2.

MNZH MAKE NEW ZEALAND HEALTHY

Put Prevention Back at the Heart of Healthcare. In Brief.

THE PROBLEM: No wonder patients and health care practitioners have been frustrated. New Zealand's health law and policy architecture does not explicitly recognise nutrition, diet, or nutrient sufficiency as foundational determinants of health. While the system has expanded clinical services and pharmaceutical management, the legislative and institutional framework guiding the Ministry of Health places limited emphasis on the biological role of nutrition in metabolic and neurological function. Frankly, nutrition has been left out in the cold – for decades.

The Pae Ora (Healthy Futures) Act 2022 does not expressly recognise nutrition or metabolic health as determinants requiring systematic attention, and the regulatory environment created by the Medicines Act 1981 has discouraged explicit discussion of nutrients in therapeutic contexts. New Zealand's micronutrient policies only focus on preventing deficiency.

There's no sign of change. Policy analyses associated with therapeutic products reform show limited engagement with nutrient biology or with the growing burden of multimorbidity, polypharmacy, and adverse drug reactions. Official decisions suggest that nutrients are viewed from a toxicological perspective to be as equally risky as pharmaceutical drugs, which is incorrect.

Nutrition is core to biological health, yet the core policy architecture does not prioritise nutrition, raising questions about the regulatory fitness of a health system faced with rising metabolic disease. New Zealand's illness burden arises from chronic metabolic and brain-related conditions, rather than from genetic or infectious disease. Medical training and treatment protocols leave doctors largely unable to view nutritional interventions as a legitimate treatment pathway.

Metabolic health is central to daily functioning and foundational to key indicators of national wellbeing, including self-rated health, life expectancy, population mortality, and maternal and post-natal outcomes, yet our systems downplay the drivers *and* downplay the multimorbid impact. Multiple chronic conditions: *multimorbidity* is more common than a single condition.

More medicines promptly prescribed, does not equate to improved quality of life. Yet doctors and clinicians are caught in a system where the health targets prioritise access to medical treatment and services, rather than addressing the upstream metabolic drivers of illness. Diabetes, for example, frequently coexists with cardiovascular disease, kidney disease, neuropathy, and other chronic conditions. At its core diabetes functions less as a single disease than as a multimorbidity platform, generating sustained prescribing for cardiometabolic, renal, and neurological complications. Poor diets, insulin resistance and related cardiometabolic regulation also increase risk for a spectrum of mental illnesses.

THE SOLUTION: An integrative system empowering healthcare professionals to identify root causes of illness (multimorbidity) and optimise nutritional interventions for optimal health.

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MNZH POLICY RECOMMENDATIONS

THE SOLUTION: The Ministry of Health has not prioritised nutrition for health. For 50+ years the system has expanded its capacity in clinical services and expanded the pharmaceutical budget. At the same time, the legislative and institutional framework guiding the Ministry of Health has placed limited emphasis on the biological role of nutrition in metabolic and neurological health.

New Zealand is well positioned to lead internationally in the integration of metabolic and nutritional approaches to mental and chronic disease reform. The country hosts globally recognised expertise in metabolic health, including work by Professor Grant Schofield and Caryn Zinn on dietary strategies for reversing elevated blood glucose and triglycerides, as well as pioneering research into nutritional approaches to mental health, including the multinutrient clinical research programme led by Julia Rucklidge at the University of Canterbury.

With such credible domestic expertise, New Zealand has the opportunity to develop policy grounded in its own scientific capacity rather than relying solely on external models that, to date, have not halted the rise of chronic metabolic and mental illness. Historically, smaller jurisdictions have often driven innovation in public health precisely because they are able to act on strong local research capability and policy agility.

Increase the sophistication of current approaches to multi-chronic disease assessment.

Impose an ethical duty to correct modifiable biological constraints before or commensurately with medical treatment approaches. Pharmacological interventions typically target discrete biochemical pathways to suppress or modulate symptoms. Their evaluation through clinical trials assesses efficacy (e.g., symptom suppression, mortality reduction) and safety (e.g., incidence of adverse events). Nutritional therapeutics, by contrast, act systemically, supporting metabolism, reducing inflammation, harmonising hormonal and immune function, and restoring cellular integrity.

The trustworthiness of medicine depends on the quality and completeness of clinical information. For information to support informed consent, it must include:

- Mechanistic understanding of symptom drivers.
- Comparative risk–benefit data for pharmacological vs. non-pharmacological options.
- Clarity on uncertainties and evidence gaps.

(A) METABOLIC HEALTH STRATEGY: SCREENING FOR UNWELL POPULATIONS

Support doctors to help patients detect common drivers of chronic conditions, improve patients' metabolic health and lower the prevalence and incidence of complex, chronic conditions, including brain related conditions. Upgrade screening capacity and provide funding, for the following screens on presentation of complex chronic conditions and brain related conditions.

Before initiating pharmacotherapy at any age, clinicians have an ethical responsibility to screen for patients who present with a two or more of these symptoms: suspected mild or subclinical thyroid dysfunction, a gastrointestinal disorder, a diagnosed mental health disorder, fatigue syndromes, chronic inflammation, joint pain, pregnant, have difficulty concentrating or cognitive slowing,

menstrual disturbance, weight gain who are on a list or scheduled for surgery and/or suspected cancer or diagnosed cancer.

Metabolic dysregulation from these factors impacts neurocognitive function, endocrine balance, immune response, mitochondrial activity, and inflammation control, each contributing to resilience, healing, and symptom recurrence risk. People presenting with the symptoms described above often sit along the 'sub-optimum' spectrum.

Upgrade and extend screening to improve metabolic health parameter screening. Current baseline panels include a full blood count, electrolytes/renal function, liver function tests, lipid profile, HbA1c, TSH and ferritin/iron studies. Ensure that doctors have the discretion (and that the funding is available) to expand serum testing including for key nutrients (See Chapter 8).:

a. Standard lipids: add apoB, and only require testing for Lp(a) once in adulthood.
b. Glucose/HbA1c: add fasting insulin or TyG logic.
c. Liver function tests: add Gamma-glutamyl transferase (GGT) and calculate FIB-4 in. metabolically at-risk adults.
d. Consider hs-CRP and uric acid for better upstream risk capture.
e. vitamin D, vitamin B12, folate, iron (ferritin + transferrin saturation), magnesium, zinc, and selenium.
f. Functional methylation impairment.

If there are gastrointestinal symptoms, weight loss, chronic diarrhoea, or malabsorption clues, conduct celiac testing and more focused gastrointestinal evaluation.

Where persistent hyperhomocysteinaemia or unexplained deficiency is identified, selective testing for common MTHFR polymorphisms (C677T and A1298C) may be considered to assist interpretation of metabolic vulnerability and guide nutritional management. Screening should be prioritised for:

- Women planning pregnancy or in early pregnancy, given the established link between folate metabolism, neural tube defects and adverse pregnancy outcomes.
- Individuals with recurrent pregnancy loss or unexplained obstetric complications.
- Patients with persistent hyperhomocysteinaemia or unexplained cardiovascular risk at younger ages.
- Individuals with chronic fatigue, cognitive dysfunction, or treatment-resistant mood disorders where nutrient insufficiency or methylation disturbance is suspected.
- People with malabsorption disorders, restrictive diets, or long-term medications affecting folate/B-vitamin metabolism.

The purpose of such targeted screening is not genetic diagnosis per se, but improved identification of metabolic and nutritional insufficiencies that are potentially correctable through diet, supplementation, or clinical management

(B) IMPLEMENT HEALTH COACHING FOR A NUTRITIONAL UPGRADE.

Implementation Strategy – Ten Year Plan.

Objective: Integrate metabolic health coaching into primary care, mental health, and addiction services to reduce diabetes burden, improve mental wellbeing, and lower long-term system costs.

Prioritise: Individuals diagnosed with prediabetes or type 2 diabetes, obesity with self-reported food addiction, or those receiving mental health and addiction services should be offered structured health coaching as part of routine care. The purpose is to support stabilisation of blood glucose, promote healthier insulin signalling, improve diet quality, and reduce systemic inflammation through practical lifestyle and nutritional strategies. Priority access should be given to groups at higher risk of poor outcomes, including people with treatment-resistant mental health conditions or severe medication side effects, pregnant women, Māori and Pasifika communities and populations experiencing high deprivation, and young people under 25. Integrating health coaching into clinical pathways provides a low-risk, preventive approach that addresses the metabolic drivers of both chronic disease and mental distress while supporting patient capability and long-term self-management.

- ✓ Evaluation metrics should include metabolic markers, medication use, relapse rates, and wellbeing outcomes. National rollout through Health NZ commissioning frameworks.
- ✓ Expand the New Zealand Primary Health Organisations (PHOs) Health Improvement Practitioners and Health Coaches work scope beyond general social or life-coaching' functions to diet and nutrition coaching.
- ✓ Expand the Integrated Primary Mental Health and Addiction (IPMHA) Health Coach Training programme to broader high risk population categories.
- ✓ Integrate information and education to primary care practitioners to support this integration.

1. Terms of Reference for Programmes

Scope:

i. Deliver whole-food carbohydrate-reduction support.
ii. Integrate food addiction counselling and behaviour change coaching.
iii. Recognise prediabetes as early type 2 diabetes to enable early intervention.
iv. Provide continuous glucose monitoring (CGM) access for metabolic self-management.
v. Support deprescribing where metabolic markers improve.
vi. Deliver culturally aligned, whānau-centred care.

Service Model

- ✓ GP oversight with health-coach delivery.
- ✓ Integration across primary care, mental health, addiction services.
- ✓ Community and peer support components.
- ✓ Digital tools and data tracking for monitoring outcomes.

2. Priority High-Risk Populations – Initial focus cohorts:

a. Individuals with prediabetes or type 2 diabetes.
b. People with obesity and self-disclosed food addiction.
c. People receiving mental health and addiction services:
    i. Individuals with treatment resistance or who experience severe side effects from medication.
    ii. Pregnant women.

iii. Māori and Pasifika and high deprivation populations.
iv. Young people under 25.

3. Continuous Glucose Monitoring Access.

Fund CGM for patients with prediabetes and diabetes in priority cohorts. Integrate CGM data into coaching and clinical review. Use CGM to support behaviour change, medication titration, and relapse prevention.

4. Funding & Rollout Plan (10 Years)

Phase 1 (Years 1–2): Pilot & Infrastructure. Fund pilot sites in high-need regions. Expand Integrated Primary Mental Health and Addiction (IPMHA) health coach training scope. Establish data systems and CGM procurement pathways. Expand CGM access for priority groups.

Phase 2 (Years 2–4): Expansion. Scale through PHOs and Māori & Pasifika providers. Embed services in mental health and addiction pathways.

Phase 3 (Years 5+): National Integration. Commission services nationwide via Health NZ. Integrate into standard diabetes and mental health care pathways. Link funding to outcomes (HbA1c, medication reduction, hospitalisation rates).

5. Investment Logic

Upfront investment in coaching, CGM access, and workforce training is expected to reduce diabetes incidence and complications; lower pharmaceutical expenditure and polypharmacy; improve mental health stability and addiction recovery; and reduce hospital admissions and long-term disability.

This phased approach enables targeted early impact while building a sustainable national model aligned with equity and prevention priorities.

(C) MENTAL HEALTH: MULTINUTRIENT SUPPLEMENTATION AS A FIRST LINE INTERVENTION

Establish a first-line or early-stage intervention option for vulnerable populations, particularly where current treatments carry significant side-effect burdens or are poorly tolerated.

Potential priority populations could include children and adolescents with ADHD or mood dysregulation, pregnant women experiencing anxiety or depression, individuals with treatment-resistant mental health conditions, and patients who experience severe adverse effects from antidepressant medications. These groups often face limited treatment options and may benefit from interventions with comparatively low risk profiles.

Given the growing burden of mental illness in New Zealand, alongside emerging clinical evidence supporting broad-spectrum micronutrient approaches to mental health[1], a targeted fast-track evaluation of Hardy's multinutrients could be considered within the current policy and funding framework.

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[1] Rucklidge JJ, Johnstone JM, Villagomez A, Ranjbar N, Kaplan BJ (2023) Broad Spectrum Micronutrients and Mental Health. Chapter 9. In Nutritional psychiatry: A primer for clinicians, pages 152-171. Edited by Ted Dinan. Cambridge University Press. DOI: 10.1017/978100929986

Multiple lines of research suggest robust metabolic and nutritional status may influence the severity, resilience, and recovery trajectory of mental illness. Several systematic reviews and large cohort studies now show that metabolic dysfunction (including insulin resistance, obesity, mitochondrial dysfunction and systemic inflammation) is strongly associated with worse mental-health outcomes and poorer treatment response.

Importantly, New Zealand already has internationally recognised research expertise in this area through the University of Canterbury micronutrient research programme. This provides a credible scientific foundation for domestic evaluation and avoids the need to rely solely on overseas evidence.

However, the solution cannot be arrived at without appreciating the institutional barriers to securing access. Under New Zealand's medicines funding framework, there are several mechanisms through which the evaluation and potential funding of Hardy's multinutrients could be initiated, however, an existing case study provides a model for a shift in approach to securing multinutrient supplements for mental health, with a demonstrated positive safety and efficacy profile on the Pharmac Schedule.

Fast-Track Evaluation and Funding of Hardy's Multinutrients for Vulnerable Populations

PHARMAC operates within a pharmaceutical funding framework designed to evaluate discrete medicines used to treat identifiable clinical conditions, rather than broader public health interventions aimed at improving nutritional status in the population. Funding applications must therefore fit the model of a specific product, dose, indication, and patient population, supported by clinical trials demonstrating efficacy for a defined disease state. Preventive strategies, such as improving dietary nutrient intake, addressing subclinical deficiencies, or supporting metabolic health through nutritional interventions, often fall outside this structure because they are not framed as treatments for a diagnosed illness and may not involve a single proprietary product.

New Zealand should establish a fast-track PHARMAC assessment and targeted funding pathway for Hardy's broad-spectrum multinutrients as a first-line or early-line option for vulnerable populations, particularly children and adolescents with ADHD or mood dysregulation, pregnant women with depression or anxiety, people with treatment-resistant symptoms, and patients who experience serious adverse effects from antidepressants or other psychotropics.

The case for fast-tracking is clinical *and* institutional. PHARMAC's standard processes were built around single-agent pharmaceuticals for defined disease indications, yet Hardy's sits in a different category: a multi-nutrient intervention with a comparatively benign safety profile and a growing evidence base across ADHD, emotional dysregulation, anxiety, depression, and other psychiatric conditions

The PHARMAC application pathways are not configured to evaluate this kind of intervention robustly, particularly where officials may not have deep expertise in nutritional psychiatry, systems biology, and the comparative safety analysis needed to weigh micronutrients fairly against psychiatric medicines. That is precisely why a formal fast-track approach is justified. PHARMAC's own framework allows it to consider need, health benefits, costs and savings, and suitability, and

it explicitly prioritises impacts on groups with worse health outcomes, including Māori. The funding strategy should have four parts.

1. Hardy's should be formally entered or re-entered into PHARMAC's funding process with a clearly defined indication set for vulnerable populations and a dossier built around the best available RCT, follow-up, and safety evidence. Anyone can make a general funding application to PHARMAC, including a supplier, health professional, or any New Zealander, so this route is already open.
2. PHARMAC should send the application to PTAC and any relevant specialist advisory committees, and where necessary obtain additional subject-matter advice on nutritional psychiatry, perinatal mental health, child and adolescent psychiatry, and adverse-effect comparison with antidepressants. PTAC's terms expressly allow specialist advice to be sought where subject-matter expertise is required.
3. If PHARMAC considers the full national listing still too uncertain, it should use a time-limited funded access pathway for defined high-need groups, coupled with outcome monitoring. PHARMAC's statutory framework and operating policies allow it to engage in research and in responsible-use activities, which provides a lawful basis for a structured pilot or monitored implementation approach.
4. If individual patients cannot wait, clinicians should be encouraged to test whether access can be pursued through NPPA or other exceptional-circumstances mechanisms where the clinical facts are strong, although NPPA is a case-by-case route and not a substitute for a proper population funding decision.

(D) HELPING DOCTORS AND CLINICIANS

Make New Zealand Healthy (MNZH) presents evidence-based options, but it is essential to recognise the long-term institutional and educational barriers faced by general practitioners (GPs) and clinicians. Doctors operate within a system whose legal, regulatory, and scientific foundations were largely established before current knowledge of metabolic health emerged. While GPs and clinicians are committed to improving patient outcomes, their tools and institutional support are not aligned with contemporary scientific evidence. They are, in effect, working within constrained parameters that limit their capacity to address upstream drivers of disease.

New Zealand's health governance framework provides well-established pathways for pharmaceutical treatment, yet the science system presents significant barriers to discovery-driven research into nutrition and diet for metabolic and brain health, undermining the role of nutrition. Nutrition science within the Ministry of Health remains narrow in scope and largely oriented toward preventing deficiency rather than optimising physiological function or addressing the chronic disease burden. Medical training reflects this imbalance, with limited focus on therapeutic nutrition and a predominant emphasis on pharmaceutical intervention.

MNZH's policy platform is designed to address these constraints through an integrated programme of reform. Policy (1) proposes a staged approach to metabolic health, enabling clinicians to provide dietary guidance that supports metabolic resilience. Current dietary guidelines often underemphasise the role of protein and healthy fats while insufficiently addressing the impact of refined carbohydrates on metabolic disease. This places clinicians in a position where evidence-based dietary advice may conflict with official guidance. Complementary fiscal measures, such as removing GST from essential whole foods and applying targeted levies to ultra-processed foods

and sugar-sweetened beverages, would improve access to nutrient-dense diets. Restoration of nutrient-rich Ka Ora, Ka Ako school lunches and stronger regulation of food marketing further reinforce this upstream approach outlined in our first policy.

New Zealand currently lacks a dedicated environmental and human health research institute capable of undertaking long-term, systems-level research into nutrition, metabolism, and environmental exposures. Establishing such an institute would strengthen the evidence base across sectors, informing health policy, education, and clinical practice. It would support a continuum of knowledge from mechanistic biology through to clinical and population-level studies, and enable modern analytical tools to synthesise and translate this evidence into practice (policy 9).

Clinicians and general practice doctors (GPs) learn about the role of nutrient cofactors and the human metabolism in undergraduate training, however, education in nutrition forms only a very small element of under- and post-graduate medical training. A chilling effect on nutrients as a therapeutic practice has been furthered by punitive Medical Council actions if doctors contradict health agency guidelines, no matter if they contradict a broader scientific evidence base. Doctors with a deeper education in nutrition will be more confident in working with nutrients to support the treatment of the patient before them, and they may be more confident challenging out of date science, in order to support the health of their patients (policy 4).

In addition, the New Zealand Formulary does not consistently provide clear, accessible data on efficacy and safety, including numbers needed to treat and harm, or stratification by age and sex. This limits the ability of clinicians to provide fully informed consent. Strengthening transparency and evidentiary clarity critical if doctors are to be able to meaningfully judge the risks and benefits of medical and nutritional treatments (policy 5).

Regulatory barriers also limit clinicians' ability to use nutrition therapeutically. Nutrient regulations remain oriented toward deficiency prevention, and prescribing nutrients above conservative guideline thresholds can expose clinicians to professional risk, despite low biological hazard and established safety profiles (policy 4 and 5). This creates a disincentive to address correctable nutritional insufficiencies. MNZH therefore proposes removing Pharmac and Medsafe barriers that restrict access to safe, non-patentable nutrients. Current Pharmac funding pathways are structured around pharmaceutical models requiring costly industry-sponsored trials, which are not feasible for nutrient-based interventions (policy 6).

Finally, clinicians face substantial time and administrative burdens. Beyond short consultation windows, GPs often spend significant additional hours on follow-up, reporting, and compliance tasks. Reducing this burden is critical to restoring clinical capacity and enabling more meaningful patient care (policy 7). Taken together, these reforms would better equip clinicians to:

- ✓ Detect metabolic risk, inflammation, and nutrient insufficiency earlier.
- ✓ Apply nutrition therapeutically at levels that support optimal health.
- ✓ Operate within a system informed by contemporary science.
- ✓ Access research and regulatory frameworks that support nutritional approaches.

- ✓ Ensure they are supported by robust advertising frameworks, health coaching and complementary community education and practice.
- ✓ reduce administrative burden and improve patient engagement.

This is not a critique of clinicians, but of the system in which they work. Reform is required to align governance, science, and clinical practice with the realities of modern chronic disease and to enable doctors to deliver truly preventive and restorative care.

BACKGROUND TO THIS POLICY

Primary and secondary care differ in both function and orientation. Primary care is the first point of contact and is intended to deliver continuous, comprehensive, and person-centred care across the life course, with a core emphasis on prevention, early intervention, and the management of multimorbidity in real-world contexts. Secondary care, by contrast, is specialist, hospital-based care focused on the diagnosis and treatment of defined conditions through targeted investigation and intervention. Accordingly, secondary care is episodic and problem-specific, whereas primary care is longitudinal, integrative, and responsible for coordination, complexity management, and addressing biological, behavioural, and social determinants of health.

In practice, however, the primary care workforce is trained predominantly within secondary care settings, with doctors training in hospital environments. This approach encourages a diagnosis- and treatment-focused clinical model. Primary care skills are subsequently developed within this secondary care framework, and general practices are funded and equipped to deliver a form of community-based secondary care. This creates a professional misalignment: primary medical care is not consistently trained, resourced, or supported to fulfil the broader mandate of primary health care. The New Zealand system therefore tends to conflate primary medical care with primary health care, leaving preventive, behavioural, environmental, and social dimensions underdeveloped.

The result is a service model that is heavily weighted toward intervention, with limited capacity to support informed health decision-making or sustained behaviour change. This gap is recognised by many clinicians and contributes to workforce dissatisfaction. Core areas required for effective primary care, such as nutrition, lifestyle medicine, psychological and trauma-informed care, environmental health, musculoskeletal care, and social prescribing, remain variably integrated into training and practice. While not all of these functions sit within the direct scope of the GP, the ability to understand, coordinate, and appropriately refer across these domains is essential to a functioning primary health care system.

[1] NUTRITION: SILENCE ACROSS HEALTH POLICY & LEGISLATION ARCHITECTURE

New Zealand’s statutory and regulatory health framework does not explicitly recognise optimal nutrition and dietary quality as foundational determinants of health. This absence is significant because the biological foundations of health: adequate nutrient status and healthy dietary patterns - are well established as primary determinants of non-communicable disease risk.

Consequently, the biological role of nutrition in maintaining metabolic function, supporting neurological health, and preventing chronic disease has remained largely peripheral within the Ministry of Health’s policy architecture. While the health system has progressively expanded its

capacity in clinical services, pharmaceuticals, and disease management, the legislative and institutional structures guiding decision-making do not clearly establish nutrition and diet as core components of health protection and disease prevention. This institutional silence has contributed to a policy environment in which the upstream drivers of metabolic and chronic disease remain comparatively under-recognised.

This outcome reflects deeper features of how modern health systems developed. Much of the legal architecture governing health was designed in the mid-twentieth century when the dominant threats to population health were infectious disease, trauma, and acute illness. Accordingly, statutes, institutions, and professional training were constructed around clinical services, hospitals, pharmaceuticals, and professional regulation. Nutrition policy, by contrast, historically evolved through agriculture and food security frameworks, where the primary objective was preventing classical deficiency diseases and ensuring adequate food supply. The concept of optimal nutrition as a determinant of metabolic health emerged much later in scientific literature and has not yet been systematically integrated into health law or governance structures.

This impacts healthcare directly.

Metabolic Health: Eight System Failures

Several institutional features act in conjunction to undermine New Zealand's approach and make it difficult to reverse the chronic disease crisis.

First, the Pae Ora (Healthy Futures) Act 2022, which now provides the primary statutory framework for the health system, stated purpose of protecting, promoting, and improving the health of New Zealanders while reducing inequities in health outcomes. The Act embeds principles of prevention, population health, and recognition of the wider determinants of health. The Act does not expressly recognise nutrition, diet, nutrient sufficiency, or metabolic health as determinants requiring systematic attention. Medicines including pharmaceuticals are explicitly defined and supported through statutory institutions such as Pharmac. Although the Act refers to prevention, public health, and the wider determinants of health, it does not require the health system to identify, monitor, or mitigate dietary and nutrient drivers of disease. The statutory framework therefore permits attention to these factors but does not clearly oblige officials to prioritise them.

Second, the Medicines Act 1981 created a regulatory environment in which nutrients may fall within medicines regulation when therapeutic claims are made that refer to physiological, metabolic, or immunological pathways. Yet micronutrients are defined biologically by precisely these roles: they support metabolic regulation, cellular signalling, and immune function across multiple systems. In practice, this regulatory framing has had a chilling effect on communication about the systemic benefits of nutrients, discouraging more explicit articulation of their therapeutic potential in maintaining or restoring biological function.

Third, medical education recognises vitamins and minerals as cofactors in metabolic pathways, but clinical training has traditionally focused more heavily on pharmacological management of disease than on the therapeutic application of nutrition in addressing metabolic dysfunction. As a

result, many practitioners are highly skilled in pharmacological intervention but less equipped to apply nutritional strategies within clinical care.[2] [3] [4] [5]

Fourth, current dietary guideline frameworks are primarily designed to prevent deficiency diseases. Recommended intake levels are generally derived from minimum thresholds required to avoid deficiency, rather than from evidence concerning optimal intake for metabolic or neurological health. Upper intake levels are often interpreted as toxicity thresholds even though many reflect complex nutrient interactions rather than direct toxic risk. In some cases, risk claims have been confounded by trial designs involving multiple nutrients—for example vitamin D trials conducted alongside calcium supplementation—resulting in risk communication that may obscure the independent biological effects of specific nutrients.

Fifth, New Zealand lacks a dedicated national research institute or programme focused on optimal nutrition and nutrient biology. In the absence of sustained institutional capability, officials have generally relied on the Australia–New Zealand Nutrient Reference Values and related dietary guidelines. Much of the evidence base underpinning these frameworks originate from studies conducted prior to the expansion of modern systems-biology, metabolomics, and nutritional epidemiology research. Consequently, evolving scientific understanding of nutrient interactions, metabolic regulation, and the role of nutrition in chronic disease has not always been systematically incorporated into domestic policy analysis.[6]

Sixth, the professional regulatory environment has become increasingly cautious. Heightened scrutiny of clinicians by professional bodies has created a perception among some practitioners that recommending nutrient intakes beyond established dietary guideline levels may attract professional risk. Whether formally justified or not, this environment can discourage clinicians from discussing or applying emerging evidence relating to nutrition and metabolic health.

Seventh, the policy environment risks becoming self-reinforcing. Policy analyses supporting recent therapeutic product reforms, including Regulatory Impact Assessments and Regulatory Impact Statements, suggest that nutrition and metabolic determinants are not being systematically considered within regulatory design. Contracted analyses examining nutrient safety have in some instances failed to address nutrient cofactor relationships, interaction effects, or confounding factors within the literature, and have not clearly distinguished between risks associated with ingredient quality or formulation and those arising from the intrinsic biological activity of nutrients. At the same time, there is little evidence that policy work has systematically examined the combined burden of multimorbidity, polypharmacy, and adverse drug reactions within the New Zealand population, including metrics such as numbers needed to treat or numbers needed to harm. This absence of analysis creates a risk that regulatory attention may

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[2] Crowley J, Ball L, Hiddink GJ. (2019). Nutrition in medical education: a systematic review. The Lancet Planetary Health, 3(9):e379 - e389

[3] 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.

[4] 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

[5] 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

[6] See: MNZH POLICY [7] ESTABLISH A PUBLIC-GOOD ENVIRONMENTAL HEALTH RESEARCH INSTITUTE.

overestimate nutrient-related risks while underestimating pharmaceutical risks, particularly in contexts where multiple medications are prescribed concurrently.

Eighth, the institutional design of the pharmaceutical funding system creates a governance bias toward downstream treatment rather than upstream prevention. Pharmac evaluates and funds discrete medicines for defined clinical indications within a fixed pharmaceutical budget. As a result, high-dose nutrient formulations may be funded when used to treat diagnosed deficiency states, such as vitamin B12 injections or therapeutic vitamin D, yet the system has limited mechanisms to support broader preventive strategies aimed at improving population-level nutrient sufficiency or metabolic health. Nutritional and metabolic interventions often involve dietary patterns, multi-nutrient approaches, and longer time horizons for measurable outcomes, which do not fit easily within a pharmaceutical funding framework designed around single products and disease indications. This institutional asymmetry reinforces a policy environment in which deficiencies are treated once disease has developed, while the upstream nutritional drivers of chronic disease receive comparatively less systematic attention.

Medical Treatments Operationalised Without Adequate Reporting Systems in Place

These institutional dynamics collectively contribute to a policy environment in which nutrition holds a marginal role within the health system, despite its central role in biological function. At the same time, New Zealand faces rising rates of metabolic illness, multimorbidity, and brain-related disorders, many of which are strongly associated with dietary patterns and metabolic dysregulation.

The statutory framework and related policy supports an equity framework that is articulated primarily in terms of equitable access to services and health outcomes, rather than equity in the underlying conditions necessary for biological health. The health system operationalises equity largely as equitable access to diagnosis, treatment, and pharmaceuticals, rather than as equity in exposure to the conditions required for metabolic health.

The statutory framework emphasises equitable outcomes for population groups but does not require systematic recognition or reporting of the extent to which multiple chronic conditions co-occur within individuals. It is largely blind to the extent and economic burden of multimorbidity, as it operationalises equity in terms of access to diagnosis and treatment. In contemporary health systems, multimorbidity is increasingly the norm rather than the exception, particularly among middle-aged and older populations. This pattern has direct implications for prescribing practices. Where multiple conditions are managed through condition-specific treatment pathways, the clinicians will prescribe several medications – up to fifty, simultaneously. [7] [8]

Current legislation and policies do not explicitly address the broader drivers of these dynamics and the burden of suffering from current concomitant conditions and prescribing regimes, which

[7] 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

[8] 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.

can include complex adverse effects that require additional medications to manage these side effects.

Three major faultlines lead to medical harm being under-reported which then results in patients not being adequately informed on medication efficacy and risks. Safety-data sheets are managed by the drug sponsor and are not updated independently as the scientific literature builds a picture of harm. Adverse event reporting systems do not provide an adequate signal of the extent of side effects by age and gender from medications, with only the severe adverse effects retained by government officials. Many uncomfortable and debilitating effects, not considered severe but which impair quality of life, are retained by the pharmaceutical sponsor. Finally, the New Zealand Formulary does not have an adequate way of translating the numbers needed to treat and the incidence of adverse events so that the individual patient may understand their risk profile, particularly when they are prescribed, which is common, to two or more medicines.[9]

Although there was a recent reshuffling of roles and obligations, such concerns have largely not been addressed to improve informational transparency to practicing clinicians and the general public. They are also not addressed in underlying policy intended to support the drafting of a future Medical Products Bill.

A Fundamental Question of Regulatory Fitness

With such contexts taken into account, the continuing silence of core health institutions on the nutritional drivers of these conditions raises a fundamental question of regulatory fitness. Where the dominant burden of disease is increasingly shaped by metabolic dysfunction, a legislative and institutional framework that does not explicitly recognise nutrition and diet as foundational determinants may no longer be adequate to meet the health challenges of the present era.

As a consequence, the legislation does not clearly establish a statutory expectation that officials must identify and address nutrition as a primary determinant of health, nor does it require that nutritional strategies or dietary interventions be systematically integrated into programmes designed to prevent or reverse chronic disease. The absence of explicit legislative recognition therefore leaves the health system without a clear mandate to prioritise nutritional and dietary measures as central tools in addressing the early emergence of multimorbidity among high-risk populations.

In contrast to the under-recognition of metabolic health and nutrition, New Zealand legislation embeds a robust statutory machinery for pharmaceutical management. As a result, the legal architecture more clearly supports intervention through treatment pathways than through systematic attention to upstream biological determinants. In statutory terms, pharmaceuticals are operationalised as a core component of the health system, whereas nutritional determinants are left within discretionary domains such as health promotion or cross-sector collaboration. This encourages an institutional bias toward treatment-based responses, particularly in a health system where chronic disease management dominates service delivery.

The consequences of this asymmetry are particularly evident when considered in light of the epidemiology of contemporary chronic disease. Conditions such as type 2 diabetes,

[9] See: MNZH POLICY [3] GUARANTEE SCIENTIFIC INTEGRITY IN INFORMED CONSENT.

cardiovascular disease, and fatty liver disease are widely recognised to be strongly associated with dietary patterns and metabolic dysfunction.

Case Study: The Hardy's Multinutrient Application to PHARMAC

The application to PHARMAC to fund Hardy's broad-spectrum multinutrients, developed and studied through the University of Canterbury research programme led by Professor Julia Rucklidge, illustrates the barriers to funding nutrients of benefit, particularly for low-income and young people. The formulation has been investigated across a series of clinical and exploratory studies examining outcomes in ADHD, mood dysregulation, anxiety, depression, stress-related symptoms, and emotional regulation, including randomised controlled trials and long-term safety follow-up studies. The research programme has consistently reported improvements in symptom domains and functioning in at least a subset of participants, alongside a comparatively benign safety profile, with adverse effects generally limited to mild and transient gastrointestinal symptoms.

An application to Pharmac to fund the broad-spectrum micronutrient formulation (Hardy's Daily Essential Nutrients, DEN) was led by Professor Julia Rucklidge alongside psychiatrist Matt Eggleston and was supported by substantial patient and family correspondence. Families reported that, due to cost, they were unable to continue accessing the product and would revert to funded pharmaceutical treatments, often with poorer tolerability or outcomes from their perspective.

The application was rejected.

This case study demonstrates that the pathway to funding such an intervention awkwardly within the institutional design of PHARMAC's decision-making framework. PHARMAC is structured to evaluate discrete pharmaceutical products intended to treat defined disease states within a fixed medicines budget. Its assessment methods rely heavily on randomised clinical trial evidence, cost-utility modelling, and comparisons with existing pharmaceutical treatments. Broad-spectrum micronutrient interventions, however, do not fit neatly within this model. They are multi-component biological interventions, often intended to improve underlying metabolic function across multiple systems rather than targeting a single receptor pathway or disease category. Their benefits may also be expressed across multiple symptom domains, including mood regulation, stress tolerance, cognitive function, and overall wellbeing, rather than through a single narrow clinical endpoint.

This creates an inconsistent and ethically questionable policy dynamic between the nature of nutritional interventions and the evaluation architecture used by pharmaceutical funding agencies. As a result, evidence that may be meaningful within clinical nutrition or systems-biology frameworks can be difficult to translate into the cost-effectiveness and disease-specific models typically used for pharmaceutical funding decisions. In practice, this means that even where credible clinical evidence exists, multinutrient interventions may struggle to progress through the PHARMAC funding process because they do not resemble conventional medicines in either mechanism or evaluation design.

The Hardy's multinutrient application is particularly significant because the target populations include groups for whom treatment options can be limited or problematic. These include children and adolescents with ADHD or mood dysregulation, pregnant women experiencing depression or

anxiety, and individuals with treatment-resistant symptoms or intolerance to standard antidepressant medications. In such populations, the safety profile of available treatments becomes a central policy concern. Antidepressants and other psychotropic medications carry well-recognised adverse-effect risks, including behavioural activation, akathisia, metabolic side effects, and regulatory warnings relating to suicidality in younger populations. Withdrawal effects associated with antidepressant discontinuation are also increasingly recognised in the clinical literature. In contrast, the Canterbury micronutrient studies have not reported comparable serious adverse-effect signals, although continued monitoring and research remain important.

From a health equity perspective, this case study also intersects with broader concerns about access to safe and effective mental health interventions. Vulnerable populations, including young people, Māori communities, and individuals with limited access to specialist care, often experience both higher burdens of mental distress and greater exposure to environmental and nutritional determinants of health. Interventions aimed at improving nutritional sufficiency or metabolic resilience may therefore have particular relevance for equity-focused health policy, especially if they offer low-risk treatment options in contexts where conventional pharmacological approaches are not well tolerated or not preferred.

The Hardy's multinutrient application therefore highlights a wider policy question. The issue is not simply whether a specific product should be funded, but whether current institutional frameworks are well suited to evaluating metabolic and nutritional interventions that fall outside traditional pharmaceutical paradigms. When evidence suggests potential benefit with comparatively low risk, and when the target populations include individuals for whom existing treatments may be limited or problematic, a purely pharmaceutical evaluation framework may not fully capture the potential value of such interventions.

In this sense, the Hardy's case can be understood as a policy test of the broader institutional asymmetry within the health system: a system capable of funding high-dose nutrient therapy once deficiency disease is diagnosed, yet less equipped to evaluate multi-nutrient interventions aimed at improving underlying metabolic or neurobiological function. As the burden of mental illness and metabolic disease continues to grow, the question for policymakers is whether the existing architecture of evidence assessment and funding is sufficiently flexible to incorporate nutritional and metabolic approaches alongside conventional pharmaceutical treatments, or whether new evaluation pathways may be required.

[2] THE BURDEN OF MULTIPLE CHRONIC CONDITIONS (MULTIMORBIDITY)

New Zealand prescribing trends and health-system cost analyses together suggest that diabetes is now a central driver of multimorbidity and ongoing pharmaceutical exposure in the population.

New Zealand data reveals that Māori and people in lower-income groups experience multimorbidity approximately a decade earlier than more advantaged populations. This earlier onset is closely associated with environmental determinants of health, among which diet quality, nutrient sufficiency, and reliable access to healthy food play a central role in shaping metabolic risk and chronic disease progression.

Yet neither multimorbidity nor nutrition and dietary determinants are expressly recognised within the Pae Ora (Healthy Futures) Act 2022 and this issue is underplayed in policy documentation and government communications to the general public.

The lived experience of diabetes for many patients involves not simply glucose control but the ongoing management of multiple symptoms, medications, and comorbid conditions. Rising prescribing volumes for diabetes-related medicines are not merely a marker of treatment availability; they are also a proxy indicator of the expanding burden of chronic disease and multimorbidity in the population. As people age with diabetes, medicine use often expands beyond glucose-lowering therapy to include cardiovascular, renal, neurological, and mental-health medications, reflecting the complex clinical landscape that develops over time.

Multiple chronic diseases, as defined by the presence of 2 or more chronic conditions in a single individual increases both health system costs and individual suffering.[10] Frequently reported clustered conditions (known as dyads) result in a spike in both suffering and medical system costs. A U.S. paper identified that co-occurring respiratory and mental health condition, diabetes and heart/vascular condition, and cancer and mental health condition in the first year after cancer diagnosis produced the most burdensome system costs.[11]

The economic burden of diabetes is driven primarily by multimorbidity and complications rather than by glucose-lowering treatment itself. This information is not new.

Early evidence came from Struijs et al. (2006), which showed that healthcare utilisation among people with diabetes increases sharply as additional conditions accumulate. Their analysis demonstrated that comorbidity, not diabetes alone, is the principal driver of medical costs, with substantial increases in specialist care, hospitalisation, and medicine use once cardiovascular, renal, or other chronic conditions develop.[12]

Subsequently, Hex et al. (2012) estimated the national cost of diabetes in the United Kingdom and found that around 80% of healthcare expenditure associated with diabetes is attributable to the management of complications, including cardiovascular disease, renal failure, neuropathy, and eye disease. Routine glucose management accounts for a relatively small share of total costs. Importantly, this analysis was published fourteen years ago, meaning that the broader cost drivers of diabetes have long been recognised in the scientific literature.[13]

More recent work reinforces these conclusions. A systematic review by Tran et al. (2022) shows that multimorbidity consistently multiplies healthcare costs across health systems, with combinations involving diabetes and cardiovascular disease among the most expensive clusters of chronic illness. [14] Likewise, Lee et al. (2025) demonstrates that metabolic risk factors associated with diabetes, including elevated blood pressure and dyslipidaemia, are already detectable in

[10] Yao L, Li Q, Liu Y, Li Q, Wang T, Zhou Z and Yin J (2025) How to assess multimorbidity: a systematic review. Front. Public Health 13:1525593. doi: 10.3389/fpubh.2025.1525593

[11] Tran PB, Kazibwe J, Nikolaidis GF, Linnosmaa I, Rijken M, van Olmen J. Costs of multimorbidity: a systematic review and meta-analyses. BMC Med. 2022 Jul 19;20(1):234. doi: 10.1186/s12916-022-02427-9.

[12] Struijs, J.N., Baan, C.A., Schellevis, F.G. et al. Comorbidity in patients with diabetes mellitus: impact on medical health care utilization. BMC Health Serv Res 6, 84 (2006). https://doi.org/10.1186/1472-6963-6-84

[13] Hex, N., Bartlett, C., Wright, D., Taylor, M. and Varley, D. (2012), Estimating the current and future costs of Type 1 and Type 2 diabetes in the UK, including direct health costs and indirect societal and productivity costs. Diabetic Medicine, 29: 855-862. https://doi.org/10.1111/j.1464-5491.2012.03698.x

[14] Tran PB, Kazibwe J, Nikolaidis GF, Linnosmaa I, Rijken M, van Olmen J. Costs of multimorbidity: a systematic review and meta-analyses. BMC Med. 2022 Jul 19;20(1):234. doi: 10.1186/s12916-022-02427-9.

young adults with prediabetes, highlighting that cardiometabolic dysfunction often develops years before clinical diabetes is diagnosed. [15]

Taken together, these studies indicate that the economic burden of diabetes arises from progressive metabolic dysfunction and the accumulation of cardiometabolic disease across the life course. Given that this pattern has been clearly documented for more than a decade, the persistence of health-system strategies focused predominantly on pharmaceutical management of established disease rather than early metabolic prevention and reversal suggests a significant lag between scientific understanding and health-system response.

[3] PRESCRIBING GROWTH IN NZ: DIABETES AS A MULTIMORBIDITY 'PLATFORM'

In 2021 the total current annual cost of type 2 diabetes in New Zealand was estimated to be $2.1 billion. The estimate included the cost of hospital care, complications, lost productivity and disability.[16]

In 2024, 346,800 people were estimated to have diabetes in Aotearoa New Zealand, with an estimated age-standardised prevalence of 46.7 per 1000 population. In 2013 the age-standardised prevalence of diabetes was 36.6 per 1000 population.[17]

Pharmac's annual reports show steady growth in the number of funded prescriptions and increasing expenditure in therapeutic groups linked to long-term cardiometabolic disease, including diabetes medicines, antihypertensives, lipid-lowering drugs, and antithrombotics. This pattern reflects the reality that diabetes rarely occurs in isolation. As prevalence rises with age, particularly from midlife onward, the condition frequently coexists with cardiovascular disease, chronic kidney disease, neuropathy, and other metabolic complications.[18]

Strikingly, when Pharmac prescribing growth is compared with diabetes prevalence since 2005, the trajectories track closely. This provides one of the clearest population-level indications that the expansion of pharmaceutical use in New Zealand is closely linked to the rising burden of chronic disease.

In New Zealand, diabetes increasingly functions less as a single disease and more as a platform for multimorbidity, particularly from midlife onward. From around age 45, and more markedly after age 60, diabetes frequently coexists with cardiovascular disease, chronic kidney disease, neuropathy, and other long-term conditions. As these complications accumulate, patients typically require expanding treatment regimens that include cardiometabolic, renal, neurological, and mental-health medicines, leading to sustained pharmaceutical exposure and rising healthcare utilisation. Within this clinical trajectory, diabetes illustrates how chronic disease progressively drives both prescribing growth and system costs.

Economic modelling of type 2 diabetes in New Zealand shows how sharply healthcare expenditure increases as disease severity progresses. Annual healthcare costs are estimated at approximately

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[15] Lee, S., Son, DS., Kim, JI. et al. Risk factors for prediabetes in young adults: a study based on Korea National health and nutrition examination survey data. Sci Rep 15, 27119 (2025). https://doi.org/10.1038/s41598-025-13378-5

[16] Blakely T, et al. The Economic and Social Cost of Type 2 Diabetes. Healthier Lives National Science Challenge / Edgar Diabetes and Obesity Research Centre, 2021. https://healthierlives.co.nz/wp-content/uploads/Economic-and-Social-Cost-of-Type-2-Diabetes-FINAL-REPORT_Secure-5.pdf

[17] 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

[18] Pharmac. Year in Review reports (2018–2024) and pharmaceutical utilisation statistics.

NZ$180 per person per year in early disease, rising to about NZ$3,500 annually when complications are present but stable, and reaching roughly NZ$14,000 per year when complications become unstable or severe. Across the population, the average health-system cost of type 2 diabetes is estimated at around NZ$4,600 per person annually, with total national costs of the condition estimated to be around NZ$2 billion each year.

Pharmaceutical spending represents only a portion of this burden. Pharmac data indicate that diabetes medicines alone account for approximately NZ$175–180 million annually, equivalent to roughly NZ$600–1,000 per treated patient per year, depending on treatment intensity.[19] Taken together, these figures illustrate how diabetes acts as a long-term driver of multimorbidity, escalating prescribing volumes and healthcare expenditure as patients age with the disease.

Importantly, diabetes is also associated with a wider range of conditions that are not always described as cardiometabolic but contribute substantially to treatment burden. Epidemiological studies show strong associations between diabetes and mental health conditions such as anxiety and depression, as well as with vision disorders including diabetic retinopathy and age-related macular degeneration. These comorbidities increase the likelihood of additional medicines and specialist care, further amplifying polypharmacy risk and healthcare utilisation. In addition, commonly used diabetes medicines themselves can carry adverse effects that patients must manage over long periods.

Frequently reported side effects include nausea, gastrointestinal discomfort, gas, indigestion, vomiting, diarrhoea, headaches, and increased sweating, while rare but serious reactions, such as lactic acidosis with certain medicines, require clinical vigilance. In clinical practice the cost of side-effect management tends to come from extra GP visits, anti-nausea or gastrointestinal medications, proton pump inhibitors, laboratory monitoring and occasional hospitalisation for rare complications (e.g. lactic acidosis).

These costs are rarely separated in national cost models, so they are effectively hidden within broader diabetes expenditure estimates.

Increasingly, rising rates of prediabetes are being detected in younger age groups, indicating that metabolic dysfunction is emerging earlier than in previous generations.[20] If this trajectory remains unaddressed, it is likely to expand both the lifetime health and economic burden of diabetes. Earlier onset extends the period during which individuals live with the disease and increases the likelihood that complications will develop over time. Consequently, the stage at which annual healthcare costs average around NZ$3,500 per person, when complications are present but relatively stable, may begin to occur earlier in the life course for these cohorts, potentially shifting a significant portion of diabetes-related healthcare expenditure into earlier decades of adulthood.

Lee et al. (2025), analysing adults aged 20–39 years from the Korea National Health and Nutrition Examination Survey, found that individuals with prediabetes exhibited significantly higher systolic

[19] Pharmac (2023). Pharmac prescription drug spending since 2010. https://www.pharmac.govt.nz/news-and-resources/official-information-act/official-information-act-responses/pharmac-prescription-drug-spending-since-2010

[20] Luk, Andrea et al. 2025 Early-onset type 2 diabetes: the next major diabetes transition, The Lancet, Volume 405, Issue 10497, 2313 - 2326

and diastolic blood pressure than participants with healthy blood sugar levels. Although blood pressure was not retained in the final regression model, the authors note that its consistent elevation among those with prediabetes reinforces its role as a feature of metabolic syndrome, closely linked with other cardiometabolic abnormalities such as dyslipidaemia and insulin resistance. These findings highlight that metabolic risk clustering, rather than isolated glucose dysregulation, is already detectable in young adults with prediabetes. [21]

The study therefore argues for a shift in diabetes prevention towards younger populations, emphasising early identification of metabolic risk factors before progression to overt disease. The authors highlight the potential value of screening approaches that incorporate modifiable lifestyle risk factors and early metabolic monitoring, including blood pressure and lipid markers, alongside glucose measures. Such strategies, combined with physical activity promotion and age-specific health education, could enable earlier intervention and help reduce the long-term burden of diabetes and related cardiometabolic disease across the life course.

[4] BI-DIRECTIONAL IMPACT: PREDIABETES, DIABETES, ANXIETY & DEPRESSION

Prediabetes, Diabetes and Mental Health: Evidence for a Metabolic–Psychiatric Interface

A growing body of scientific literature indicates that prediabetes, diabetes, depression and anxiety frequently co-occur and may reinforce one another across the life course, suggesting that metabolic dysfunction and mental illness should not be considered in isolation within health systems. Evidence from multiple systematic reviews and meta-analyses shows that the association is detectable before diabetes is clinically diagnosed, strengthens once diabetes is established, and is increasingly observed in younger age groups. These findings indicate that mental health conditions are not peripheral complications but common comorbidities within diabetes populations, with implications for treatment adherence, quality of life and long-term disease management. Together, these findings have important implications for clinical screening protocols and the design of integrated metabolic–mental health interventions.

Evidence of association begins at the prediabetes stage. A meta-analysis by Yu and Wan (2024) found that individuals with prediabetes have a significantly higher prevalence of depressive symptoms compared with normoglycaemic populations. Importantly, the association was particularly evident among individuals younger than 50 years, suggesting that metabolic dysfunction and mental health vulnerability may emerge in parallel well before clinical diabetes is diagnosed. [22]

Once diabetes is established, the association becomes more pronounced. A systematic review and meta-analysis by Farooqi et al. (2022) found that depression occurs substantially more frequently among individuals with diabetes than among those without the condition. Their analysis estimated depression prevalence at approximately 22% among people with type 1 diabetes and

[21] Lee, S., Son, DS., Kim, JI. et al. Risk factors for prediabetes in young adults: a study based on Korea National health and nutrition examination survey data. Sci Rep 15, 27119 (2025). https://doi.org/10.1038/s41598-025-13378-5
[22] 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

19% among people with type 2 diabetes, compared with around 11–13% in populations without diabetes.[23]

Evidence from younger populations reinforces the same pattern, however they are particularly concerning because early-onset metabolic disease extends the lifetime duration of exposure to both metabolic and psychiatric morbidity. Malkawi et al.(2025) reviewed studies of adolescents with type 1 diabetes and found consistently elevated rates of both depression and anxiety, with psychological distress associated with poorer glycaemic control and higher HbA1c levels.[24] Similarly, McVoy et al. (2023) reviewed the literature on youth-onset type 2 diabetes, concluding that mental health disorders, including depression and anxiety, are common in this population and can significantly complicate disease management.[25]

Synthesising the field, Fanelli et al.(2025) argue that diabetes and depression should be understood as part of a bidirectional metabolic-psychiatric interface, in which each condition increases the risk and severity of the other.[26] The authors highlight multiple shared biological and behavioural pathways that may contribute to this relationship, including dysregulated glucose metabolism, chronic stress physiology, inflammatory signalling, disrupted sleep, sedentary behaviour and dietary factors. Importantly, psychiatric symptoms can impair adherence to diabetes self-management, while metabolic dysregulation may worsen psychological symptoms through both biological and psychosocial pathways.

Across these reviews, several consistent biological and clinical parameters emerge as likely contributors to the observed association. The most consistently reported marker is poor glycaemic control, reflected in elevated HbA1c levels and broader indicators of metabolic dysfunction. In addition, authors frequently discuss the role of insulin resistance, chronic inflammation, hypothalamic–pituitary–adrenal (HPA) axis activation, metabolic syndrome features (such as hypertension and dyslipidaemia), sleep disturbance, and lifestyle factors including diet and physical inactivity. While individual studies vary in the mechanisms emphasised, the literature increasingly supports the view that metabolic dysregulation and psychological distress are interconnected processes rather than independent conditions.

These findings have clear implications for clinical practice and health-system design. At present, patients presenting with depression or anxiety are typically assessed primarily through psychiatric frameworks, with metabolic risk factors often evaluated only later, if diabetes is suspected or diagnosed. However, the evidence reviewed above suggests that metabolic dysfunction may already be present in many individuals presenting with mental health symptoms, particularly among younger adults. Earlier identification of metabolic abnormalities may therefore provide an

[23] Farooqi A, Gillies C, Sathanapally H, Abner S, Seidu S, Davies MJ, Polonsky WH, Khunti K. A systematic review and meta-analysis to compare the prevalence of depression between people with and without Type 1 and Type 2 diabetes. Prim Care Diabetes. 2022 Feb;16(1):1-10. doi: 10.1016/j.pcd.2021.11.001.

[24] Malkawi S, Bani Hani S, Al shikh H. Depression and Anxiety among Adolescents with Type 1 Diabetes Mellitus: Systematic Review of Literature . Open Nurs J, 2025; 19: e18744346393599. http://dx.doi.org/10.2174/0118744346393599250908085129

[25] McVoy, M., Hardin, H., Fulchiero, E., Caforio, K., Briggs, F., Neudecker, M., & Sajatovic, M. (2023). Mental health comorbidity and youth onset type 2 diabetes: A systematic review of the literature. The International Journal of Psychiatry in Medicine, 58(1), 37-55.

[26] Fanelli G, Raschi E, Hafez G, Matura S, Schiweck C, Poluzzi E, Lunghi C. The interface of depression and diabetes: treatment considerations. Transl Psychiatry. 2025 Jan 24;15(1):22. doi: 10.1038/s41398-025-03234-5.

opportunity for preventive intervention before the onset of overt diabetes or the accumulation of complications.

A policy-relevant response would be to incorporate routine metabolic screening into the assessment of patients presenting with depression or anxiety, particularly where symptoms are recurrent, treatment-resistant, or accompanied by fatigue, sleep disturbance, weight gain or other cardiometabolic features. At minimum, such screening could include HbA1c or fasting glucose measurements, alongside broader cardiometabolic indicators such as blood pressure, lipid markers and anthropometric measures. Where clinically appropriate, additional assessment of nutritional status and micronutrient insufficiency may also be relevant, given emerging evidence linking nutrient deficiencies with both metabolic dysfunction and mental health outcomes.

Importantly, the literature reviewed here also emphasises the need for earlier intervention across the life course. The presence of metabolic dysfunction and psychiatric comorbidity in adolescents and young adults suggests that waiting until later adulthood, when diabetes complications are already established, misses a critical window for prevention. Earlier metabolic screening and targeted interventions may therefore help reduce both the lifetime burden of diabetes and the associated mental health burden, particularly as younger generations increasingly show signs of earlier metabolic dysregulation.

Taken together, these five reviews indicate that the co-occurrence of metabolic disease and mental illness is systematic, clinically significant, and detectable early in the disease trajectory. Health systems that continue to treat depression, anxiety and metabolic disease as largely separate domains risk missing opportunities for earlier diagnosis and more effective intervention. A more integrated approach, combining mental health assessment with metabolic screening and preventive metabolic interventions, may therefore represent a more appropriate response to the evolving evidence base.

[5] EXERCISE MORE? ADDRESSING THE FATIGUE PROBLEM

Fatigue is a common but physiologically complex clinical presentation. As one of the most common symptoms encountered in primary care, fatigue is typically characterised by low energy, reduced endurance, impaired cognitive performance, and poor recovery after exertion. Reviews of fatigue in clinical practice emphasise that it arises from a wide spectrum of physiological and psychological conditions, including sleep disorders, metabolic disease, nutritional deficiencies, endocrine disorders, medication side-effects, and chronic inflammation. Because fatigue is non-specific, clinical evaluation should prioritise identifying treatable underlying biological causes before recommending behavioural interventions alone.

Fatigue may arise from poor dietary patterns, particularly those characterised by high consumption of refined or ultra-processed foods. These diets tend to be low in micronutrients, fibre, and protein, and are associated with metabolic instability, inflammation, and cardiometabolic disease. Biologically, fatigue may emerge through several pathways:

- impaired mitochondrial energy production
- chronic low-grade inflammation
- glycaemic variability and insulin resistance
- inadequate protein for muscle and metabolic maintenance
- micronutrient depletion associated with refined diets

Fatigue is a common and clinically recognised feature of metabolic disorders such as diabetes. It can arise through several interacting mechanisms, including hyperglycaemia and glycaemic variability, chronic low-grade inflammation, sleep disturbance, psychological stress or depression, and diabetes-related complications such as neuropathy. These factors can impair energy metabolism, cognitive function, and physical endurance. In many cases fatigue may therefore represent an early manifestation of metabolic dysfunction, rather than simply a behavioural or motivational issue. Persistent fatigue should accordingly prompt consideration of metabolic screening, including HbA1c and related markers, to identify dysglycaemia or other underlying metabolic disturbances that may be contributing to reduced energy and wellbeing.

Poor digestion and digestive disorders can also contribute to fatigue by impairing nutrient absorption or increasing inflammatory burden. Gastrointestinal diseases such as inflammatory bowel disease and celiac disease are associated with fatigue through mechanisms including: iron deficiency, vitamin B12 and folate deficiency, systemic inflammation, disrupted sleep and pain. Digestive disorders can be provoked by chronic exposure to diets that are high in refined and industrially formulated ingredients.

Even in the absence of diagnosed disease, diets high in refined foods and low in fibre may impair gut microbial diversity and digestive efficiency, which can indirectly influence energy metabolism.

A substantial body of literature recognises the role of inadequate micronutrient status, particularly nutrients required for oxygen transport, mitochondrial energy metabolism, and neurotransmitter synthesis.

- Iron deficiency is one of the most common biological causes of fatigue globally. Iron is essential for hemoglobin synthesis and cellular oxygen transport, and deficiency reduces tissue oxygen delivery and physical work capacity. Fatigue and reduced endurance can occur even in iron deficiency without anaemia, and supplementation has been shown to significantly reduce fatigue symptoms in several trials.[27]
- Vitamin B12 and folate deficiencies impair red blood cell formation and neurological function, leading to weakness, fatigue, cognitive impairment, and reduced exercise tolerance. These nutrients are also involved in neurotransmitter synthesis and myelin maintenance, linking deficiency to both physical and mental fatigue.[28]
- Magnesium is a cofactor in hundreds of enzymatic reactions including ATP production and neuromuscular signalling. Deficiency has been associated with muscle weakness, lethargy, reduced physical performance, and fatigue.

[27] Tardy AL, Pouteau E, Marquez D, Yilmaz C, Scholey A. Vitamins and Minerals for Energy, Fatigue and Cognition: A Narrative Review of the Biochemical and Clinical Evidence. Nutrients. 2020 Jan 16;12(1):228. doi: 10.3390/nu12010228.

[28] Umekar M, Premchandani T, Tadoe A *et al.* 2025. Vitamin B12 deficiency and cognitive impairment: A comprehensive review of neurological impact, Brain Disorders, 18:100220 DOI: 10.1016/j.dscb.2025.100220

- Moderate deficiencies of nutrients such as vitamin C have been associated with poorer vitality and reduced physical functional health, indicating that fatigue can arise from combined micronutrient insufficiency rather than a single deficiency. [29]

[6] DOCTORS/CLINICIANS FACE BARRIERS TO ADDRESSING ROOT CAUSES

The above sections show the problems and challenges faced by clinicians and general practice doctors (GPs), which extend to managing multiple medical prescriptions, the side effects and interactions of the medicines, which unfortunately, is unable to remedy the origins of the conditions.

However, clinicians and GPs face barriers in optimising nutrition to support metabolic health.

Clinicians and GPs generally lack training in recognising and addressing nutritional or environmental drivers of chronic illness and applying nutritional strategies as therapeutic treatments. While medical curricula acknowledge the biochemical necessity of nutrients, they rarely teach their therapeutic applications, unless a nutrient has been classified as a pharmaceutical product.

Medical training emphasises avoidance of overt deficiency (the minimum), not restoration of optimal physiological function (the optimum). Consequently, therapeutic use of nutrients falls outside most clinicians' perceived scope of expertise, a gap that this policy seeks to address.

Pharmacological interventions typically target discrete biochemical pathways to suppress or modulate symptoms. Their evaluation through clinical trials assesses efficacy (e.g., symptom suppression, mortality reduction) and safety (e.g., incidence of adverse events).

Nutritional therapeutics, by contrast, act systemically, supporting metabolism, reducing inflammation, harmonising hormonal and immune function, and restoring cellular integrity.

The two approaches need not be seen as oppositional but complementary. Ethical practice requires that clinicians weigh both drug-mediated symptom relief and nutrient-mediated system restoration, asking whether the chosen intervention alleviates suffering without precipitating greater harm.

The lack of expertise and the barriers to higher nutrient prescribing, limits a clinician's capacity to identify reversible causes of suffering or to fulfil the duty of non-maleficence by correcting modifiable biological and environmental constraints before drug initiation. The professional preference to medical solutions can be observed in prescribing patterns. For example, antidepressant prescribing has rapidly increased in New Zealand since 2005, it crosses all age groups, and overlaps heavily with diabetes and metabolic disease populations.

If patients have been exposed to toxins or environmental exposures, doctors are largely unequipped to consider pathways for diagnosis, detoxification or therapeutic nutrition to reverse the symptoms presented before them. Medical education rarely provides doctors with sufficient expertise to engage in diagnostic pathways, and medical screening guidelines are largely unsuitable, should a patient present with a complex condition that they suspect was caused by a

[29] Tardy AL, Pouteau E, Marquez D, Yilmaz C, Scholey A. Vitamins and Minerals for Energy, Fatigue and Cognition: A Narrative Review of the Biochemical and Clinical Evidence. Nutrients. 2020 Jan 16;12(1):228. doi: 10.3390/nu12010228.

workplace or environmental exposure. The avenues for addressing environmental exposures are usually only open to individuals with the financial resources to pursue solutions.

In addition, regulatory system safety signals are not clear for doctors. If a nutrient is categorised as a medicine because it supports a therapeutic pathway, it does not have to exert any toxic risk, however as a medicine clinicians will, and do, infer toxic risk because a nutrient has been designated a medicine because of a therapeutic pathway. Clinicians are also unlikely to recommend nutrients at higher than maintenance levels, even if it addresses a known deficiency, as it puts them at risk of being sanctioned by the Medical Council of New Zealand for exceeding guideline recommendations.

MNZH aim to support integrative system change via the wider policy platform but also through the expansion of biomarker screening, the integration of health coaching into mainstream services, and the introduction of multinutrient supplementation as an evidence-based therapeutic intervention for people with psychiatric conditions.

[7] EXPANSION OF BIOMARKER SCREENING

A growing body of clinical and epidemiological literature supports expanding routine biochemical screening panels to better capture upstream metabolic and inflammatory risk, particularly in populations where chronic disease and multimorbidity are rising.

Expanding serum screening to include the suite in this paper's recommendations, reflects a shift toward upstream detection of metabolic and inflammatory dysfunction, rather than waiting for advanced disease to appear. Such an approach aligns with preventive medicine and public-health principles: identifying early biological signals, supporting metabolic resilience, and intervening before chronic illness becomes entrenched.

Traditional screening frameworks were designed largely to detect established disease: hyperlipidaemia, overt diabetes, or organ damage, rather than the earlier physiological disturbances that precede these conditions. Advances in metabolic science now allow more sensitive detection of early dysregulation through a broader but still clinically practical set of biomarkers. Expanding screening parameters is therefore not an exercise in excessive testing but an attempt to identify subclinical metabolic stress, inflammatory signalling, and nutrient insufficiency before these disturbances manifest as chronic disease.

Lipids: For lipid assessment, the conventional lipid panel (total cholesterol, LDL-C, HDL-C and triglycerides) remains useful but does not always capture the true burden of atherogenic particles. Measurement of apolipoprotein B (apoB) provides a direct estimate of the number of atherogenic lipoprotein particles and has been shown in large cohort studies and meta-analyses to be a stronger predictor of cardiovascular risk than LDL cholesterol alone. In parallel, lipoprotein(a) [Lp(a)] represents a largely genetically determined cardiovascular risk factor. Because levels remain relatively stable throughout life, most international guidelines now recommend measuring Lp(a) once in adulthood to identify individuals with markedly elevated inherited risk.

Diabetes risk: Glucose metabolism is another area where earlier detection is possible. Standard screening often relies on fasting glucose or HbA1c, which typically rise only after significant metabolic dysfunction has developed. Incorporating fasting insulin allows estimation of insulin

resistance, a central feature of metabolic syndrome and type-2 diabetes risk. Alternatively, the Triglyceride–Glucose (TyG) index, derived from fasting triglycerides and glucose, has been shown in multiple cohort studies to correlate closely with insulin resistance and cardiometabolic risk. Including one of these markers can therefore reveal metabolic impairment years before diabetes becomes clinically apparent.

Liver function: Liver biomarkers can similarly provide insight into early metabolic stress. Standard liver function tests detect overt hepatic injury, but adding gamma-glutamyl transferase (GGT) improves sensitivity to metabolic and oxidative stress, including early non-alcoholic fatty liver disease (NAFLD). In adults with metabolic risk factors, combining age, AST, ALT and platelet count to calculate the FIB-4 score allows simple risk stratification for liver fibrosis without immediate reliance on imaging. Because fatty liver disease is increasingly recognised as a hepatic manifestation of systemic metabolic dysfunction, identifying early liver changes can help detect broader cardiometabolic risk.

High-sensitivity C-reactive protein (hs-CRP) and serum uric acid: These markers provide valuable insight into systemic inflammatory and metabolic signalling. hs-CRP is a sensitive indicator of low-grade inflammation and has been associated in large prospective studies with increased risk of cardiovascular disease, diabetes and some neuropsychiatric disorders. Elevated hs-CRP may therefore reveal early inflammatory processes linked to metabolic syndrome, adipose tissue dysfunction and immune activation. Uric acid, historically associated primarily with gout, is now recognised as a marker and potential mediator of metabolic dysfunction. Elevated levels correlate with insulin resistance, hypertension, fatty liver disease and cardiovascular risk, and may signal disturbances in purine metabolism and oxidative stress before overt disease emerges. Including hs-CRP and uric acid can therefore help identify metabolic and inflammatory dysregulation at an earlier stage, providing an opportunity for preventive intervention.

Key Nutrients: Assessment of key nutrient biomarkers can strengthen the biological interpretation of metabolic risk and support early correction of deficiencies that influence physiological resilience. Nutrients such as vitamin D, vitamin B12, folate, iron (assessed through ferritin and transferrin saturation), magnesium, zinc and selenium participate in critical processes including mitochondrial energy metabolism, redox balance, immune function, neurotransmitter synthesis and endocrine regulation.

Nutrient screening can identify patterns of insufficiency, i.e. where a patient has a broadly inferior diet that cannot enable that person to maintain optimum metabolic and brain health. Low-income communities, young people having growth spurts, people with poor methylation, or individuals with gastrointestinal problems can be prioritised.

Inadequate levels of these nutrients have been associated with fatigue, impaired cognitive function, immune dysregulation and metabolic disturbance. While nutrient testing should be guided by clinical context, incorporating a targeted panel in populations at risk, such as those with fatigue, chronic illness, malabsorption, restrictive diets or pregnancy, can help identify correctable biological contributors to broader health problems.

Functional methylation impairment: A precautionary and proportionate approach for New Zealand would be targeted rather than universal screening for functional methylation impairment, integrating biochemical markers with selective genetic testing where clinically justified. The

primary goal should be early detection of correctable metabolic contributors to chronic disease risk, pregnancy complications, neurodevelopmental vulnerability, and persistent fatigue or neuropsychiatric symptoms.

New Zealand's clinical screening framework should incorporate biochemical assessment of one-carbon metabolism in defined risk groups by adding serum homocysteine, vitamin B12, folate and riboflavin status to existing laboratory panels in primary care. Where persistent hyperhomocysteinaemia or unexplained deficiency is identified, selective testing for common MTHFR polymorphisms (C677T and A1298C) may be considered to assist interpretation of metabolic vulnerability and guide nutritional management. Screening should be prioritised for:

- Women planning pregnancy or in early pregnancy, given the established link between folate metabolism, neural tube defects and adverse pregnancy outcomes.
- Individuals with recurrent pregnancy loss or unexplained obstetric complications.
- Patients with persistent hyperhomocysteinaemia or unexplained cardiovascular risk at younger ages.
- Individuals with chronic fatigue, cognitive dysfunction, or treatment-resistant mood disorders where nutrient insufficiency or methylation disturbance is suspected.
- People with malabsorption disorders, restrictive diets, or long-term medications affecting folate/B-vitamin metabolism.

The purpose of such targeted screening is not genetic diagnosis per se, but improved identification of metabolic and nutritional insufficiencies that are potentially correctable through diet, supplementation, or clinical management. This approach aligns with preventive medicine principles: detecting early biochemical dysfunction, supporting metabolic resilience, and reducing downstream disease burden while avoiding unnecessary population-wide genetic testing

[8] HEALTH COACHING TO IMPROVE NUTRITION

Dietary interventions that lower glucose and insulin levels frequently improve metabolic markers and psychiatric symptoms concurrently. This dual benefit challenges traditional treatment silos and supports a metabolic-first model of mental health stabilisation.

Emerging clinical audits and cohort evidence, including the New Zealand audit reported by Zinn et al. (2025), alongside a wider literature spanning mechanistic research, clinical case series, and population cohorts, indicate that integrating health coaching with carbohydrate-reduction approaches can improve glycaemic control, reduce medication dependence, and enhance overall

wellbeing. Evidence also suggests that programmes can support a deprescribing approach, to ensure that as metabolic markers improve, medication use is reduced.[30] [31] [32] [33] [34]

Taken together, this evidence suggests that wider deployment of health coaching is a proportionate and evidence-informed response to the escalating costs and complexity of multimorbidity.

A repeat pattern underpins New Zealand's chronic illness crisis - a crisis where brain related health conditions commonly co-exist with a range of inflammatory metabolic conditions. The science is now building a large and integrated picture, the drivers of harms at the mitochondrial level, predominantly and consistently include poor dietary intakes, including the consumption of high sugar and starch intakes.

Emerging evidence shows that insulin resistance, inflammation, and glycaemic volatility contribute to obesity, mood dysregulation, cognitive impairment, and fatigue. Stabilising blood glucose through carbohydrate reduction and whole-food nutrition improves energy regulation, emotional stability, and cognitive clarity.

Diets which are high in starch-based carbohydrates and ultraprocessed foods, tend to be low in the protein and fat macronutrient groups, and low in micronutrients, including vitamins and minerals

There is enormous evidence that high intakes of sugar and starchy foods may come under the description of a substance-use addiction.

But unlike alcohol and tobacco, people cannot avoid food! Health coaching supports a transition away from hyper-palatable and potentially addictive foods, to foods that improve a wide spectrum of metabolic parameters. This includes education and tips to support and manage difficult shifts in eating habits, while ensuring full autonomy of the patient.

Doctors and scientists now recognise that diets can be safely altered to lower the intake of sugary and starchy foods. These diets can ensure that people consume a range of healthy macronutrients from the carbohydrate, fat and protein groups that keep people not feeling hungry and snacking. These diets avoid carbohydrate forms that spike blood sugar, result in glucose levels going up and down, and produce addiction-like dopaminergic reactions in the brain.

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[30] 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

[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

[32] 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

[33] 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.

[34] 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

When addiction is in the background, it is no wonder that people struggle to stay on a healthy diet. A large body of evidence suggests that the impact of unstable blood glucose, elevated blood lipids and unstable insulin, produce systemic effects, including inflammation, diabetes and obesity.

Health coaching that supports dietary change supports brain health. People living with mental illness face disproportionate risks of metabolic syndrome, type 2 diabetes, and addiction disorders. Antipsychotic medications, socioeconomic disadvantage, addictive food environments, and glycaemic instability combine to drive poor outcomes. These risks are compounded by health service structures that treat mental health, addiction, and metabolic disease separately.

Health coaching within Health New Zealand currently focuses on supporting individuals receiving psychiatric services with daily functioning and service engagement, not on diet and nutrition. While valuable, this scope does not address the metabolic and behavioural drivers that contribute to both mental illness and chronic disease.

Expanding health coaching to include dietary transition support, carbohydrate reduction, and food addiction counselling supports people in learning, to adopt strategies when faced with 'cravings' and impulses, and with tips on how to shift to new dietary patterns without too much effort. Health coaching is well studied, and there are many doctors' clinics that have integrated health coaching into their practice. As such, health coaching offers a clinically coherent and cost-effective strategy to reduce diabetes risk and enable remission of prediabetes and diabetes, improve mental health stability, and support addiction recovery.

An expanded health coaching model recognises that metabolic dysfunction, addictive eating behaviours, and mental illness are interlinked conditions requiring integrated care.

Health coaches require training in metabolic health, low-carbohydrate dietary approaches, food addiction counselling, trauma-informed care, and cultural competency. Integration across primary care, mental health, and addiction services allows coordinated care and continuity of support.

Expected Outcomes and System Benefits

Nutritionally-integrative health coaching is expected to:

- reduce HbA1c and diabetes incidence
- improve blood pressure and cardiovascular risk
- stabilise mood and improve mental wellbeing
- reduce medication burden and polypharmacy
- improve addiction recovery outcomes
- reduce long-term healthcare costs

Food Addiction: The Missing Link in Behaviour Change

Highly processed starchy carbohydrates, including ultra-processed foods rich in refined sugars and starches, can stimulate dopamine reward pathways in ways that parallel mechanisms observed in substance addiction. Many individuals experience compulsive consumption, cravings, and relapse cycles consistent with addictive behaviour. Food addiction is most strongly

associated with ultra-processed foods, particularly those combining refined carbohydrates and fats.

Some processed starchy foods fall outside ultra-processed classifications yet still contribute to a cumulative carbohydrate burden. These foods can drive glycaemic volatility, promote hyperinsulinemia and hypertriglyceridemia, and contribute to recurrent blood glucose highs and lows.[35] Such metabolic instability may amplify reward signalling and reinforce cravings.

Food addiction frequently coexists with alcohol, drug, and behavioural addictions. Unlike alcohol or drugs, however, abstinence from food is neither possible nor desirable, which makes management uniquely challenging.

For this reason, health coaching focuses on reducing foods that destabilise blood glucose rather than targeting ultra-processed foods alone. Coaches support individuals to transition toward nutrient-dense whole foods, including adequate protein, healthy fats, and non-starchy carbohydrates that promote satiety and metabolic stability.

Without addressing addictive eating patterns, dietary advice often fails, reinforcing patient frustration and clinician pessimism. Integrating food-addiction counselling into health coaching enables individuals to understand reward-driven eating behaviours and develop practical, sustainable coping strategies.

Continuous Glucose Monitoring Devices

Continuous glucose monitoring (CGM) should be extended beyond intensive insulin users to include people with type 2 diabetes and prediabetes, particularly those with co-existing metabolic and mental health conditions. CGM provides real-time feedback on glycaemic responses to foods, stress, sleep disruption, and medication, enabling patients and clinicians to identify drivers of glucose volatility and tailor interventions accordingly.

For individuals experiencing mood instability, fatigue, or medication-related metabolic effects, visualising glucose patterns can improve self-management, reinforce behaviour change, and support safer medication titration. Evidence from behavioural and metabolic care settings indicates that CGM use enhances adherence, accelerates glycaemic improvement, and strengthens patient agency. Extending access within integrated care pathways would support earlier intervention, reduce long-term complications, and improve both metabolic and mental health outcomes.

Target Populations and Equity Considerations

Priority groups include individuals receiving mental health services, patients with prediabetes or diabetes, people prescribed antipsychotic medications, and those receiving addiction treatment. These populations carry the highest burden of metabolic disease and experience systemic barriers to dietary change.

Culturally responsive delivery through Māori and Pasifika providers is essential to ensure equity and effectiveness.

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[35] 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.

High-risk populations warrant prioritised, integrated metabolic support due to heightened vulnerability to long-term health complications and intergenerational effects. Young people under 25 with prediabetes or type 2 diabetes face early exposure to insulin resistance and elevated lifetime risk of cardiovascular, renal, and mental health disorders.

Pregnant women with pre-existing diabetes, prediabetes, or gestational dysglycaemia require intensive metabolic support to reduce risks to both mother and child, including pre-eclampsia, birth complications, and future metabolic disease in offspring.

Individuals living with prediabetes or diabetes alongside a diagnosed mental health condition represent a particularly high-risk group, as medication effects, socioeconomic stressors, addictive eating patterns, and glycaemic instability interact to worsen outcomes. Prioritising these populations for integrated health coaching, metabolic monitoring, and behavioural support offers a preventive, equity-focused strategy to reduce long-term morbidity and health system burden.

Evidence for Policy. NZ Study: Holistic Carbohydrate Reduction Model

A multi-year audit of three New Zealand primary care practices evaluated a holistic diabetes management model combining dietary change, health coaching, and community support. The findings provide real-world evidence that integrated lifestyle-focused care can improve glycaemic control and support remission in routine clinical settings.

The model centres on a three-pronged approach:

- Whole-food carbohydrate reduction to stabilise blood glucose and reduce insulin demand.
- Health-coach-led behaviour change support to improve adherence and patient agency.
- Community and peer support initiatives to reinforce education, motivation, and sustainability.

a. **Key outcomes.** The audit demonstrated meaningful metabolic improvements:
   - Median HbA1c decreased significantly across practices.
   - 32% of participants with type 2 diabetes achieved reversal.
   - 44% of those with prediabetes returned to normoglycaemia.
   - Weight loss was associated with greater HbA1c improvement.
   - Liver enzyme improvements suggested reduced metabolic stress.

Results were consistent across diverse real-world practice settings.

b. **Implementation insights.** Health coaching played a central role in translating dietary advice into sustained behaviour change:
   - personalised dietary education and practical guidance
   - motivational support and accountability
   - digital tools and group learning opportunities
   - integration with GP oversight and medication review

Community-based and peer initiatives strengthened engagement and helped sustain lifestyle change.

c. **Equity and population implications.** Diabetes prevalence and complications remain disproportionately high among Māori and Pacific peoples, reflecting social and economic inequities and barriers to culturally appropriate care.

   The audit included substantial Māori and Pacific participation and demonstrated clinically meaningful improvements in high-risk and socioeconomically disadvantaged populations.

d. **Key equity insights:**
   - culturally aligned, community-embedded delivery improves engagement
   - peer and whānau-based approaches enhance sustainability
   - locally tailored models are essential to reduce persistent health disparities

Policy relevance. The findings indicate that integrating health coaching with carbohydrate-reduction approaches within primary care is feasible, effective, and scalable. This model supports improved glycaemic control, reduced medication dependence, and improved mental wellbeing while taking into account individual glycaemic variability.

Implications for health system reform:

- supports diabetes remission and early intervention strategies.
- reduces reliance on medication-centred care.
- strengthens patient self-management and long-term adherence.
- offers a culturally adaptable pathway to reducing inequities.
- provides a practical model for addressing the growing burden of metabolic disease.

Together, this evidence supports wider implementation of holistic, health-coach-supported metabolic care as a proportionate and equity-focused response to New Zealand's diabetes burden.

Expanding health coaching to integrate metabolic health and food addiction support represents a practical, evidence-informed policy response to New Zealand's intertwined mental health, addiction, and diabetes crises. By addressing upstream drivers and supporting sustainable behaviour change, this approach strengthens patient agency, improves health outcomes, and reduces long-term system costs.

[8] MULTINUTRIENT SUPPLEMENTATION AS KEY HEALTH INTERVENTION

There is now a substantial and expanding literature showing that metabolic dysfunction and dietary and nutritional status are relevant across a wider range of psychiatric conditions, including but not limited to bipolar disorder, ADHD, schizophrenia and depression.

This paper couples health coaching and multinutrients as a first line intervention because of the close relationship of poor diets with nutrient deficiency, and the corresponding overlap between metabolic dysfunction and poor brain health. The field has shifted toward viewing psychiatric and brain-related conditions through a neuro-metabolic and immuno-inflammatory lens, although

causality remains complex and bidirectional.[36] [37] [38] The term 'nutritional psychiatry' is increasingly recognised in the scientific literature, with a recent position statement outlining a proposed definition (Marx et al 2026).[39]

> *'Nutritional Psychiatry is an evidence-based transdisciplinary investigation and transdiagnostic application of how diet and nutrients influence mental and brain health across the life course. Grounded in a biopsychosocial model with an emphasis on biological mechanisms, it encompasses prevention, treatment, and adjunctive care; integrates with standard therapies; supports both self-directed, peer-supported, and professionally guided approaches; spans individual and population-level interventions; and addresses both individual and structural dietary determinants.'*

Across case reports, observational cohorts, open-label trials, blinded randomised trials, and long-term safety follow-up, the direction of effect from micronutrient supplementation is strikingly consistent:

1) Lower diet quality, adverse eating behaviours and poor methylation of key nutrients are associated with psychiatric disorders.[40] [41] [42] [43]
2) Broad-spectrum multinutrient formulations are repeatedly associated with improvements in attention, mood, emotional regulation, aggression, stress symptoms, or general functioning;[44]
3) The adverse-effect profile reported in these studies is generally light, transient, and often gastrointestinal, rather than marked by the serious behavioural, neurological, or withdrawal concerns that accompany many psychotropic medicines.[45]

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[36] Malhi GS, Bassett D, Boyce P, Bryant R, Fitzgerald PB, Fritz K, et al. (2015) Royal Australian and New Zealand college of psychiatrists clinical practice guidelines for mood disorders. Aust N Z J Psychiatry, 49 (12) (2015 Dec 1), pp. 1087-1206

[37] Johnstone, J.M., Hughes, A., Goldenberg, J.Z., Romijn, A.R., Rucklidge, J.J. (2021) Multinutrients for the Treatment of Psychiatric Symptoms in Clinical Samples: A Systematic Review and Meta-Analysis of Randomized Controlled Trials. Nutrients 2020, 12, 3394. https://doi.org/10.3390/nu12113394

[38] Rucklidge JJ, Johnstone JM, Villagomez A, Ranjbar N, Kaplan BJ (2023) Broad Spectrum Micronutrients and Mental Health. Chapter 9. In Nutritional psychiatry: A primer for clinicians, pages 152-171. Edited by Ted Dinan. Cambridge University Press. DOI: 10.1017/978100929986

[39] Marx W, Mueller-Stierlin AS, Wallace C et al, (2026). What is Nutritional Psychiatry? Position statement from the International Society for Nutritional Psychiatry Research. Nutritional Psychiatry 2:100015. DOI: 10.1016/j.nupsyc.2026.100015

[40] Teasdale SB, Ward PB, Samaras K, et al. Dietary intake of people with severe mental illness: systematic review and meta-analysis. The British Journal of Psychiatry. 2019;214(5):251-259. doi:10.1192/bjp.2019.20

[41] Saghazadeh A, Mahmoudi M, Shahrokhi S et al (2020). Trace elements in schizophrenia: a systematic review and meta-analysis of 39 studies (N = 5151 participants), Nutrition Reviews, Volume 78, Issue 4, April 2020, Pages 278–303, https://doi.org/10.1093/nutrit/nuz059

[42] Firth F, Carney R, Stubbs B, et al (2018) Nutritional Deficiencies and Clinical Correlates in First-Episode Psychosis: A Systematic Review and Meta-analysis, Schizophrenia Bulletin, 44(6):1275–1292, DOI: 10.1093/schbul/sbx162

[43] Marano G, Boggio G, Abate F, et al. (2025). From Food to Mood: Psychological and Psychiatric Impact of Diet in Bipolar Disorder. Nutrients. 2025; 17(23):3728. https://doi.org/10.3390/nu17233728

[44] Chand, A., Darling, K. & Rucklidge, J.J. Duration effects of micronutrients in children with ADHD: Randomised controlled trial vs. Open-Label extension. Eur Child Adolesc Psychiatry 35, 463–475 (2026). https://doi.org/10.1007/s00787-025-02841-3

[45] Simpson JSA, Crawford SG, Goldstein ET, Field C, Burgess E, & Kaplan B. J. (2011). Systematic review of safety and tolerability of a complex micronutrient formula used in mental health. BMC Psychiatry, 11(62). http://www.biomedcentral.com/1471-244X/11/62

4) Broad-spectrum micronutrients are unlikely to interact with commonly prescribed medicines.[46]

Where an intervention repeatedly shows benefit across different designs and populations, and does so without a comparable signal of serious harm or a recognised withdrawal syndrome in the published studies, there is an ethical basis for offering it as a legitimate part of stepped care.[47]

Robust metabolic and nutritional status can influence the severity, resilience, and recovery trajectory of stress and mental illness. From a scientific and public-health perspective, the modulatory role of nutrition in brain health is well established, and particularly important in early and developmental periods.[48] Evidence for these modulatory roles arises from multiple converging domains: mechanistic studies of cellular and mitochondrial function; experimental and animal models; clinical case observations; epidemiological cohorts linking nutrient status to health outcomes; and controlled trials examining dietary patterns or micronutrient correction.

An increasingly broad range of psychiatric conditions benefit from nutritional, and more frequently broad spectrum treatments. These include:

- Anxiety and depression (including post- and ante-natal depression).[49] [50] [51] [52]
- ADHD[53] [54] [55] [56]

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[46] Kew BM, Doogue MP, McNeill R, et al. Investigation of a broad-spectrum micronutrient formulation as a possible precipitant of pharmacokinetic micronutrient–drug interactions. Br J Clin Pharmacol. 2025;91(7):1987-1995. doi:10.1002/bcp.70014

[47] Sarris J, Logan AC, Amminger GP, et al. (2015). Nutritional Medicine as Mainstream in Psychiatry: A Consensus Position Statement from The International Society for Nutritional Psychiatry Research (ISNPR). Lancet Psychiatry, 2, 271-274. http://www.thelancet.com/journals/lanpsy/article/PIIS2215-0366(14)00051-0/abstract

[48] Rucklidge, J. J., Johnstone, J. M., & Kaplan, B. J. (2021). Nutrition Provides the Essential Foundation for Optimizing Mental Health. Evidence-Based Practice in Child and Adolescent Mental Health, 6(1), 131–154. https://doi.org/10.1080/23794925.2021.1875342

[49] Blampied M, Bell C, Gilbert C & Rucklidge JJ (2020). Broad spectrum micronutrient formulas for the treatment of symptoms of depression, stress, and/or anxiety: a systematic review, Expert Review of Neurotherapeutics, 20:4, 351-371, DOI: 10.1080/14737175.2020.1740595

[50] Mitchell, M., Bradley, H., Blampied, N.M., Mulder, R.T., Rucklidge, J.J. (2025). Protective effect of micronutrients used to treat antenatal depression on rates of postnatal depression at six months: A secondary analysis of NUTRIMUM. Journal of Affective Disorders 388 (2025) 119560. DOI: 10.1016/j.jad.2025.119560

[51] Bradley HA, Moltchanova E, Mulder RT, Dixon L, Henderson J, Rucklidge JJ. (2024) Efficacy and safety of a mineral and vitamin treatment on symptoms of antenatal depression: 12-week fully blinded randomised placebo-controlled trial (NUTRIMUM). BJPsych Open. 2024 Jun 3;10(4):e119. doi: 10.1192/bjo.2024.706.

[52] Kimball, S., Mirhosseini, N., & Rucklidge, J. J. (2018). Database Analysis of Depression and Anxiety in a Community Sample-Response to a Micronutrient Intervention. Nutrients, 10(2):152. http://www.mdpi.com/2072-6643/10/2/152

[53] Lange, K. W., Lange, K. M., Nakamura, Y., & Reissmann, A. (2023). Nutrition in the Management of ADHD: A Review of Recent Research. Current Nutrition Reports, 12(3), 383–394. https://doi.org/10.1007/s13668-023-00487-8

[54] Pinto, S., Correia-de-Sá, T., Sampaio-Maia, B., Vasconcelos, C., Moreira, P., & Ferreira-Gomes, J. (2022). Eating Patterns and Dietary Interventions in ADHD: A Narrative Review. Nutrients, 14(20), 4332. https://doi.org/10.3390/nu14204332

[55] Rucklidge, J. J., Eggleston, M. J. F., Boggis, A., Darling, K., Gorman, B., & Frampton, C. M. (2021). Do Changes in Blood Nutrient Levels Mediate Treatment Response in Children and Adults With ADHD Consuming a Vitamin–Mineral Supplement? Journal of Attention Disorders, 25(8), 1107–1119. https://doi.org/10.1177/1087054719886363

[56] Rucklidge, J. J., Eggleston, M. J. F., Johnstone, J. M., Darling, K., & Frampton, C. M. (2018). Vitamin-mineral treatment improves aggression and emotional regulation in children with ADHD: A fully blinded, randomized, placebo-controlled trial. Journal of Child Psychology and Psychiatry, 59(3), 232–246. https://doi.org/10.1111/jcpp.12817

- Psychosis including Schizophrenia[57] [58] [59]
- Obsessive Compulsive Disorder[60]
- Bipolar Disorder[61]
- Paediatric and Adolescent Psychiatric and Brain-related conditions.[62] [63] [64]

Nutrition: Biological Key to Brain Resilience

Nutrition is increasingly recognised as a contributor to brain resilience, including to support people who are experiencing short- and long-term trauma and distress.[65]

Human biology operates through networks of enzymes, cofactors and signalling molecules in which vitamins, minerals, amino acids and fatty acids function as essential participants in cellular and mitochondrial processes. These nutrients are not foreign pharmacological agents but recognised biological substrates integrated into human physiology, influencing energy metabolism, immune signalling, neurotransmitter synthesis, endocrine regulation and epigenetic expression.

Nutrients are essential for neurotransmitter synthesis, mitochondrial energy production, antioxidant defence and immune regulation. Reviews in nutritional psychiatry report associations between poor diet quality and higher risk of depression, while nutrient deficiencies, including iron, B-vitamins, zinc, omega-3 fatty acids and vitamin D, are linked with fatigue, impaired cognition and mood dysregulation.

Reviews in *The Lancet Psychiatry* and *World Psychiatry* increasingly recognise psychiatric conditions as involving interacting systems - metabolism, immune function, endocrine signalling and brain circuitry, rather than isolated neurotransmitter deficits. Nutritional status intersects with these systems through its role in mitochondrial function, redox balance, neurotransmitter

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[57] Fornaro, M., Caiazza, C., Billeci, M. et al. Nutraceuticals and phytoceuticals in the treatment of schizophrenia: a systematic review and network meta-analysis “Nutra NMA SCZ”. Mol Psychiatry 30, 168–187 (2025). https://doi.org/10.1038/s41380-024-02645-y

[58] Suschana E, Anderson T, Hong C, Narikatte A, Silverberg J and Sharma MS (2025) The role of anti-inflammatory diets and supplementation in metabolic syndrome and symptom remission in adults with schizophrenia: a systematic review. Front. Psychiatry 15:1506353. doi: 10.3389/fpsyt.2024.1506353

[59] Kaplan, B. J., Isaranuwatchai, W., & Hoch, J. S. (2017). Hospitalization cost of conventional psychiatric care compared to broad-spectrum micronutrient treatment: literature review and case study of adult psychosis. Int J Ment Health Syst, 11, 14. https://link.springer.com/article/10.1186/s13033-017-0122-x

[60] 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

[61] Gabriel FC, Oliveira M, De M. Martella B,et al. (2023). Nutrition and bipolar disorder: a systematic review. Nutritional Neuroscience, 26(7), 637–651. https://doi.org/10.1080/1028415X.2022.2077031

[62] Rucklidge JJ, Bruton A, Welsh A et al. (2024). Annual Research Review: Micronutrients and their role in the treatment of paediatric mental illness. J Child Psychology and Psychiatry. 66(4):477-497. DOI: 10.1111/jcpp.14091

[63] Rucklidge JJ, Sherwin AH, Mulder RT, Manna L & Boden JM. (2026) Efficacy and Safety of Micronutrient Treatment for Irritability in Teenagers: 8-Week Double-Blinded Randomized Placebo-Controlled Trial (BEAM). Journal of the American Academy of Child & Adolescent Psychiatry.

[64] Sole, E. J., Rucklidge, J. J., & Blampied, N. M. (2017). Anxiety and Stress in Children Following an Earthquake: Clinically Beneficial Effects of Treatment with Micronutrients. Journal of Child and Family Studies, 1-10. doi: 10.1007/s10826-016-0607-2
https://link.springer.com/article/10.1007%2Fs10826-016-0607-2

[65] Rucklidge JJ, Usman Azfali M, Kaplan BJ et al (2021). Massacre, Earthquake, Flood: Translational science evidence that the use of micronutrients post-disaster reduces the risk of post-traumatic stress in survivors of disasters. International Perspectives in Psychology 10(1):39–54. DOI: 10.1027/2157-3891/a000003.

synthesis, and immune regulation, providing a plausible pathway by which deficiency or dietary patterns can influence symptom expression and course. [66] [67] [68]

Importantly, the literature does not support a single unified cause across disorders. Instead, the current evidence supports a model in which metabolic dysfunction and nutrient insufficiency act as amplifiers or modifiers of psychiatric illness, influencing onset, severity, treatment response and long-term outcomes. This perspective has practical implications: it strengthens the case for routine assessment of metabolic health and key nutrient status in psychiatric populations, and for integrating metabolic and nutritional strategies alongside conventional pharmacological and psychological care. Individuals with metabolic syndrome or impaired glucose regulation consistently demonstrate greater symptom severity, increased fatigue and cognitive impairment, and higher relapse risk.[69] [70] [71]

Sleep and stress physiology provide another mechanistic pathway connecting metabolic health and psychological functioning. Disruption of sleep alters emotional regulation circuits and activates the hypothalamic-pituitary-adrenal (HPA) axis, elevating cortisol and impairing cognitive processing of stress. Sleep loss increases amygdala reactivity and reduces prefrontal regulation of emotion, contributing to anxiety, fatigue and impaired coping.[72] Because metabolic instability, inflammation and circadian disruption interact with the HPA axis, researchers increasingly describe a stress-sleep-metabolic feedback loop influencing mood and cognition.[73] [74] [75] [76] [77]

Finally, behavioural cycles such as addiction may reinforce metabolic dysregulation and prolong psychological distress, and providing support for health coaching in an integrated approach to supporting metabolic health in New Zealand. Research on reward neurobiology shows that highly palatable foods, alcohol and other substances activate dopamine pathways in ways that can sustain compulsive behaviour and emotional dysregulation. When combined with poor sleep,

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[66] Milaneschi Y, Simmons WK, van Rossum EFC, Penninx BW. Depression and obesity: evidence of shared biological mechanisms. Mol Psychiatry. 2019 Jan;24(1):18-33. doi: 10.1038/s41380-018-0017-5

[67] Penninx B, Lamers F, Jansen R et al. (2024). Immuno-metabolic depression: from concept to implementation The Lancet Regional Health – Europe, 2024; 48

[68] Chourpiliadis C, Zeng Y, Lovik A, et al. Metabolic Profile and Long-Term Risk of Depression, Anxiety, and Stress-Related Disorders. JAMA Netw Open. 2024;7(4):e244525. doi:10.1001/jamanetworkopen.2024.4525

[69] Qiu W, Cai X, Zheng C, Qiu S, Ke H, Huang Y. Update on the Relationship Between Depression and Neuroendocrine Metabolism. Front Neurosci. 2021 Aug 31;15:728810. doi: 10.3389/fnins.2021.728810.

[70] Al-Khatib Y, Akhtar MA, Kanawati MA, Mucheke R, Mahfouz M, Al-Nufoury M. Depression and Metabolic Syndrome: A Narrative Review. Cureus. 2022 Feb 12;14(2):e22153. doi: 10.7759/cureus.22153.

[71] Ocon AJ (2013) Caught in the thickness of brain fog: exploring the cognitive symptoms of Chronic Fatigue Syndrome. Front. Physiol. 4:63. doi: 10.3389/fphys.2013.00063

[72] Walker MP, van der Helm E. Overnight therapy? The role of sleep in emotional brain processing. Psychol Bull. 2009 Sep;135(5):731-48. doi: 10.1037/a0016570.

[73] Kim TW, Jeong JH, Hong SC. The impact of sleep and circadian disturbance on hormones and metabolism. Int J Endocrinol. 2015;2015:591729. doi: 10.1155/2015/591729.

[74] Goldstein AN, Walker MP. The role of sleep in emotional brain function. Annu Rev Clin Psychol. 2014;10:679-708. doi: 10.1146/annurev-clinpsy-032813-153716.

[75] Delpino FM, Figueiredo LM, Flores TR. et al. (2023). Intake of ultra-processed foods and sleep-related outcomes: A systematic review and meta-analysis. Nutrition, 106:111908. DOI: 10.1016/j.nut.2022.111908

[76] Zhao M, Tuo H, Wang S, Zhao L (2020). The Effects of Dietary Nutrition on Sleep and Sleep Disorders. Mediators of Inflammation. 2020 Jun 25;2020:3142874. DOI:10.1155/2020/3142874

[77] Lothian, J. A, Blampied, N., & Rucklidge, J. J. (2016). Effect of Micronutrients on Insomnia in Adults: A Multiple-Baseline Design. Clinical Psychological Science. http://cpx.sagepub.com/content/early/2016/05/21/2167702616631740.abstract

stress and metabolic instability, these cycles may reinforce fatigue, cognitive fog and reduced psychological resilience.

Contemporary models of mental illness therefore increasingly emphasise systems interactions among metabolism, inflammation, sleep, nutrition and social stressors, suggesting that maintaining metabolic health may improve resilience and recovery capacity, even though it is only one component within a multifactorial picture of mental illness.

University of Canterbury Programme: Micronutrients for Mental Health

The clinical and exploratory work from Te Puna Toiora, the Mental Health and Nutrition Research Lab at the University of Canterbury, led by Professor Julia Rucklidge, has added to and informed a consistent body of evidence that demonstrates that broad-spectrum micronutrients can produce clinically relevant gains with a comparatively benign safety profile.

Early research on ADHD showed that mood stability, attention, irritability, and overall functioning improved in trial participants taking broad-spectrum micronutrient formulas.[78] In adults with ADHD, the 2014 British Journal of Psychiatry trial found preliminary evidence of efficacy and explicitly reported a 'reassuring safety profile', with no group differences in adverse events between micronutrients and placebo.[79] In children with ADHD, the 2018 fully blinded placebo-controlled trial found improvements in overall functioning, reduced impairment, better emotional regulation, reduced aggression, and improved inattention, and reported no serious adverse events and no adverse-event excess relative to placebo.[80] The 2019 long-term safety study likewise reported that the formulas were safe, well tolerated, and generally effective, with no clinically significant adverse effects identified.[81]

More recently, the NoMAD randomised controlled trial demonstrated that micronutrients led to faster recovery from depression and anxiety compared with placebo. This RCT was followed by a 10 week open-label component and then a one year naturalistic review.[82] In prenatal and antenatal

[78] Rucklidge J, Taylor M, Whitehead K. Effect of micronutrients on behavior and mood in adults With ADHD: evidence from an 8-week open label trial with natural extension. J Atten Disord. 2011 Jan;15(1):79-91. doi: 10.1177/1087054709356173.

[79] Rucklidge JJ, Frampton CM, Gorman B, Boggis A. Vitamin-mineral treatment of attention-deficit hyperactivity disorder in adults: double-blind randomised placebo-controlled trial. Br J Psychiatry. 2014;204:306-15. doi: 10.1192/bjp.bp.113.132126.

[80] Rucklidge JJ et al (2018). Vitamin-mineral treatment improves aggression and emotional regulation in children with ADHD: A fully blinded, randomized, placebo-controlled trial. Journal of Child Psychology and Psychiatry.

[81] Rucklidge JJ, Eggleston MJF, Ealam B, Beaglehole B, Mulder RT. An Observational Preliminary Study on the Safety of Long-Term Consumption of Micronutrients for the Treatment of Psychiatric Symptoms. The Journal of Alternative and Complementary Medicine: Paradigm, Practice, and Policy Advancing Integrative Health. 2019;25(6):613-622. doi:10.1089/acm.2018.0352

[82] Coët A, Blampied M, Rucklidge JJ. (2025) 1-year naturalistic follow-up of a Randomised Double-Blind, Placebo-Controlled Trial ("NoMAD") Exploring the Effectiveness of Micronutrients in Improving Symptoms of Anxiety and Depression. Journal of Affective Disorders Reports, 20:100913, DOI: 10.1016/j.jadr.2025.100913

depression, the 2024 NUTRIMUM randomised trial reported no evidence of harm while also showing benefit in functioning. [83] [84]

The likely biochemical pathways reflect real-world biological interactions. These formulations do not act like single-target receptor drugs. Rather, they supply multiple vitamin and mineral cofactors required for mitochondrial energy production, neurotransmitter synthesis, methylation, antioxidant defence, immune regulation, neuronal membrane stability, and enzyme function. That systems-level rationale matters because psychiatric symptoms often arise in biologically complex states involving poor diet, inflammation, stress, sleep disruption, glycaemic dysregulation, and suboptimal nutrient status.

The Canterbury programme reflects a neuroscience, rather than conventional psychiatric approach, viewing mental distress not simply as a neurotransmitter deficit to be pharmacologically treated, but as a potential manifestation of broader metabolic and neurobiological dysregulation that may be responsive to improved nutrient sufficiency.

On adverse effects, the pattern across the accessible papers is notably mild. Where side effects are reported, they are usually general gastrointestinal issues such as nausea, stomach upset, appetite change, or transient discomfort, the sort of tolerability issues commonly seen across many nutrient interventions. Micronutrients are unlikely to adversely affect co-prescribed medications. But the evidence available so far supports the claim that the safety concerns are not of the same order as those attached to many psychotropics.[85] [86] [87]

In the major controlled ADHD trials of children and adults, adverse events were systematically monitored, and the reported findings did not show a serious treatment-related harm signal.

The Canterbury studies consistently show that recipients often improve, sometimes across multiple symptom domains and functioning measures, while the harms reported are usually negligible or limited to mild gastrointestinal effects. When the alternative standard-care pathway can involve medicines with serious neuropsychiatric adverse effects, boxed suicidality warnings in youth, and withdrawal concerns, it is difficult to justify treating multinutrient approaches as peripheral or unserious. A proportionate, evidence-based response would be to recognise broad-spectrum multinutrients as a legitimate low-harm intervention, especially within a stepped model that begins with metabolic and nutritional assessment, identifies underlying drivers, and reserves higher-risk pharmacological interventions for cases where they are clearly necessary. That

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[83] Bradley HA, Moltchanova E, Mulder RT, Dixon L, Henderson J, Rucklidge JJ. (2024) Efficacy and safety of a mineral and vitamin treatment on symptoms of antenatal depression: 12-week fully blinded randomised placebo-controlled trial (NUTRIMUM). BJPsych Open. 2024 Jun 3;10(4):e119. doi: 10.1192/bjo.2024.706.

[84] Kimball, S., Mirhosseini, N., & Rucklidge, J. J. (2018). Database Analysis of Depression and Anxiety in a Community Sample-Response to a Micronutrient Intervention. Nutrients, 10(2):152. http://www.mdpi.com/2072-6643/10/2/152

[85] Kew BM, Doogue MP, McNeill R, et al. Investigation of a broad-spectrum micronutrient formulation as a possible precipitant of pharmacokinetic micronutrient–drug interactions. Br J Clin Pharmacol. 2025;91(7):1987-1995. doi:10.1002/bcp.70014

[86] Rucklidge JJ et al. (2021). Nutrition Provides the Essential Foundation for Optimizing Mental Health.

[87] Coët A et al (2025) 1-year naturalistic follow-up of a Randomised Double-Blind, Placebo-Controlled Trial (“NoMAD”) Exploring the Effectiveness of Micronutrients in Improving Symptoms of Anxiety and Depression.

position is scientifically defensible and ethically stronger than a system that defaults too quickly to drugs while under-recognising nutritional therapeutics. [88]

When independent lines of evidence converge across levels of biological organisation, the resulting picture meets a core principle of scientific reasoning, consilience. In public health and medical ethics, this convergence matters: recognising modulation as scientifically meaningful reflects evidence-based reasoning rather than reductionist dogma, acknowledging that human health is regulated through complex biological systems and that safeguarding the nutrient foundations of those systems is a legitimate and rational objective of modern public health.

Micronutrient use is not associated with withdrawal syndrome.

Informed Consent for Psychiatric Medication, When Risks include Suicidality

A substantial body of historical and contemporary evidence demonstrates that antidepressant use, particularly in younger populations, is associated with clinically significant safety signals that warrant careful scrutiny. Work by David Healy and others has drawn attention to behavioural activation syndromes, including akathisia, which can present as profound agitation, emotional destabilisation, and in some cases increased suicidality.[89] Akathisia is a recognised adverse effect of psychotropic medications that may occur not only during treatment initiation or dose escalation, but also following dose reduction or discontinuation. This phenomenon, often termed 'withdrawal akathisia', is well described in the literature and can be clinically indistinguishable from acute akathisia.[90]

The U.S. Food and Drug Administration pooled analysis of paediatric trials identified an increased risk of suicidal ideation and behaviour in children and adolescents treated with antidepressants, findings subsequently reinforced by meta-analyses of clinical trial data. [91] [92] [93] More recent systematic reviews drawing on full clinical study reports rather than published summaries have confirmed elevated risks of suicidality and aggression in younger populations.[94] In parallel, the evidence base now recognises that antidepressants can produce withdrawal or discontinuation effects that may be prolonged and clinically significant.[95] Taken together, this literature identifies

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[88] Rucklidge JJ, Johnstone JM, Villagomez A, Ranjbar N, Kaplan BJ (2023) Broad Spectrum Micronutrients and Mental Health. Chapter 9. In Nutritional psychiatry: A primer for clinicians, pages 152-171. Edited by Ted Dinan. Cambridge University Press. DOI: 10.1017/978100929986

[89] Healy D, Herxheimer A, Menkes DB (2006) Antidepressants and Violence: Problems at the Interface of Medicine and Law. PLoS Med 3(9): e372. https://doi.org/10.1371/journal.pmed.0030372

[90] Pringsheim T, Gardner D, Addington D, (2018). The Assessment and Treatment of Antipsychotic-Induced Akathisia. Can J Psychiatry. 2018 Nov;63(11):719-729. doi: 10.1177/0706743718760288.

[91] Hammad TA, Laughren T, Racoosin J. (2006) Suicidality in pediatric patients treated with antidepressant drugs. Arch Gen Psychiatry. 2006 Mar;63(3):332-9. doi: 10.1001/archpsyc.63.3.332.

[92] Bridge JA, Iyengar S, Salary CB, Barbe RP, Birmaher B, Pincus HA, Ren L, Brent DA.(2007). Clinical response and risk for reported suicidal ideation and suicide attempts in pediatric antidepressant treatment: a meta-analysis of randomized controlled trials. JAMA. 2007 Apr 18;297(15):1683-96. doi: 10.1001/jama.297.15.1683.

[93] FDA (2018). Suicidality in Children and Adolescents Being Treated With Antidepressant Medications. U.S. Food and Drug Administration. https://www.fda.gov/drugs/postmarket-drug-safety-information-patients-and-providers/suicidality-children-and-adolescents-being-treated-antidepressant-medications

[94] Sharma T, Guski L S , Freund N, Gøtzsche PC.(2016) Suicidality and aggression during antidepressant treatment: systematic review and meta-analyses based on clinical study reports BMJ 2016; 352 :i65 doi:10.1136/bmj.i65

[95] Horowitz MA, Taylor D. (2019) Tapering of SSRI treatment to mitigate withdrawal symptoms. Lancet Psychiatry. 2019;6(6):538–546. doi:10.1016/S2215-0366(19)30032-X

consistent limitations in trial duration, adverse-event detection, and reporting practices, and establishes that these risk signals are real, reproducible, and clinically relevant. While antidepressants may provide benefit for some patients, ethical and evidence-based practice requires that these risks are transparently acknowledged and weighed alongside alternative approaches, particularly in populations where vulnerability is highest.

CONCLUSION: METABOLIC HEALTH REFORM - A GOVERNANCE IMPERATIVE

There is also a unique capacity consideration for New Zealand. Governments often defer to larger jurisdictions when setting regulatory and clinical standards. However, New Zealand is home to internationally recognised expertise in metabolic approaches to metabolic health, including Professors Grant Schofield and Caryn Zinn, and nutritional approaches to mental health, including the multinutrient research programme based at the University of Canterbury and led by Professor Julia Rucklidge. Where credible domestic expertise exists, there is a reasonable argument that the country should exercise intellectual leadership rather than rely solely on external policy models. Smaller jurisdictions have historically led innovation in public health precisely by acting on strong local research capacity.

Recent advances in data science, analytical capacity, and digital research tools further strengthen the case for a more proactive national approach. Modern public health agencies now have access to large administrative datasets, advanced statistical methods, and rapidly evolving analytical tools including machine learning and artificial intelligence, which significantly expand the ability to analyse health trends, evaluate interventions, and synthesise global scientific literature. These capabilities allow agencies to conduct independent evidence assessments, monitor emerging risks, and evaluate policy effectiveness in real time. As a result, the historical need for smaller jurisdictions to rely heavily on regulatory conclusions from larger countries is diminishing. This is particularly relevant where overseas guidelines may be narrowly framed, based on older datasets, or not reflective of New Zealand's population characteristics, dietary patterns, health inequities, or environmental conditions. Modern analytical tools make it increasingly feasible for New Zealand institutions to undertake their own evidence reviews and policy modelling tailored to local circumstances.

The issue extends beyond healthcare systems into the broader domains of economic productivity and societal wellbeing. Metabolic and mental health conditions affect workforce participation, educational attainment and long-term social outcomes. If nutritional and metabolic interventions can contribute to improved functioning or resilience, even modestly, the benefits could extend beyond clinical outcomes to include reduced healthcare costs, improved productivity and stronger community wellbeing.

Taken together, these considerations suggest that nutritional and metabolic approaches to mental health should not be viewed merely as alternative or complementary therapies. Rather, they raise a legitimate public governance question: whether health systems should expand their frameworks to incorporate biological and nutritional determinants of mental health alongside conventional psychiatric care. Framed in this way, the issue becomes not simply an ethical opportunity but a governance imperative, grounded in governance responsibility grounded in administrative law principles, statutory health duties, and broader constitutional commitments.