



ACTIVIT (DOCTORS PRESCRIBED): A CLINICALLY OPTIMIZED, METHYLATED MULTIVITAMIN FOR SUPERIOR MICRONUTRIENT BIOAVAILABILITY AND FUNCTIONAL HEALTH

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Abstract

Micronutrient deficiencies remain a pervasive global health concern despite widespread use of multivitamin supplements. Emerging evidence suggests that persistent deficiencies are driven not only by inadequate intake but also by declining nutrient density of food, impaired gastrointestinal absorption, increased physiological demand, pharmaceutical interference, and genetic polymorphisms affecting nutrient metabolism. Conventional multivitamin formulations frequently rely on synthetic vitamin forms and inorganic mineral salts, which may demonstrate limited bioavailability and metabolic utilization. In particular, reduced activity of the methylenetetrahydrofolate reductase (MTHFR) enzyme compromises folate metabolism and methylation capacity in a substantial proportion of the population.

The present randomized, double-blind, controlled experimental study evaluated ACTIVIT (Doctors Prescribed), a clinically optimized multivitamin containing methylated B-vitamins and amino acid-chelated minerals, for its effects on micronutrient bioavailability and functional health outcomes. One hundred twenty healthy adults were randomized to receive either ACTIVIT (Doctors Prescribed) (n = 60) or a conventional multivitamin (n = 60) for 12 weeks. Primary outcomes included serum vitamin B12, red blood cell (RBC) folate, plasma homocysteine, serum magnesium, and zinc. Secondary outcomes included fatigue severity scores.

ACTIVIT(Doctors Prescribed) supplementation resulted in significantly greater increases in serum B12 ($\uparrow 42.6\%$, $p < 0.001$) and RBC folate ($\uparrow 38.2\%$, $p < 0.001$), along with a significant reduction in plasma homocysteine (-21.4% , $p < 0.001$) compared with controls. Chelated mineral delivery produced higher serum magnesium ($\uparrow 19.3\%$, $p = 0.002$) and zinc levels ($\uparrow 17.8\%$, $p = 0.004$). Participants receiving ACTIVIT also demonstrated a marked improvement in fatigue severity scores (-31.6% , $p < 0.001$). These findings support the clinical relevance of bioactive vitamin forms and chelated minerals for addressing modern micronutrient insufficiency.

Keywords: methylated vitamins, chelated minerals, micronutrient deficiency, MTHFR polymorphism, homocysteine metabolism, functional health

Introduction

Micronutrients are fundamental to human physiology, serving as enzymatic cofactors, regulators of gene expression, structural components of tissues, and modulators of neurotransmission, immune function, oxidative balance, and cellular energy production. Historically, severe micronutrient deficiencies manifested as overt clinical syndromes such as scurvy, rickets, pellagra, and beriberi. The identification of vitamins and minerals during the late nineteenth and early twentieth centuries

revolutionized public health, leading to fortification programs and significant reductions in classical deficiency diseases.¹⁻³

Despite these advances, micronutrient deficiencies have not been eliminated. Instead, they have evolved into a subtler but more pervasive form often described as “hidden hunger.” In this state, individuals may consume sufficient calories and appear clinically normal while experiencing inadequate intracellular availability of essential vitamins and minerals. This form of deficiency does not immediately produce dramatic symptoms but progressively destabilizes metabolic, neurological, cardiovascular, endocrine, and immune systems.⁴⁻⁵

Contrary to the assumption that micronutrient deficiencies are confined to undernourished populations, contemporary evidence demonstrates that they are widespread across both low- and high-income regions. Individuals who consume “balanced” diets, engage in regular exercise, and maintain laboratory values within conventional reference ranges may still exhibit functional deficiencies due to impaired absorption, inefficient metabolic activation, increased physiological demand, medication use, and genetic variability.⁶

Global Burden of Micronutrient Deficiencies

Micronutrient deficiencies represent one of the most significant yet underestimated global public health challenges. Large-scale population analyses indicate that approximately 56% of preschool-aged children worldwide exhibit deficiency in at least one essential micronutrient, most commonly iron, zinc, or vitamin A. Among women of reproductive age, the burden is even higher, with nearly 69% globally affected by at least one deficiency involving iron, zinc, or folate.

Dietary intake modeling based on global food consumption data suggests that more than 65% of the world’s population fails to meet recommended iron intake. Similarly, approximately 55% consume insufficient riboflavin, 54% inadequate folate, and 53% insufficient vitamin C. Deficiencies of iodine, calcium, and vitamin E affect an estimated 60–70% of individuals worldwide, illustrating that micronutrient inadequacy is not confined to any single nutrient or region.

Regional disparities are pronounced. In South Asia and Sub-Saharan Africa, micronutrient deficiency prevalence among children aged 6–59 months ranges from 57% to over 60%, compared with approximately 45–50% in Europe, Central Asia, and Latin America. Women in these regions frequently exhibit deficiency rates exceeding 70%, driven by limited dietary diversity, food insecurity, recurrent infections, and increased nutritional demands during pregnancy and lactation.

Importantly, high-income countries are not exempt. Surveys in developed regions reveal that substantial proportions of populations fail to achieve adequate intake of vitamin D, magnesium, and B-vitamins. Nearly half of women and children in some high-income settings demonstrate at least one micronutrient deficiency when assessed using biochemical markers, underscoring the global nature of hidden hunger.

Modern Causes of Micronutrient Deficiency

Declining Nutrient Density of Food

One major contributor to modern micronutrient insufficiency is the declining nutrient density of food. Industrial agricultural practices, soil mineral depletion, monocropping, and reliance on chemical fertilizers have progressively reduced the mineral content of fruits, vegetables, and grains. As a result, even diets rich in whole foods may fail to provide adequate magnesium, zinc, selenium, potassium, and trace minerals.

Increased Physiological Demand and Lifestyle Stressors

Ultra-processed foods provide excess calories with minimal micronutrient content while increasing metabolic demand for vitamins and minerals involved in carbohydrate metabolism, oxidative stress regulation, and neurotransmitter synthesis. Chronic psychological stress further accelerates micronutrient depletion by increasing renal magnesium loss, altering intracellular potassium distribution, and suppressing digestive enzyme secretion.

Excessive sweating due to physical training or heat exposure leads to additional losses of sodium, potassium, and magnesium. Alcohol, caffeine, and diuretic use further exacerbate electrolyte depletion.

Impaired Gastrointestinal Absorption

Gastrointestinal dysfunction plays a central role in micronutrient deficiency. Conditions such as hypochlorhydria, chronic gastritis, *Helicobacter pylori* infection, inflammatory bowel disorders, and age-related reductions in gastric acid impair absorption of vitamin B12, iron, calcium, magnesium, and zinc. In such cases, adequate dietary intake does not ensure cellular sufficiency.

Pharmaceutical-Induced Depletion

Common medications significantly influence micronutrient status. Proton pump inhibitors reduce absorption of magnesium and vitamin B12, metformin is associated with progressive B12 depletion, statins reduce coenzyme Q10 and vitamin D availability, and diuretics increase renal loss of electrolytes. These deficiencies often develop gradually and remain undetected until neurological, muscular, or cardiovascular symptoms appear.

Genetic Constraints on Micronutrient Utilization: MTHFR Polymorphisms

Beyond intake and absorption, genetic variability plays a critical role in micronutrient metabolism. The methylenetetrahydrofolate reductase (MTHFR) enzyme catalyzes the conversion of dietary folate into 5-methyltetrahydrofolate (5-MTHF), the biologically active form required for methylation reactions.

Common polymorphisms, particularly C677T and A1298C, reduce enzymatic activity by approximately 30–70%, depending on genotype. Individuals with these variants frequently exhibit functional deficiencies of folate, vitamin B12, vitamin B6, riboflavin, and magnesium, even when serum levels appear normal.

Impaired methylation disrupts homocysteine clearance, DNA synthesis and repair, neurotransmitter production, detoxification pathways, and hormone metabolism. Clinically, this manifests as elevated homocysteine, increased cardiovascular risk, anxiety, depression, poor stress tolerance, and reduced response to conventional supplementation. Synthetic folic acid requires MTHFR-dependent conversion and may accumulate unmetabolized, whereas methylated folate bypasses this metabolic bottleneck.

Materials and Methods

Study Design

A 12-week randomized, double-blind, parallel-group experimental study was conducted at Queen's University Canada at an academic clinical research facility.

Sample

A total of 120 healthy adults aged 25–55 years were enrolled and randomized into two groups: ACTIVIT (Doctors Prescribed) (n = 60) and control multivitamin (n = 60).

Inclusion and Exclusion Criteria

As previously described.

Intervention

The ACTIVIT (Doctors Prescribed) group received one capsule daily containing methylated B-complex vitamins, vitamin D3, and amino acid-chelated minerals. The control group received a standard multivitamin containing synthetic vitamin forms and inorganic minerals.

Outcome Measures

Primary and secondary outcomes were assessed as originally reported.

Statistical Analysis

Data were analyzed using SPSS version 26. A p-value < 0.05 was considered statistically significant.

Results

Table 1. Changes in Vitamin Biomarkers After 12 Weeks

Serum B12: 612 ± 88 pg/mL (ACTIVIT)(Doctors Prescribed) vs 429 ± 76 pg/mL (control), p < 0.001
RBC Folate: 548 ± 64 ng/mL vs 396 ± 59 ng/mL, p < 0.001
Homocysteine: 8.4 ± 1.6 µmol/L vs 11.2 ± 2.1 µmol/L, p < 0.001

Table 2. Mineral Bioavailability

Magnesium: 2.12 ± 0.21 mg/dL vs 1.78 ± 0.19 mg/dL, p = 0.002
Zinc: 102 ± 11 µg/dL vs 86 ± 10 µg/dL, p = 0.004

Table 3. Functional Outcome

Fatigue Severity Score reduction: -31.6% vs -12.4%, p < 0.001

Discussion

The results demonstrate that ACTIVIT (Doctors Prescribed) significantly improves micronutrient bioavailability and functional outcomes compared with a conventional multivitamin. The magnitude of improvement in serum B12, RBC folate, and homocysteine reduction supports enhanced methylation efficiency, particularly relevant in populations with impaired folate metabolism.⁷⁻¹⁰ Improved magnesium and zinc status reflects the superior absorption of amino acid–chelated minerals, which utilize distinct intestinal transport pathways and exhibit reduced competitive inhibition.¹¹⁻¹² The observed reduction in fatigue severity is consistent with improved mitochondrial energy production and neuromuscular stability.¹³⁻¹⁴

Conclusion

Micronutrient deficiencies are systemic, multifactorial, and globally prevalent, driven by modern food systems, lifestyle stressors, pharmaceutical exposure, gastrointestinal dysfunction, and genetic variability such as MTHFR polymorphisms. ACTIVIT (Doctors Prescribed) addresses these challenges through the use of bioactive, methylated vitamins and chelated minerals, resulting in superior biochemical correction and functional improvement within a short intervention period. These findings support the role of clinically optimized micronutrient formulations in addressing hidden nutritional insufficiency.

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Informed Consent

Verbal informed consent was obtained from all participants prior to enrollment.

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