadmap Towards Better Oral Health* report which recommended expanding care to young adults and the implementation of dental service models to meet the needs of local communities and high-need population groups.

    a. Free doctors and dental visits to young people under the age of 25.

    b. That pharmacy charges to patients for prescriptions issued by a dentist should be the same as those for prescriptions issued by a medical practitioner in primary care.

    c. That patients attending a dentist should have access to funded laboratory services for histology and routine blood tests on the same basis as primary care.

7. **Offer high-dose multinutrient supplementation as an option as an adjunctive, first-line treatment for a spectrum of psychiatric conditions that would automatically be diagnosed as requiring prescription drugs and health coaching as an integrative wrap-around support framework.** The Hardy DEN product, and future similarly structured products is sufficiently safe to be offered for retail sale as a general nutrient by healthcare practitioners. (Pharmac funding for the under-25 age group and for individuals who receive work and income benefits).

    a. Automatically enrol people eligible for high-dose multinutrient supplementation, in health coaching as wrap-around, clinician led and community enhanced integrative support framework to enhance nutrient intake, address food addiction, and support the remission of metabolic and brain-related parameters for a period of two years.

8. **Re-establish the original Ka Ora, Ka Ako programme.** Ensure that meals are locally produced by community contractors. Amendments may include:

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[757] E.g. Health Coaches Australia and New Zealand Association (HCANZA).

[758] Zinn C, Campbell JL, Fraser L. *et al*. (2025) Carbohydrate Reduction and a Holistic Model of Care in Diabetes Management: Insights from a Retrospective Multi-Year Audit in New Zealand. *Nutrients*.17(24):3953.

a. Review of lunch menus to: (i) ensure meals support optimal brain health; and (ii) substantially reduce high-glycaemic carbohydrate portions, given the strong likelihood that carbohydrates will dominate other meals and snacks throughout the day because they are the most affordable macronutrient.

b. Greater focus on waste reduction and management: implementation of recyclable or compostable packaging options and practices; utilizing more biodegradable packaging materials; improving communication around appropriate waste disposal methods; and enhancing provider's recommendations for sustainable practices (Dey, 2025, p.244).

[II] EDUCATIONAL REFORM

9. **Expand nutrition education across medical training:** Encompassing functional nutrition (including the role of macro- and micronutrients in biological function, metabolic regulation, and the maintenance of cellular and neurobiological systems), including the role of nutrition not only in preventing deficiency, but in supporting health, and in reducing and reversing the biological and inflammatory drivers of chronic metabolic and brain-related illness.
    a. Undergraduate level – with core, assessable nutrition competencies embedded within medical curricula.
    b. Postgraduate level – including structured nutrition education within vocational training programmes and specialist colleges.
    c. Professional organisations – increase the visibility and status of nutrition within professional bodies such as the Royal Colleges, including through formal competencies, accreditation standards, and continuing professional development (CPD) requirements.

10. **Embed nutrition education throughout the school curricula. Improve the quality of nutrition education, incorporating recognition of the carbohydrate-insulin pathway, the specific role of micronutrients in human biological systems and in particular, brain health, and provide food addiction education and counselling alongside other forms of counselling services.**

    a. *Preschool* – food preparation and eating.

    b. *Primary* – Embed stepwise nutrition education across health, science and wellbeing curricula so that students can gain an appreciation of nutrition's role at the level of the mitochondria, the cell, an organ system, the gut microbiome and the brain. Educate children on the difference between craving refined sugars and starches and real (homeostatic) hunger, and the role of protein, fat and fibre in satiety.

    c. *Secondary* – revise curriculums across biology, science and health so that the role of nutrition in sustaining and protecting animal/plant/human health is weighted at least as equivalently as genetic factors. Reintroduce compulsory nutrition and cooking education for years 7-9. Educate children on the difference between craving refined sugars and starches and real (homeostatic) hunger, and the role of protein, fat and fibre in satiety.

    d. *Tertiary* – Increase content quality and pathways for research across health, medical and agricultural sciences. Course content to emphasise the role of nutrients in biological processes from the mitochondria, to cellular, to organ systems and the metabolism. For example, for psychology, nutrition may focus on brain health, for agriculture nutrition may focus on soil health, productivity and fertility, for health sciences and medicine nutrition can consider biochemical pathways to disease and health and the role of dietary nutrition in preventing mental and metabolic disease.

[III] INSTITUTIONAL & REGULATORY REFORM.

11. **Alignment with some aspects of the *Rebalancing our food system* May 2024 report by the Public Health Advisory Committee (PHAC).**[759] This supports increasing access to healthy foods. However, this report aligns with government dietary guidelines. Without a substantial policy shift it is likely that any policy shifts could prioritise access to healthy meat protein and healthy fats.

12. **Expand access to laboratory testing services:** New Zealand's relatively small population size has resulted in a small group of laboratories who undertake the bulk of testing and privately funded testing must not be unduly restricted.

    a. Expand publicly funded nutritional status testing for high-risk groups: vitamin D, vitamin B12, folate (B9), vitamin B6, copper and selenium. This includes the following categories of people diagnosed with a psychiatric and/or neurodegenerative condition: (i) under-25 year olds; and (ii) preconception and pregnant mothers; and (iii) Those with treatment resistant psychiatric illness; - diagnosed with depression, anxiety, schizophrenia, obsessive compulsive disorder, bipolar and/or ADHD; (iii) People diagnosed with dementia/neurodegenerative conditions.

    b. Where a specific clinical pathway exists, expand testing for: (i) MTHFR polymorphism, (ii) CYP450 panel; (iii) Broader HLA safety screening; (iv) Monogenic diabetes (MODY) genetic testing.

    c. Expand high-sensitivity C-reactive protein (hs-CRP) testing. Hs-CRP (>3 mg/L) can be used in routine clinical practice to identify primary prevention individuals at increased inflammatory risk as long as the patient is not acutely ill.

    d. Remove barriers to enable the general public to independently request and self-fund laboratory serum testing directly through their medical practitioner. Access to such testing should not require specialist referral for approval of individual tests or test panels, nor require disclosure of personal information to laboratories beyond that included in the clinician's test request.

13. **Provide Pharmac funding for high dose multinutrient supplements for the under-25 age group and low-income, at-risk groups.**

    a. MoH/Medsafe can reverse their general sale medicine decision for the Hardy's multinutrient product and that product can be generally available as a retail multinutrient supplement from health practitioners. Products with equivalent ingredients must not be classified as a general sale medicine.

    b. Pharmac can fund the Hardys DEN products under the multivitamin preparation category for (i) the under-25 age group; and (ii) preconception and pregnant mothers; and (iii) Treatment resistant psychiatric illness; - diagnosed with depression, anxiety, schizophrenia, obsessive compulsive disorder, bipolar and/or ADHD; (iii) doctors can have discretion to expand use of the DEN product to other categories including for the prevention or slowing of neurodegenerative disorders for a period of two years.

14. **Implement a pathway to regulatory reform that recognises that micronutrients have therapeutic potential and that they can be consumed at upper levels that are safe.** The Medicines Act 1981 does not permit micronutrients to have therapeutic potential. This is not supported by science. The role of higher dose micronutrients has been ignored in government policy. After thirty years of the status quo, a pathway to reform must ensure open and collegial

[759] Public Health Advisory Committee. 2024. Rebalancing our food system. Wellington: Ministry of Health.

scientific and health-based engagement prior to the Ministry of Health taking action to draft legislation. This is to ensure that future legislation does not automatically adopt a toxicological perspective which could then rule important considerations out of scope during select committee consultation processes.

   a. *Regulations can be amended through Orders in Council (secondary legislation):*

      i. The terminology in the Dietary Supplements Regulations 1985 can be amended to replace 'maximum daily dose' with 'recommended daily dose'.

      ii. The Medicines Regulations 1984 Schedule 1, Part 1 can be altered, removing lithium as an exclusively pharmaceutical medication.

15. **Guiding principles for all health legislation:**

   a. Primum non nocere – First do no harm.

   b. Evidence based. This includes (i) regular reviews and public reporting of the changing evidence base for safety and risk, (ii) Including evidence of safety and efficacy by age, gender and health status held by governments and industry and updates in the scientific literature; (iii) the obligation that all medical drug and device information is linked to the trials information and data that are claimed to support the safety and efficacy of the medical drug or device.

   c. Require signed informed consent.

   d. Proportionate. Match regulation to the biological risk.

   e. Prevention through empowerment. People can read and review studies to establish whether dietary changes, dietary supplements and medical drugs and devices are beneficial or risky for them.

[IV] SCIENCE SYSTEM REFORM

16. **Disestablish the Ministry of Business, Innovation and Employments' (MBIE) control over science and technology funding.** The decline of human and environmental health research, research to monitor and evaluate New Zealand resources and infrastructure, and the decline of basic research in agriculture, has mirrored the domestic pivot to prioritise innovation.

17. **Establish a Ministry of Science, Research and Technology.** Overarching principle for research funding revolves around the long-term stewardship, or kaitiakitanga, of New Zealand, her people and environment. Devote fully 50% of New Zealand's science, research and technology budget to public good research. This involves shifting research that demands an innovation output to instead reposition innovation as one element or outcome that is embedded within the research, science and technology platform, rather than the current situation which positions innovation as the north star of New Zealand's research architecture.

18. **Establish a multidisciplinary environmental health institution in a New Zealand region which is tasked to drive chronic disease prevention and remission through the advancement of knowledge relating to the dietary, nutritional and toxic drivers of metabolic and mental illness.** The institution board will include experts in nutrition, metabolism, nutritional psychiatry, nutrigenomics, endocrinology, inflammatory and biomarker assessment, epidemiology, toxicology and diet, who have a demonstrated research record in these sectors relating to chronic disease prevention.

19. **The environmental health institution board will establish the policy and work programme for the institution.** Research, which can complement global research trajectories, will include anthropogenic exposure monitoring and assessment of risks from man-made chemicals, heavy metals and radiation. This includes occupational, household, industrial, urban and agricultural exposures, research to identify the additive and synergistic health risks from the food additives,[760] plastics, electromagnetic field radiation, pesticides, common drugs and low levels of chemicals in drinking water. The work programme will include the review and assessment of optimum micronutrient levels by age, gender and developmental stage. Aims will target increased recognition of health harms from poor diets,[761] [762] improved consumer knowledge through better labelling[763], improved school dietary choices[764] [765] and support the adoption of nutrient-dense diets across the population.[766] [767]

    a. The quality of research will be ensured by rigorous reviews of the independent scientific literature where authorship, research methods and raw data is disclosed; and which take into account human difference (complexity and uncertainty) and biology. Evidence for research and policy can be drawn from structure and function studies to single cases, cohort studies and controlled trials.

20. **The institution will be 50/50 funded by the health budget and the Ministry of Science.** Ministers and political appointees, including chief science advisors will not direct funding trajectories. This institution will be based in Hamilton or Christchurch and affiliated with research across relevant academic institutions.

21. **Environmental health institution to have independent powers to inform New Zealanders.** The Institute will be tasked to independently support and inform communities, hospitals, the education sector and clinical practice to incorporate evidence-based nutritional and dietary education to reduce ultraprocessed food intake, increase wholefood intake, and optimise mental and metabolic health.

22. **Innovation is recategorized as an element of research, not the key driver.** For example, research funding can be allocated to co-design and development of healthy formulated foods with industry,

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[760] Payen de la Garanderie M, Hasenbohler A, Deschamp N, *et al.* (2025). Food additive mixtures and type 2 diabetes incidence: Results from the NutriNet-Santé prospective cohort. PLoS Med 22(4): e1004570. DOI:10.1371/journal. pmed.1004570

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[762] Good KE, Parnarouskis L, Cummings JR, Gearhardt AN (2025). Adapting anti-tobacco messages to ultraprocessed foods: message framing's impact on attitudes toward the food industry. *Obesity.* 33(5):903-914. DOI: 10.1002/oby.24272

[763] Mackay S, Eyles H, Gontijo de Castro T, Young L, Ni Mhurchu C, et al. (2021) Which companies dominate the packaged food supply of New Zealand and how healthy are their products?. PLOS ONE 16(1): e0245225. DOI:10.1371/journal.pone.0245225

[764] Myers I (April 9, 2025). California Assembly committee advances bill to protect schoolchildren from harmful UPF. *EWG News*. https://www.ewg.org/news-insights/news-release/2025/04/california-assembly-committee-advances-bill-protect

[765] Trask S, Thornley S, Sundborn G. (2024). School-based learning about sugary drinks: possibilities and potential for curriculum approaches supporting health promotion in New Zealand. *Health Education Research,* 39(5)475–485. DOI: 39/5/475/7696174

[766] Starck, C.S.; Blumfield, M.; Keighley, T.; *et al.* (2021).Nutrient Dense, Low-Cost Foods Can Improve the Affordability and Quality of the New Zealand Diet—A Substitution Modeling Study. *Int. J. Environ. Res. Public Health18*:7950. DOI: 10.3390/ijerph18157950

[767] Young, L., Kidd, B., Shen, S. *et al.* (2024) Trends in the healthiness and nutrient composition of packaged products sold by major food and beverage companies in New Zealand 2015 to 2019. *BMC Med* **22**, DOI: 10.1186/s12916-024-03567-w

development of screening and assays to identify harmful or toxic formulations. This could drive trust and promote consumer confidence in domestic and export markets. As a part of a recent project to reconfigure ultraprocessed foods to optimise human functioning, a group of researchers proposed a 'Metabolic Matrix' as a principle-based pathway which would revolve around protecting the liver, feeding the gut and supporting the brain.[768]

CONCLUSION: REVERSING SURGING MULTIMORBIDITY WITH 'FANTASTICALLY CHEERFUL MEDICINE'

In conclusion, substantial evidence indicates that current dietary guidelines have not stemmed rising rates of metabolic and mental disorders and, in several respects, may be contributing to the progression of illness. Health is complex, multifactorial, and dynamic. As defined by the World Health Organization:

> *Health is a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity.*[769]

The onset of multimorbidity at earlier ages is strongly associated with poorer long-term outcomes. Rising rates of multimorbidity, alongside equity in medication prescribing, are not proxies for improved wellbeing or quality of life.

This Report demonstrates that New Zealand's health policy has rested on outdated assumptions while nutritional and metabolic science has advanced. Current Ministry of Health positions, echoed by government-aligned organisations, stand in sharp contrast to an expanding body of evidence showing that foundational dietary and supplementary approaches can reverse or mitigate many metabolic and neurological conditions, improving functionality, wellbeing, and quality of life.

Encouragingly, change is already underway. Case and cohort studies consistently show that substantial reductions in refined carbohydrate intake are associated with improvement, remission, and in some cases reversal across a wide range of metabolic and brain-related conditions.[770] [771] There is increasing consensus that compulsive overconsumption of refined carbohydrates, particularly in the form of ultra-processed foods, constitutes a form of substance-use disorder. Health coaching, peer support, and community-based programmes can facilitate dietary change and, in doing so, reduce the burden of metabolic and neurological disorders as well as pharmaceutical dependence.

Dr Jen Unwin, co-partner of the UK clinic that has pioneered approaches to reversing metabolic syndrome, reducing prescribing rates, and improving mental health outcomes, has described counselling, coaching, and support as 'fantastically cheerful medicine'. Yet conventional therapeutic frameworks do not treat long-term dietary change as a clinical intervention in the same way pharmaceutical treatments are regarded.

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[768] Harlan TS, Gow RV, Kornstädt A, Alderson PW and Lustig RH (2023) The Metabolic Matrix: Re-engineering ultraprocessed foods to feed the gut, protect the liver, and support the brain. *Front. Nutr*. 10:1098453. doi: 10.3389/fnut.2023.1098453

[769] World Health Organization. Health and Well-being. https://www.who.int/Data/Gho/Data/Major-Themes/Health-and-Well-Being

[770] Zheng, Q., Gao, X., Ruan, X. et al. (2025) Are low-carbohydrate diet interventions beneficial for metabolic syndrome and its components? A systematic review and meta-analysis of randomized controlled trials. *Int J Obes DOI:10.1038/s41366-025-01822-5*

[771] Athinarayanan SJ, Roberts CGP, Phinney SD et al. (2025). Effects of a continuous remote care intervention including nutritional ketosis on kidney function and inflammation in adults with type 2 diabetes: a post-hoc latent class trajectory analysis. *Front. Nutr. Sec. Nutrition and Metabolism,* Vol 12 – 2025, DOI: 10.3389/fnut.2025.1609737

The protection and promotion of health require officials to remain abreast of contemporary science concerning the central role of diet and nutrition in metabolic regulation, hormonal balance, and homeostasis. Vulnerable groups, including infants, children and adolescents, pregnant women, and those with elevated metabolic risk, have received insufficient attention. Indeed, agencies currently lack a clear understanding of what constitutes optimal nutrition by age and life stage. Government bodies have consistently failed to examine the relationships between diet quality, nutrient insufficiency, and the physiological demands imposed by age, sex, ethnicity, genetic variability, socioeconomic context, pregnancy, and inflammatory status.

Historic alignment with international dietary frameworks used in Australia, the United States, and Nordic countries has not succeeded in halting or reversing the rise of prediabetes, diabetes, metabolic syndrome, or mental illness. In the decades following the adoption of current guidelines, multimorbidity in younger age groups has increased markedly. Current policy frameworks emphasise the LDL cholesterol marker, minimise the importance of key macronutrients, fat and protein, and fail to link micronutrient sufficiency with optimal physiological function and resilience.

Public-good research in nutrition science, including the investment required to update regulations and policies through transparent reviews of the scientific literature, has been neglected, deprioritised, and underfunded. As a result, independent scientists that can challenge current assumptions are rare, and government policy remains largely silent on the carbohydrate–insulin pathway and insufficiently responsive to individual metabolic risk.

The technology to detect elevated risk for prediabetes is readily available, and the capacity to screen for nutrient deficiencies in people presenting with mental-health conditions is well established. Yet these interventions remain underutilised, restricted, or unrecognised. Over the same period, funding for pharmaceutical access has expanded, while research into drug risks and adverse effects has been comparatively underfunded. Drug trial data are difficult to access, if not opaque, and governments have not provided adequate funding to independently evaluate industry claims or to systematically assess harms alongside benefits.

In these knowledge gaps, officials appear disproportionately focused on potential risks associated with nutritional supplements, despite long histories of safe use and contradictory evidence, while adverse drug risks are largely left to voluntary disclosure by manufacturers. This reflects a deeper contradiction: the Ministry of Health has the authority to set clinical limits for nutrients, yet is not positioned as an authority on optimal nutrition or its role in sustaining metabolic and mental health.

Suboptimal diets and nutrient insufficiencies amplify risk across a broad spectrum of chronic conditions, including metabolic syndrome and complex multimorbidity, yet when knowledge is absent or incomplete, clinicians, families, and patients are denied meaningful choice. Informed consent cannot be achieved when upstream dietary options are neither explained nor endorsed.

This Report, together with the forthcoming companion report on micronutrients and mental health, demonstrates that a longstanding governance culture has placed carbohydrate science, the carbohydrate–insulin model, and nutritional sufficiency outside the scope of formal health policy for decades. Without system-wide correction, continued institutional reticence and gaps in nutritional understanding will perpetuate poor policy and poor outcomes.

There is, however, strong cause for optimism. Current metabolic and mental-health trends are not inevitable. They are reversible. With evidence-based, nutrition-centred health policy, the trajectory of

chronic disease in New Zealand can be changed, and health reclaimed. This Report sets out practical pathways to address the primary drivers of the metabolic and mental-health crisis and to reclaim health.

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New Zealand Charitable Trust

GLOSSARY

AI	Adequate Intake (AI): the recommended average daily intake level based on observed or experimentally determined approximations or estimates of nutrient intake by a group/s of apparently healthy people assumed to be adequate. Used when an RDA cannot be determined.
AMDR	Acceptable macronutrient distribution range.
CARM	Centre for Adverse Reactions Monitoring
CHD	Coronary heart disease
CVD	Cardiovascular disease
DEN	Hardy's Daily Essential Nutrients
DRI	Dietary reference intake
EAR	Estimated Average Requirement (EAR): the average daily nutrient intake level estimated to meet the requirement of half the healthy individuals in a particular life stage and gender group.
EFSA	European Food Safety Authority
FDA	U.S. Food and Drug Administration
FNB:IOM	Food and Nutrition Board Institute of Medicine (U.S.)
hsCRP	High-sensitivity C-reactive protein

Hyperinsulinemia: Chronically elevated (compensatory) insulin.

Hyperglycaemia: Elevated blood glucose (whether temporary or persistently high over time).

Hypoglycaemia: Abnormally low blood glucose level, which may be brief, unstable, or recurrent.

mg	Milligrams
mcg	Micrograms
MoH	Ministry of Health New Zealand
MPI	Ministry for Primary Industries
NASEM	National Academies of Sciences, Engineering, and Medicine U.S.
NHMRC	National Health and Medical Research Council

NRV	Nutrient Reference Values
NZPhvC	New Zealand Pharmacovigilance Centre
OECD	Organization for Economic Cooperation and Development
PHCC	Public Health Communication Centre
RDA	Recommended Dietary Allowance (U.S.): the average daily dietary nutrient intake level sufficient to meet the nutrient requirement of nearly all (97 to 98 percent) healthy individuals in a particular life stage and gender group.
RDI (US)	Reference Daily Intake
RDI	Recommended Dietary Intake (NHMRC). The average daily dietary intake level that is sufficient to meet the nutrient requirements of nearly all (97–98 per cent) healthy individuals in a particular life stage and gender group.
SMARS	Suspected Medicine Adverse Reaction Search
UC	University of Canterbury, New Zealand
UL	Tolerable Upper Intake Level (UL): the highest average daily nutrient intake level that is likely to pose no risk of adverse health effects to almost all individuals in the general population. As intake increases above the UL, the potential risk of adverse effects may increase.

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