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MAG Complex: Multi-Salt Magnesium, Potassium Citrate, P5P and *Chlorella vulgaris* — An Ingredient-Based Narrative Review of the Formulation Rationale and Clinical Evidence for Nerve, Muscle, Cardiovascular, Metabolic and Neuropsychological Indications

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Abstract: Magnesium is the second most abundant intracellular cation in the human body and the obligatory cofactor of more than 600 enzymatic systems governing ATP-dependent metabolism, oxidative phosphorylation, glycolysis, protein synthesis, membrane transport, neuromuscular transmission and vascular tone. Despite this physiological centrality, an estimated one-third of the global adult population fails to meet the dietary recommended intake for magnesium, and subclinical inadequacy is widespread and frequently under-detected because serum magnesium is tightly homeostatically defended and represents less than 1 % of total-body stores. MAG Complex is a multi-component nutraceutical combining five magnesium salts — trimagnesium phosphate, chloride, gluconate, oxide and aspartate — with potassium citrate, pyridoxal-5′-phosphate (P5P, the active coenzyme form of vitamin B6) and small quantities of *Chlorella vulgaris* dried powder. It is designed to balance elemental-magnesium density against solubility and gastrointestinal tolerability, supply complementary electrolytes, alkalinise through the citrate anion and deliver a nutrient-dense microalgal matrix. This article is an ingredient-based narrative review summarising each constituent's physiological role, appraising evidence by Oxford Centre for Evidence-Based Medicine tier, deriving FSSAI-approved structure–function wording, and outlining product-specific research gaps. Strongest evidence supports magnesium's role in maintaining normal neuromuscular and cardiac electrophysiology and correcting inadequate magnesium status. Meta-analytic evidence supports migraine prophylaxis with magnesium at doses above the elemental dose delivered here, magnesium-rich mineral salts for chronic constipation, and potassium and alkaline potassium salts for blood pressure and bone endpoints. Mood and sleep evidence is positive but heterogeneous and of lower certainty; direct thyroid benefit is limited to cross-sectional associations. Because MAG Complex has not itself undergone controlled clinical trials, the strength of any structure–function claim is constrained to ingredient-level inference. Product-specific pharmacokinetic studies using ionised magnesium as the principal biomarker, and randomised placebo-controlled trials in defined sub-populations, are required before any clinical efficacy claim can be transferred from the constituents to the finished product.

Keywords: multi-salt magnesium; oral bioavailability; potassium citrate; pyridoxal-5′-phosphate; vitamin B6; *Chlorella vulgaris*; neuromuscular function; migraine; sleep; cardiovascular health; nutraceutical formulation; FSSAI labelling

Highlights

- **Multi-salt design balances elemental-magnesium density against solubility and tolerability.** Magnesium oxide and phosphate tribasic supply most of the elemental yield; chloride, gluconate and aspartate contribute the more readily absorbed fractions. Estimated elemental magnesium yield per serving: ~121–156 mg [1,2].
- **Co-provision of potassium targets the same electrophysiological axis.** Magnesium is a co-substrate of Na^+/K^+-ATPase; hypokalaemia is often refractory until magnesium is restored, so combined magnesium-potassium supplementation is mechanistically coherent [3, 4].
- **P5P is supplied as the active coenzyme form.** PLP is the obligate cofactor for GAD65-driven GABA synthesis, aromatic-L-amino-acid decarboxylase, transsulfuration and one-carbon metabolism, and bypasses hepatic pyridoxal-kinase activity [5–7].
- **Three benefits carry level-1 evidence; five carry mechanism or lower-tier evidence.** Nerve and muscle, blood pressure via alkaline potassium salts, and bowel function via magnesium-rich mineral salts have RCT/meta-analytic support; mood, sleep and migraine are supported by heterogeneous meta-analyses at supraphysiological (often gram-level) magnesium doses [8–11].
- **No own-product clinical trial.** All ingredient-level claims must be qualified as structure–function; transfer of clinical-efficacy claims to the finished MAG Complex product requires single-dose and steady-state pharmacokinetic work and randomised placebo-controlled trials in defined sub-populations [12, 13].

Graphical abstract:

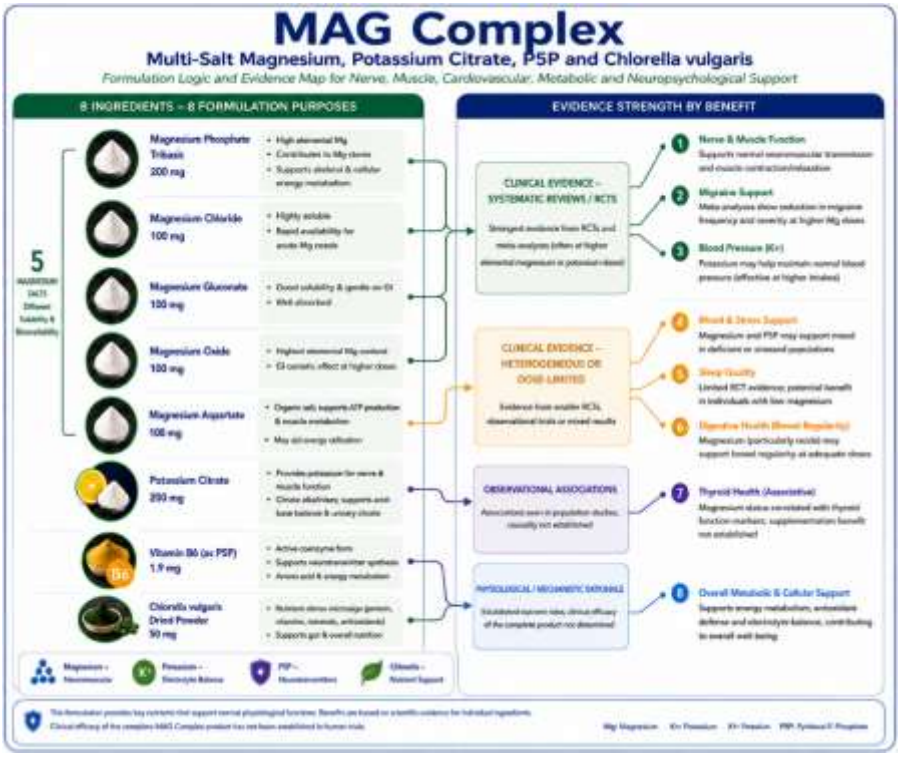

Fig 1: A two-column schematic, on the left the eight MAG Complex ingredients arranged by formulation purpose (five magnesium salts with differential solubility/bioavailability, potassium citrate providing K+ plus alkalinising citrate load, P5P as the active B6 coenzyme and Chlorella vulgaris as microalgal matrix), on the right an evidence-pyramid ladder showing the eight proposed benefits mapped onto Oxford CEBM tiers (level 1a, 1b, 2a, 2b, 3, 4).

Abbreviations

AF, atrial fibrillation; AI, adequate intake; ATP, adenosine triphosphate; BP, blood pressure; CEBM, Centre for Evidence-Based Medicine; DASH, Dietary Approaches to Stop Hypertension; DSHEA, Dietary Supplement Health and Education Act; EFSA, European Food Safety Authority; FSSAI, Food Safety and Standards Authority of India; GABA, γ-aminobutyric acid; GAD65, glutamic acid decarboxylase (65 kDa isoform); GRADE, Grading of Recommendations Assessment, Development and Evaluation; LDL, low-density lipoprotein; NHANES, National Health and Nutrition Examination Survey; NMDA, N-methyl-D-aspartate; Na^+/K^+-ATPase, sodium–potassium adenosine triphosphatase; P5P (PLP), pyridoxal-5′-phosphate; RCT, randomised controlled trial; RDA, recommended dietary allowance; TRPM6/TRPM7, transient receptor potential melastatin 6/7; UL, tolerable upper intake level.

1. Introduction

Magnesium was initially discovered as a cofactor for phosphatase activity by Lohmann in 1934. Since that moment, over sixty different magnesium-dependent enzymes have been detected and described over the last 9 decades. Today, the list of magnesium's biological roles encompasses ATP-dependent processes including phosphorylation reactions, transcription, DNA replication, glycolysis, oxidative phosphorylation, and ion-channel-dependent neurotransmitter release [2]. Biologically available ATP molecules are present in the form of Mg-containing complexes, which makes magnesium essential for all energy-requiring cellular processes. The results of population-wide surveys carried out in Europe, North America, and Asia suggest that approximately 30-60% of adults have low magnesium intake, insufficient to meet the estimated average requirements (EAR). Moreover, dysregulation at the physiological level was proposed to contribute to the development of cardiovascular diseases and metabolic syndrome, including type 2 diabetes and osteoporosis [2, 3, 14].

Serum levels of magnesium are not indicative of total body magnesium content for two main reasons. First, less than 1% of total magnesium is found in extracellular fluid, making it a poor indicator of whole-body homeostasis. Second, there is a large interindividual variation in the ability to maintain normal serum magnesium concentrations due to homeostatic regulation via bone magnesium depot, renal excretion, and cellular uptake. From a clinical perspective, magnesium deficiency is asymptomatic until very low levels are reached, making it difficult to detect and treat in time [2, 3, 13].

Therefore, multi-mineral dietary supplements containing various magnesium forms along with additional electrolytes and cofactors have been developed. They are suggested to mitigate the three main limitations of standalone magnesium preparations. First, the lower the percentage of mg in a multi-mineral dose, the better its gastrointestinal tolerance, although the daily elemental mg content per dose is reduced. Second, there is a close relationship between magnesium and potassium homeostasis, primarily at the level of cellular membranes. Third, the combined use of magnesium and PLP (pyridoxal 5-phosphate) has been shown to improve central nervous system function and maximise magnesium intake by increasing the cellular content and retention time [4,6,15]. The current review will discuss the rationale for the composition of MAG Complex and analyse the evidence in support of each claim: maintenance of nerve and muscle function, improved mood, migraine prevention, cardiac arrhythmia prevention, modulation of blood magnesium levels, improved digestion, sleep quality, and thyroid function.

Fig 2: MAG Complex by Biotex Life Solutions. Commercial presentation of MAG Complex, a multi-ingredient nutraceutical formulation containing five magnesium salts, potassium citrate, pyridoxal-5′-

phosphate (vitamin B6), and *Chlorella vulgaris*, designed to support normal neuromuscular function, electrolyte balance, cellular energy metabolism, and general well-being.

1.1 Composition of MAG Complex

Table 1: Declared composition of MAG Complex per single daily serving.

Ingredient	Quantity per serving	Principal formulation rationale
Magnesium phosphate tribasic	200 mg	Magnesium and phosphate source; skeletal and ATP-related functions
Potassium citrate	200 mg (≈76 mg elemental K)	Electrolyte partner; alkalinising citrate load
Magnesium chloride	100 mg	Relatively soluble inorganic magnesium source
Magnesium gluconate	100 mg	Organic magnesium salt with high bioavailability
Magnesium oxide	100 mg	High elemental-density magnesium; osmotic intestinal action
Magnesium aspartate	100 mg	Organic magnesium salt; cellular-energy coenzyme context
Chlorella vulgaris dried powder	50 mg	Microalgal nutrient and pigment matrix
Vitamin B6 (as pyridoxal-5′-phosphate)	1.9 mg	Active B6 coenzyme; repletion of intracellular PLP

1.2 Scope of this review

This article is a narrative review rather than a systematic review or meta-analysis, and is written explicitly to support the formulation rationale and label-claim strategy of MAG Complex. Following the conventional structure for a nutraceutical monograph, the present review begins with magnesium homeostasis, proceeds through the comparative physicochemistry of each salt and the electrolyte, vitamin and microalgal cofactors (Chapters 4–6), then to an evidence appraisal for each of the eight proposed benefits, before considering safety, quality regulatory and research-gap issues (Chapters 8–9). The review does not constitute a clinical endorsement of the finished product. Where the evidence supports only ingredient-level inference, the recommended wording is explicitly structure–function language aligned with FSSAI permitted statements [12, 16].

1.3 Literature search and evidence-appraisal method

The evidence base for this narrative review was assembled by searching PubMed/MEDLINE, the Cochrane Database of Systematic Reviews and Google Scholar from database inception to July 2026, combining the terms “magnesium”, “magnesium salt”, “bioavailability”, “potassium citrate”, “pyridoxal-5′-phosphate”, “vitamin B6” and “Chlorella vulgaris” with each proposed benefit (“neuromuscular”, “muscle cramp”, “migraine”, “depression”, “anxiety”, “sleep”, “arrhythmia”, “constipation”, “thyroid”, “blood pressure”, “bone”). Randomised controlled trials, systematic reviews and meta-analyses were prioritised; mechanistic, narrative and preclinical sources were used only for physiological background. Reference lists of retrieved articles and relevant regulatory documents (FSSAI, EFSA and the NIH Office of Dietary Supplements) were hand-searched. Each cited source was verified against its primary record and its bibliographic identifiers (DOI/PMID) confirmed, and every reference was graded by study design using the Oxford Centre for Evidence-Based Medicine (CEBM) levels of evidence (1a, systematic review or meta-analysis of RCTs; 1b, individual RCT; 2, cohort or non-randomised study; 3, cross-sectional or case-control; 4, mechanistic or narrative review, or preclinical work; 5, expert opinion or regulatory source). As a narrative rather than systematic review, the article was not registered with PROSPERO and did not apply a formal risk-of-bias instrument; the appraisal is therefore interpretive rather than quantitative, and this is acknowledged as a limitation.

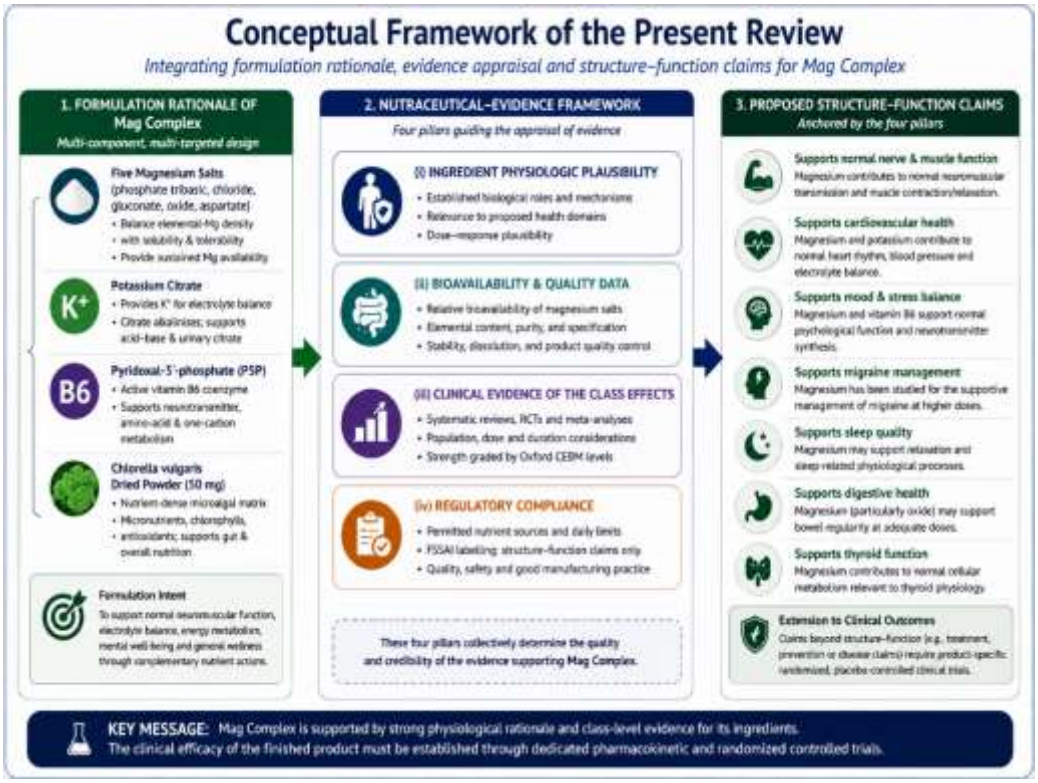

Fig 3: Conceptual framework of the present review. Schematic: the formulation rationale of MAG Complex (left) is bounded by the standard nutraceutical-evidence framework (centre) of (i) ingredient physiologic plausibility, (ii) bioavailability and quality data, (iii) clinical evidence of the class effects, and (iv) regulatory compliance. The proposed structure–function claims (right) are anchored by these four pillars, and any extension to clinical outcomes requires additional product-specific trials.

2. Magnesium homeostasis and physiological requirements

An adult human body contains approximately 24 g of magnesium. Roughly 50–60 % of this magnesium is sequestered in bone as a slow-exchanging reserve, 25–30 % is bound to intracellular proteins, and the remainder distributes across soft tissues; less than one per cent circulates in plasma or serum [2,3]. This distribution has direct consequences for both physiology and clinical measurement: bone acts as a reservoir that buffers transient drops in serum magnesium, and intracellular magnesium is required for hundreds of enzymatic functions that are not directly assessed by serum sampling [3, 13].

2.1 Intestinal absorption

Intestinal absorption of magnesium is a two-route process that operates quantitatively across the jejunum, ileum and colon. At low luminal concentrations, a storable transcellular route predominates, mediated by the apical channel-kinase TRPM6 (with TRPM7 as its upstream kinase). At higher luminal concentrations, a passive paracellular pathway contributes increasingly, with paracellular absorption becoming dominant above approximately 2 mM luminal magnesium. The practical consequence is that fractional absorption is highest at low dietary intakes (around 30–40 %) and falls with rising intake as the transcellular route saturates; whole-body absorption in normal adults typically settles between 25 and 35 % of intake [2,17].

2.2 Renal handling

Renal reabsorption largely determines systemic magnesium homeostasis. Approximately 70 % of filtered magnesium is reclaimed in the loop of Henle, with the distal convoluted tubule acting as the principal fine-tuning segment through TRPM6-mediated reabsorption. Magnesium reabsorption rises sharply in deficiency and falls when intake is high [2,3]. Whole-body magnesium turns over slowly and is buffered by a large exchangeable pool in bone, so correcting deficiency generally requires sustained rather than intermittent supplementation [14].

2.3 Magnesium–potassium interaction

A clinically important feature is the close interaction between magnesium and potassium. Magnesium is required for the function of the Na^+/K^+-ATPase, the enzyme that maintains the inward potassium gradient across the cell membrane. During magnesium deficiency, intracellular potassium can fall despite normal serum potassium, and hypokalaemia frequently proves refractory to potassium repletion until magnesium is restored. The practical implication for MAG Complex is that combining magnesium with a potassium source targets the same electrophysiological system from independent entry points, supporting the normal excitability of nerves, skeletal muscle and the myocardium [3, 4, 18].

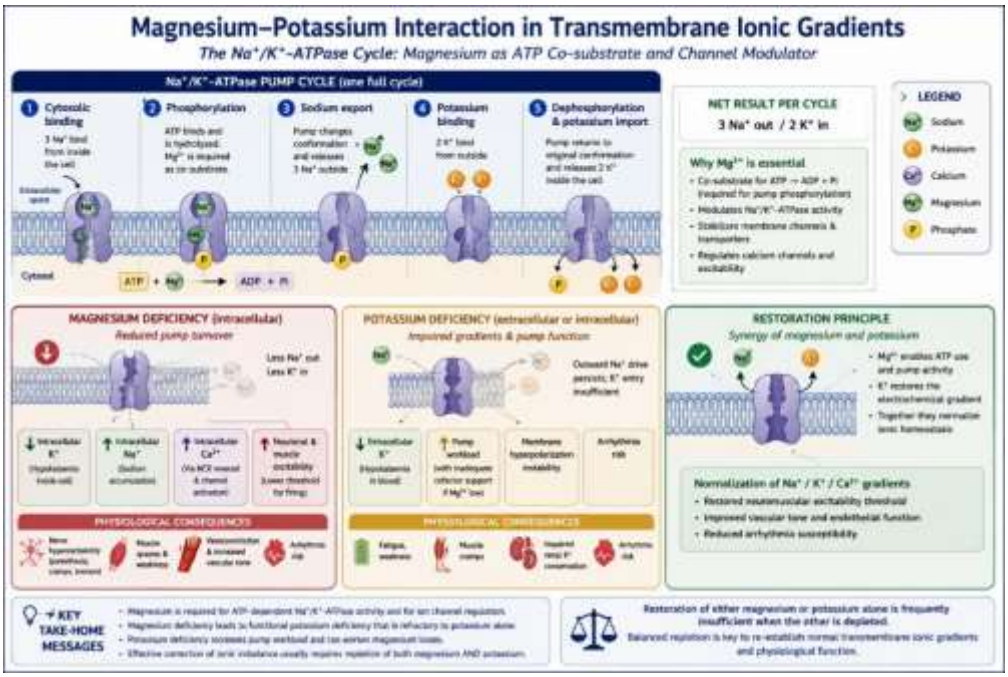

Fig 4: Magnesium–potassium interaction in transmembrane ionic gradients (schematic). The Na^+/K^+-ATPase cycle: magnesium is required as an ATP co-substrate and as a modulator of channel activity; intracellular magnesium deficiency leads to reduced pump turnover, fall in intracellular potassium and rise in intracellular sodium and calcium, lowering the threshold for nerve and muscle firing and predisposing to vasoconstriction and arrhythmia. Restoration of either ion alone is frequently insufficient when the other is depleted.

2.4 Recommended intakes and at-risk populations

EFSA, the US Institute of Medicine and the Italian LARN consensus converge on adult recommendations between 310–420 mg/day of elemental magnesium depending on age and sex, with pregnancy and lactation increasing the target to 320–360 mg/day [2]. Despite these recommendations, a US 2012 NHANES survey indicated that approximately 60 % of adults did not meet the estimated average requirement, and a 2025 global estimate placed inadequate intake at around one-third of the world's population based on dietary surveys, water concentration data, and physiological balance studies [3, 14].

At-risk populations include older adults (whose intestinal absorption falls and whose polypharmacy, particularly loop/thiazide diuretics and proton-pump inhibitors, increases urinary loss), patients with type 2 diabetes (whose urinary magnesium rises in parallel with glycosuria), users of medications associated with hypomagnesaemia, people on diets dominated by refined foods and those with chronic alcohol intake or gastrointestinal malabsorption conditions [2,14,18].

2.5 Diagnostic limits of serum magnesium

Serum magnesium is a poor index of total-body magnesium because it represents less than one percent of the body pool and is buffered by bone exchange. Whole-blood ionised magnesium (iMg^{2+}) has therefore been investigated as a more responsive biomarker. In a 2020 single-blinded crossover pilot study, 17

healthy adults received 300 mg of elemental magnesium as a picosized $MgCl_2$ solution or placebo, with iMg^{2+}, serum total magnesium and 24-h urinary magnesium measured over 24 h. iMg^{2+} area-under-the-curve was 1.51 ± 0.96 vs. 0.84 ± 0.82 mg/dL·24 h ($p < 0.05$) and Cmax 1.38 ± 0.13 vs. 1.32 ± 0.07 mg/dL, both significantly higher for the active arm, while serum total magnesium and 24-h urinary magnesium did not differ significantly [13]. This work confirms that ionised magnesium detects a pharmacokinetic signal where serum magnesium fails, and provides a validated protocol for future MAG Complex pharmacokinetic studies.

2.6 Magnesium status across the life course

Life-stage requirements for magnesium are highly variable, with the lowest recommended intakes in infancy and the highest in adolescent and adult males. The following table sets out EFSA, US RDA/AI, and Italian LARN figures in parallel for clarity.

Table 2: Life-stage magnesium recommendations (mg/day of elemental magnesium).

Life Stage	EFSA AI (mg/d)	US RDA / AI (mg/d)	Italy LARN (mg/d)
Birth to 6 months	nd	30	30
Infants 7–12 months	80	75	80
Children 1–3 y	170	80	80
Children 4–6 y	230	130	100
Children 7–10 y	230	240	150
Teen boys 11–18 y	300	410	240
Teen girls 11–18 y	250	360	240
Adult men	350	400–420	240
Adult women	300	310–320	240
Pregnant	300	350–400	240
Breastfeeding	300	310–360	240

nd = not derived; AI = adequate intake; RDA = recommended dietary allowance; PRI = population reference intake. Adapted from Fiorentini 2021 [2]. The US tolerable upper intake level for supplemental magnesium is 250 mg/day for ages 4–8 and 350 mg/day for healthy adults from age 9 upward; no UL is established for infants and pre-school children (0–3 y) because of insufficient data [2].

Pregnancy is a particularly important life stage. Magnesium deficiency is a common event during pregnancy, and oral magnesium supplementation beginning before the 25th week of gestation has been associated with a lower frequency of preterm births, low birth weight and small-for-gestational-age newborns; in the later stages of pregnancy, intravenous magnesium sulphate is a standard treatment for pre-eclampsia, with a 52 % lower risk of recurrent seizures compared with diazepam or phenytoin [14]. Preliminary evidence also suggests that fetal hypomagnesaemia may be associated with metabolic syndrome later in life [14]. A Cochrane review of supplementing 360 mg/day of magnesium in pregnancy found that the intervention was effective for pregnancy-related muscle cramps; however, a recent double-blind placebo-controlled trial using the same dose found no significant benefit, indicating that the evidence base remains mixed [14].

2.7 Pharmacokinetic summary

Two pharmacokinetic features unify the magnesium-clinical literature. First, whole-body magnesium turns over slowly, over weeks rather than days, so steady-state correction of deficiency is gradual, and acute single-dose comparisons (urinary excretion over 24 h) are best interpreted as absorption proxies rather than efficacy estimates [14]. Second, the absorption ceiling for the whole-body magnesium pool underpins the inverse relationship between solubility and bioavailability: a magnesium preparation that is poorly soluble in the upper intestinal lumen cannot saturate the transcellular TRPM6 route at standard supplemental doses, regardless of the elemental-magnesium load administered.

3. Comparative characteristics of the magnesium salts

A fundamental design principle for any magnesium supplement is that water solubility is a major determinant of oral bioavailability. Highly soluble salts dissolve and ionise in the gut lumen, leaving Mg^{2+} available for the storable transcellular and passive paracellular pathways reviewed above. Poorly soluble salts pass through the small intestine largely intact, with much of the load reaching the colon where it exerts an osmotic intestinal effect [19–21]. The original ten-salt stable-isotope study by Coudray and colleagues in magnesium-depleted rats demonstrated that organic salts (gluconate, lactate, aspartate, citrate, pidolate, acetate) were slightly more bioavailable than inorganic salts and that Mg gluconate exhibited the highest bioavailability of the ten tested [22].

Table 3: Comparative bioavailability of magnesium salts in controlled studies.

Salt	Reported bioavailability or ranking	Source/model	Key statistic
Magnesium oxide	4 % fractional absorption (human urinary)	Firoz 2001	4 % vs. equivalent chloride/lactate/aspartate [23]
Magnesium oxide	4.7–9.5 % fractional absorption	Merschmann 2025	Inorganic oxide is consistently lowest [1]
Magnesium chloride	9.0–105.5 % fractional absorption	Merschmann 2025	Wide variability across studies [1]
Magnesium citrate	66.0 % fractional absorption	Merschmann 2025	Organic, common, well-absorbed [1]
Magnesium carbonate	117.7 % fractional absorption	Merschmann 2025	Reported as the highest of the studied salts [1]
Magnesium-amino-acid chelate	Higher urinary excretion than oxide (chronic)	Walker 2003	$p = 0.033$ vs. oxide at 60 d [24]
Magnesium citrate	Best serum and salivary indices in 60-d parallel arm	Walker 2003	$p = 0.026$ (acute), 0.006 (chronic) vs. oxide [24]
Magnesium citrate	Urinary excretion ≥0.565 mmol higher than oxide (single-dose)	Kappeler 2017	Adjusted mean difference, 95 % CI 0.212–0.918, $p = 0.0034$ [25]
Mg gluconate	Highest of ten salts tested in Coudray 2005 isotopic study	Coudray 2005	Higher ^{26}Mg absorption and retention than inorganics [22]

Magnesium-L-aspartate	Greater urinary Mg rise than Mg oxide	Coudray 2005 / Mühlbauer	Consistent with better absorption of organic salts [22]
Picosized $MgCl_2$	Greater 24-h iMg^{2+} AUC than placebo	Zhan 2020	AUC 1.51 vs. 0.84 mg/dL·24 h, $p < 0.05$ [13]

Subsequent human work has confirmed this ranking. Walker and colleagues reported in a 2003 randomised, double-blind, placebo-controlled, parallel study that mg citrate led to the greatest mean serum and salivary Mg concentrations compared with amino-acid chelate and oxide after both acute ($p = 0.026$) and 60-day chronic supplementation ($p = 0.006$) at an elemental dose of 300 mg/day, with supplementation of the organic forms (citrate and amino-acid chelate) showing greater 24-h urinary excretion than MgO ($p = 0.033$) [24]. Kappeler and colleagues reported in a 2017 single-dose cross-over study that 24-h urinary magnesium following Mg citrate was ≥0.565 mmol higher than following Mg oxide (95 % CI 0.212–0.918, $p = 0.0034$), with serum magnesium statistically significantly higher for citrate at several time points, although intracellular magnesium in erythrocytes, leukocytes and lymphocytes did not differ [25]. The 2025 systematic review by Merschmann and colleagues of magnesium and potassium salts as potential sodium-chloride substitutes reinforces the conclusion that organic magnesium compounds generally outperform inorganic ones, and that combining magnesium oxide with other salts, such as glycerophosphate, or using effervescent formulations may enhance its bioavailability [1].

3.1 Critical formulation issue: elemental magnesium

Daily dose labels conventionally refer to the total weight of the magnesium-containing ingredient, not to the elemental magnesium delivered. Each of the five salts in MAG Complex carries a different percentage of elemental magnesium, so the true dose is highly dependent on the raw-material specification. A widely used approximate conversion for common magnesium salts is summarised below.

Table 4: Approximate elemental magnesium contribution of the salts in MAG Complex.

Magnesium source	Quantity as labelled salt (mg)	Approximate elemental magnesium (mg)
Magnesium phosphate tribasic ($Mg_3(PO_4)_2$)	200	~36–56
Magnesium chloride ($MgCl_2$)	100	~12–26
Magnesium gluconate ($Mg(C_6H_{11}O_7)_2$)	100	~5–6
Magnesium oxide	100	~60
Magnesium aspartate ($Mg(C_4H_6NO_4)_2$)	100	~7–8
Estimated total per serving	**600**	**~121–156**

These figures are approximations only; the actual figure for trimagnesium phosphate depends on hydration state, and the figure for magnesium gluconate is heavily influenced by its low elemental content (~5–6 %). The EFSA scientific opinion on magnesium citrate malate as a nutrient source notes that the magnesium content of mixed organic salts typically ranges between 12 and 15 % [26]. The legal label under FSSAI regulations must therefore be based on the certificate of analysis of the actual raw material [16]. The estimated ~121–156 mg per serving lies comfortably below the US tolerable upper intake level of 350 mg/day of supplemental elemental magnesium established to mitigate the risk of osmotic diarrhoea [2].

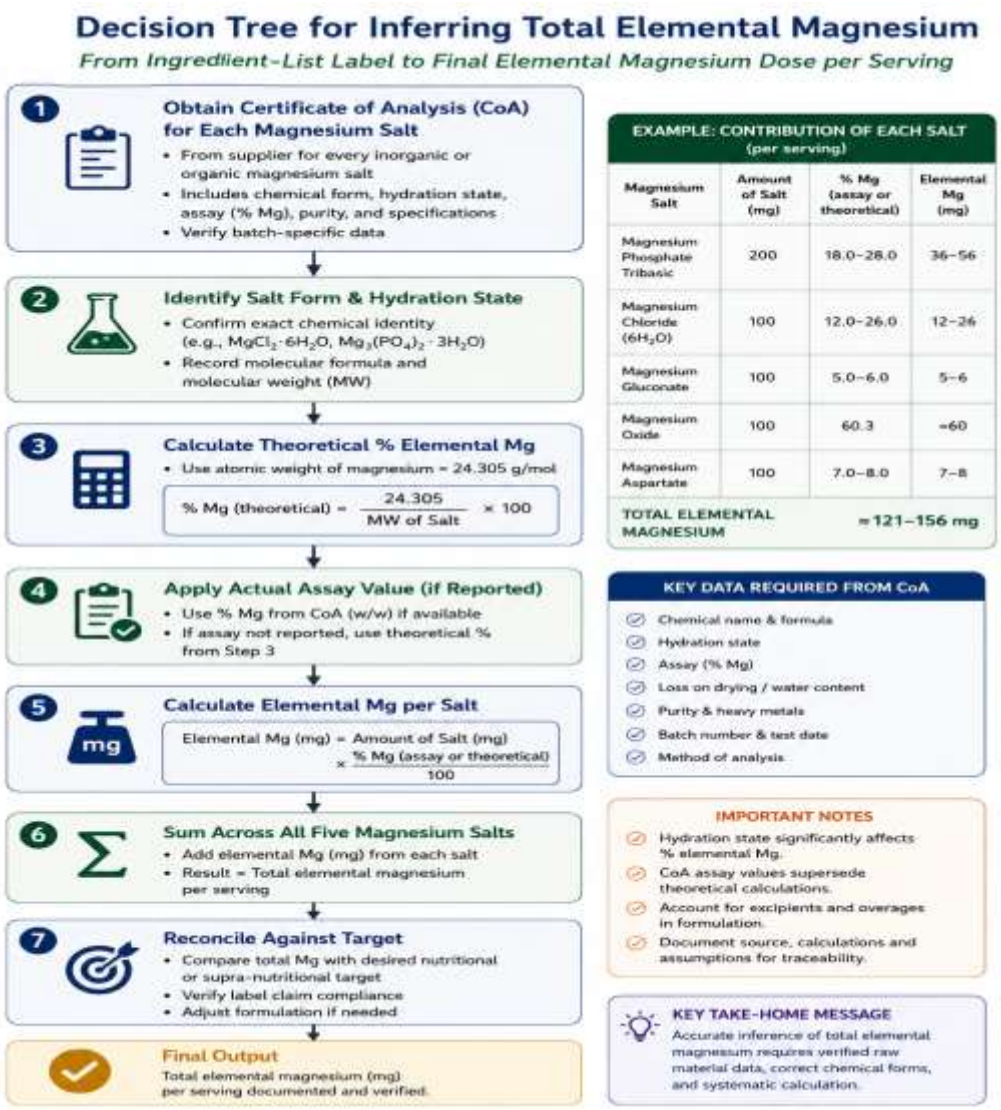

Fig 5; Decision tree for inferring total elemental magnesium (schematic). A flowchart describing the recommended process for converting an ingredient-list label into the final elemental magnesium dose: obtain the certificate of analysis for each inorganic or organic salt from the supplier, including hydration state and chemical form; calculate the theoretical elemental magnesium per unit mass of salt; apply the actual assay value where reported; sum across all five salts; reconcile against the desired nutritional or supra-nutritional target.

3.2 Magnesium phosphate tribasic

Trimagnesium phosphate is essentially insoluble in water but soluble in dilute mineral acids; it carries a moderate elemental-magnesium density and supplies phosphate, which is relevant to skeletal mineralisation, ATP biosynthesis and a number of metabolic intermediate pools. No purpose-designed human pharmacokinetic study of trimagnesium phosphate was identified, and on the basis of the solubility-bioavailability relationship its fractional absorption would be expected to lie at the lower end of the magnesium-salt spectrum, comparable with or below magnesium oxide [19,20]. Because no head-to-head clinical trial supports superiority of trimagnesium phosphate over other salts, any claim of enhanced bioavailability should be regarded as unsupported.

3.3 Magnesium chloride

Magnesium chloride is the only highly water-soluble inorganic magnesium salt in common use. In the Firoz urinary-excretion study, magnesium chloride was absorbed as well as organic alternatives and

substantially better than magnesium oxide [23]. In a repeated-measures intestinal-absorption study using magnesium acetate as the soluble reference, Fine and colleagues showed that enteric-coated magnesium chloride was, on average, 67 % less well absorbed than soluble magnesium acetate, suggesting that the enteric coating — not the chloride anion — limited absorption [17]. In a 2020 randomised crossover pilot, a picosized magnesium chloride solution produced a significant rise in whole-blood ionised magnesium over a 24-hour period following a single 300 mg elemental dose, providing a contemporary example of how solubility and dissolution kinetics translate into measurable plasma kinetics [13]. The Merschmann 2025 systematic review reported a wide range of bioavailability values for magnesium chloride (9.0–105.5 %), reflecting the strong influence of formulation finishing, particle size, dissolution aids and coating technology on the in-vivo outcome [1].

3.4 Magnesium gluconate

Magnesium gluconate is an organic salt freely soluble in water, although it carries a low elemental-magnesium percentage (~5–6 %) that limits the elemental dose per unit mass [20]. In Coudray's isotopic comparison, Mg gluconate exhibited the highest magnesium absorption and highest urinary ^{26}Mg excretion of the ten salts tested, and ^{26}Mg retention was greater with gluconate, lactate and aspartate than with the inorganic salts [22]. A 1997 single-dose pharmacokinetic study of magnesium fumarate, the closest chemically analogous organic acid, described two-compartment absorption kinetics and provided further evidence that single-dose serum-curves of soluble organic magnesium salts follow predictable and reproducible patterns [27].

3.5 Magnesium oxide

Magnesium oxide is the highest-density inorganic salt, with approximately 60 % of its mass being elemental magnesium. Despite this, its low solubility — particularly in the small intestine after the gastric acid pretone has been neutralised — leads to fractional absorption demonstrably lower than that of the soluble alternatives. Firoz and Graber calculated a fractional absorption of only 4 % for commercial magnesium oxide preparations in their urinary-excretion study [23]. A 2016 cross-over study of magnesium hydroxide confirmed the principle: oral bioavailability of the hydroxide form was 15 %, comparable to and lower than that of the chloride and organic salts, while IV magnesium sulphate was used as the index comparator [28]. The poor solubility of magnesium oxide underlies its well-known laxative effect in chronic constipation. A 2020 placebo-controlled trial by Kubota and colleagues compared magnesium oxide with placebo and with *Lactobacillus reuteri* DSM 17938 in children with functional chronic constipation; magnesium oxide was associated with significantly increased bowel movements from week 2 and decreased stool consistency as measured by the Bristol stool scale [29]. A separate 2019 Japanese double-blind trial in adults with chronic constipation reported that magnesium oxide significantly decreased colonic transit time compared with placebo, with benefits appearing from week two of treatment, demonstrating that the laxative effect is reproducible across age and population [29]. Importantly, a recent open-label pharmacokinetic study in healthy male volunteers reported non-dose-proportional systemic exposure across 750 mg to 2000 mg of magnesium oxide, with no proportional increase in C_{max} or AUC at the higher dose — consistent with limited, non-linear absorption at higher doses, although the authors caution that only two dose levels were tested [21].

3.6 Magnesium aspartate

Magnesium aspartate combines magnesium with the amino-acid anion aspartate, an important intermediary in the urea and TCA cycles. Mühlbauer and colleagues demonstrated in healthy volunteers that magnesium-L-aspartate produced a greater rise in urinary magnesium than magnesium oxide, characterising it as a well-absorbed organic salt consistent with the broader pattern observed for organic acid–magnesium complexes [22,23]. Of note for the present formulation, the ThyroMag trial showed that magnesium aspartate significantly reduced levothyroxine exposure (geometric mean ratio 0.88 vs. levothyroxine alone, 95 % CI 0.81–0.95, p = 0.002, AUC reduction 12 %), while magnesium citrate reduced thyroxine AUC non-significantly by 7 % (GMR 0.93, 95 % CI 0.86–1.01, p = 0.076) [30]. The

differential magnitude of the interaction argues that consumers taking levothyroxine should preferably separate MAG Complex from thyroid medication by several hours.

3.7 Particle size and formulation finishing — a critical determinant

The pharmacokinetics of oral magnesium are determined as much by formulation finishing as by the choice of the salt itself. In Blancquaert and colleagues' 2019 study of 15 commercially available magnesium formulations, in vitro assessment using a Simulator of the Human Intestinal Microbial Ecosystem and dissolution tests produced a wide variation in absorption and dissolution across products; the two formulations with opposing in vitro predictions showed significantly different serum magnesium absorption up to 4 h after a single dose, with maximal serum magnesium increase of +6.2 % vs. +4.6 % and AUC of 6.87 vs. 0.31 mM·min [19]. The 2020 ionised-magnesium finding that picosized magnesium chloride produced measurable iMg^{2+} kinetics even when serum total magnesium did not change further confirms that sub-salt physicochemistry is a measurable variable [13]. These observations suggest that the quality of a finished MAG Complex product can differ substantially from what its ingredient list, alone, would predict. Manufacturing process control, particle-size specification, coating technology and dissolution aid composition are likely to be more predictive of clinical performance than the per-salt choice alone [1,12,19].

4. Potassium citrate and electrolyte balance

In the cellular economy, potassium is the principal intracellular cation and the chief determinant of the resting membrane potential governing nerve-impulse transmission and skeletal- and cardiac-muscle contraction [4]. The citrate anion is metabolised to bicarbonate, so potassium citrate functions as an alkali load that neutralises dietary acid. This dual mode of action — potassium donation plus base load — underlies the salt's pleiotropic effects on blood pressure, bone density, urinary citrate and renal-stone formation.

4.1 Cardiovascular evidence

A landmark WHO-commissioned systematic review and meta-analysis by Aburto and colleagues established that higher potassium intake lowers blood pressure in adults and is associated with a 24 % reduction in stroke risk with no adverse effect on blood lipids, catecholamines or renal function [31]. More recent dose-response meta-analyses of trials published between 2000 and 2024 have confirmed the inverse relationship between potassium intake and blood pressure in hypertensive subjects and identified a mild dose-response that is most pronounced in higher-sodium consumers [32,33]. A 2014 Women's Health Initiative postmenopausal cohort (n = 90,137, mean follow-up 11 years) reported a hazard ratio of 0.88 (95 % CI 0.79–0.98) for total stroke in the highest quartile of dietary potassium intake versus the lowest, with the effect broadly consistent in hypertensive and non-hypertensive subgroups [34]; an early California cohort reported a 40 % reduction in stroke-associated mortality per 10 mmol increase in daily potassium intake, independent of other cardiovascular risk factors [35]. A meta-analysis of prospective cohort studies found that higher potassium intake was associated with a significantly lower risk of stroke (pooled relative risk of approximately 0.79 for an about 1.64 g/day higher intake), with favourable but non-significant trends for coronary heart disease and total cardiovascular disease [36]. A 2007 Rotterdam Study analysis (n = 5,448, 7.9-year median follow-up) reported a relative-risk reduction for cardiovascular events and all-cause mortality with high potassium intake, particularly in combination with low sodium intake [37]. The Poorolajal 2017 meta-analysis of 23 RCTs concluded that oral potassium supplementation significantly reduced systolic blood pressure by 4.76 mm Hg (95 % CI 0.74–8.78) overall and by 7.16 mmHg (95 % CI 4.06–10.26) when limited to hypertensive subjects [11].

4.2 Acid–base considerations

The citrate anion has direct alkalinising activity. Once absorbed, citrate is metabolised in the liver to bicarbonate, which neutralises dietary acid load, reduces net renal acid excretion, and lowers urinary calcium excretion. The randomised trial by Granchi and colleagues in 2018 reported that 30 mEq/day potassium citrate significantly decreased biochemical markers of bone loss in a randomised double-blind

placebo-controlled pilot study in osteopenic postmenopausal women [38]. The dual action of potassium citrate — moderate potassium elevation with simultaneous base loading — is rarely seen with other potassium salts such as chloride and may therefore explain some of the bone-related advantage [4,38].

4.3 Nephrolithiasis

Potassium citrate is a long-established therapy for hypocitraturic calcium renal stones, since increased urinary citrate chelates urinary calcium and inhibits calcium-oxalate crystallisation. Although MAG Complex does not contain therapeutic doses of potassium citrate, the pairing is consistent with broader systemic alkalinisation rather than direct treatment of nephrolithiasis.

4.4 Potassium dose in MAG Complex

The product's declared 200 mg of potassium citrate per serving does not, by mass, represent the elemental potassium delivered. Elemental potassium from potassium citrate is approximately 38 % of the salt's mass; 200 mg of potassium citrate would therefore represent approximately 76 mg of elemental potassium.

4.5 Bioavailability of potassium salts

The Merschmann 2025 systematic review reported that potassium citrate and KCl exhibit adequate bioavailability, with KCl ranging between 27.4 % and 338.8 % (median 77.6 %), and with no significant difference between KCl and potassium citrate in head-to-head comparisons [1]. The median rather than the arithmetic mean is used because the maximum value is implausibly high and likely reflects analytical outliers, so the authors advise caution against over-interpretation of high-end values. The key implication for MAG Complex is that the 200 mg of potassium citrate provides approximately 76 mg of adequately bioavailable elemental potassium per serving, and that the alkalinising action of citrate is biologically meaningful at the single-serving scale even when the absolute elemental dose is small.

5. Pyridoxal-5′-phosphate, the active coenzyme form of vitamin B6

Vitamin B6 in its phosphorylated form (pyridoxal-5′-phosphate, PLP) is the obligatory cofactor of more than 160 enzymatic reactions, predominantly in amino-acid and neurotransmitter metabolism, but also in glycogen utilisation, haem synthesis, homocysteine transsulfuration and one-carbon metabolism linking to the folate cycle [5,6]. PLP participates in the synthesis of serotonin, dopamine, γ-aminobutyric acid and noradrenaline through decarboxylases — aromatic L-amino-acid decarboxylase in the first case, glutamic-acid decarboxylase in the second — and is essential for the balance between glutamatergic excitation and GABAergic inhibition in the central nervous system [5].

5.1 PLP and GABA

A high-dose vitamin B6 supplementation study by Field and colleagues demonstrated that 1 month of high-dose B6 reduced self-reported anxiety and strengthened visual surround suppression, consistent with increased inhibitory GABAergic influence [15]. In a recently developed pyridoxal-phosphatase knockout animal model, intracellular PLP concentrations in brain, skeletal muscle and red blood cells were three-fold higher than wild-type controls, and brain GABA concentrations rose by approximately 20 %, while dopamine, serotonin, epinephrine and glutamate remained unchanged; the PDXP-deficient mice also exhibited improved spatial learning and memory and a mild anxiety-like phenotype, supporting the proposal that intracellular PLP shapes the excitation–inhibition balance via GAD65-driven GABA synthesis [7,39]. The 2025 monograph on GABA and anxiety-disorder pathophysiology frames the GABA-ergic axis as a clinically actionable target and provides a useful anchor for the linkage between PLP availability and mood regulation [40].

5.2 PLP–magnesium interaction

A preliminary 1981 study by Abraham and others in premenopausal women observed that pyridoxine supplementation raised red-blood-cell magnesium concentrations, providing the historical basis for combining B6 with magnesium salts in clinical formulations [5,6]. A 2018 phase-IV multicentre randomised single-blind trial in healthy adults with severe stress and low magnesaemia reported that the combination

of magnesium plus vitamin B6 was superior to magnesium alone in reducing stress, with secondary analyses suggesting that the B6 component also contributed to changes in magnesium status, mental health and quality of life [5,6]. The pivotal trial administered vitamin B6 predominantly as pyridoxine rather than PLP; although hepatic pyridoxal-kinase converts pyridoxine to PLP, providing PLP directly bypasses this conversion and may be advantageous where hepatic kinase activity is suboptimal. Disorders of B6 metabolism that impair pyridoxal-kinase activity (pyridoxine-responsive epilepsy, PNPO deficiency, classical homocystinuria) demonstrate the clinical relevance of this bypass pathway [41].

5.3 B6 toxicity differentiation: pyridoxine vs. P5P

A long-standing safety concern with vitamin B6 supplementation is sensory peripheral neuropathy observed after prolonged high-dose pyridoxine exposure, typically at gram-level daily doses administered over months, secondary to interference with pyridoxal-phosphatase activity and the resulting disturbance in PLP-dependent GABAergic and serotonergic synthesis. P5P at nutritional doses does not exhibit this safety concern, and the EFSA and IOM have set the nutritional upper level for vitamin B6 explicitly to keep chronic intake below the threshold at which neuropathy risk emerges [6,42]. The MAG Complex dose of 1.9 mg PLP/day is therefore well within the safe envelope.

5.4 Recommended dose and FSSAI compliance

FSSAI permits vitamin B6 in health supplements at a Nutrient Reference Value of 1.9 mg/day for adults, expressed consistently as pyridoxal-5′-phosphate or pyridoxine hydrochloride [16]. The MAG Complex dose of 1.9 mg PLP is therefore fully consistent with recommended intake, offers the active coenzyme form, and complements magnesium's action across the shared pathway of amino-acid and neurotransmitter metabolism [6].

Table 5: B6 species and the differential implications of pyridoxine versus P5P at the MAG Complex dose.

Form	Conversion requirement	Required kinase activity	Toxicity profile at MAG dose
Pyridoxine hydrochloride	Hepatic pyridoxal-kinase phosphorylates to PLP	Normal	Neuropathy risk only at gram-level chronic doses [42]
Pyridoxal-5′-phosphate	None — direct coenzyme form	Not required	No neuropathy signal at nutritional dose [6]
MAG Complex dose 1.9 mg PLP	n/a	n/a	Well within safe envelope; consistent with NRV [16]

6. *Chlorella vulgaris* and the microalgal component

Chlorella vulgaris is a unicellular freshwater green microalga that contains roughly 50–60 % protein, 8–12 % lipids (with a meaningful polyunsaturated fraction), the chlorophylls a and b, the major carotenoids lutein and β-carotene, B-group vitamins (notably folate and riboflavin), vitamin C, vitamin E, bioactive antioxidant molecules and a broad mineral profile that includes magnesium [43,44]. Because *Chlorella* cell walls require mechanical disruption to liberate intracellular contents, most commercial preparations are sold as broken-cell-wall dried powder.

6.1 Evidence from human and animal studies

The most robust human evidence relates to cardiometabolic risk factors. A 2018 meta-analysis of pooled randomised controlled trials concluded that *Chlorella* supplementation significantly reduced total cholesterol and LDL cholesterol, and in subgroup analyses also produced mild improvements in fasting glucose and blood pressure. A 2022 dose-response meta-analysis reported similar reductions in total cholesterol, LDL cholesterol and triglycerides, with significant between-study heterogeneity [43,45]. A 2026 GRADE-assessed systematic review and dose-response meta-analysis ranked the evidence for *Chlorella* on a number of general-population endpoints and supported supplementation benefits for blood lipids, blood pressure and antioxidant capacity [43]. A 2025 systematic review and meta-analysis by Pinto-Leite and colleagues concluded that *Chlorella* and *Spirulina* are reasonable adjuvants to standard cardiovascular risk

factor management [45]. Many individual randomised trials have extended these results to specific populations:

- **Type 2 diabetes:** a 2021 double-blind trial reported that 8 weeks of *Chlorella* supplementation improved glycemic and lipid markers in adults with type 2 diabetes [46].
- **Immune function:** an 8-week double-blind RCT demonstrated significant increases in natural-killer-cell activity, interferon-γ and IL-1β in healthy adults receiving 5 g/day of dried *Chlorella* [47].
- **Gut microbiota:** an exploratory industry co-authored trial reported that the metabolic efficacy of *Chlorella* supplementation depends on the individual's baseline gut microbial profile, suggesting that response is personalised rather than universal [48].
- **Liver-function biomarkers:** a systematic review and meta-analysis of *Chlorella vulgaris* on liver function biomarkers reported mixed but broadly supportive results, with the highest-quality evidence focused on alkaline phosphatase and AST [49].

In animal models including stroke-prone spontaneously hypertensive rats, dietary *Chlorella* lowered blood pressure in a dose-dependent manner (10 % and 20 % *Chlorella* in the diet significantly reduced systolic blood pressure compared with untreated controls), provided protection from hypercholesterolaemia, and showed antioxidant activity [44]. A 2023 Indian randomised double-blind trial of *Spirulina* sauce in type 2 diabetic patients reported reductions in glycemic index, lipid profile and oxidative stress, supporting the broader inference that microalgal adjuncts can complement standard diabetes dietary counselling [50].

6.2 Dose limitation in MAG Complex

A consistent limitation of the published *Chlorella* trial base is the gram-level doses tested (1.5–10 g/day, sometimes up to 10 % of the diet in animal studies), which are 30- to 200-fold higher than the 50 mg of dried powder included in MAG Complex. Therefore, none of the human cardio-metabolic, immunological or liver-function outcomes reported in the *Chlorella* literature can be transferred directly to MAG Complex. The *Chlorella* component is best characterised as a supportive microalgal ingredient contributing minerals, B-group vitamins, carotenoids and a fibre-rich matrix.

6.3 FSSAI status of *Chlorella vulgaris* and quality requirements

Chlorella vulgaris is recognised under the Indian Food Safety and Standards Regulations, 2022 as a permitted ingredient in health supplements and nutraceuticals, subject to specific impurity limits and product-quality standards [16]. Manufacturers must verify heavy-metal limits (lead, cadmium, arsenic, mercury), microbial limits (total plate count, yeast/mould, coliforms, absence of pathogens), and compliance with the relevant chapter requirements for finished-product labelling.

7. Evidence-graded appraisal of the proposed benefits

Each proposed benefit is assessed at the level of the individual ingredients, with the strength of evidence scored on a 5-point Oxford Centre for Evidence-Based Medicine scale adapted for nutrition research. Disease-treatment or disease-prevention language is avoided; only structure–function wording is recommended.

7.1 Nerve and muscle function — strong physiological rationale

This is the most defensible central claim of MAG Complex. Magnesium is required for the Na^+/K^+-ATPase (Section 2.3), acts as a physiological antagonist at voltage-gated calcium channels and NMDA glutamate receptors, and stabilises the resting membrane potential of muscle and nerve cells [2,3,18]. Magnesium deficiency lowers the excitation threshold, generating hyperexcitability, fasciculations, cramps and tetany; correction of deficiency by either intravenous or oral routes reliably normalises the clinical picture [3, 14].

For idiopathic skeletal-muscle cramps, however, the highest-quality clinical evidence base is mixed. A Cochrane review including 11 trials of magnesium supplementation in the older general population did not detect a clinically meaningful benefit of oral magnesium on cramp frequency or intensity. A 2021 multicentre randomised double-blind placebo-controlled study of magnesium oxide monohydrate (the Nocturnal Leg Cramps trial, n = 233) reported a modest but significant reduction in nocturnal leg-cramp frequency, supporting a small positive effect that the Cochrane analysis may have missed.

Suggested structure–function claim: Contributes to normal neuromuscular function, electrolyte balance and muscle relaxation.

Evidence tier: Mechanism = level 4; cramp efficacy = level 1a against benefit with conflicting positive RCT (level 1b).

7.2 Mood — moderate evidence, not product-proven

Randomised and meta-analytic evidence on magnesium and mood has been broadly positive but heterogeneous. A 2023 systematic review and meta-analysis of 11 RCTs in adults with depressive disorder concluded that magnesium supplementation beneficially affected depression severity [14]. A 2018 systematic review in *BJPsych Open* reached a more cautious conclusion: magnesium was associated with benefit only in uncontrolled studies, with no significant effect emerging from the placebo-controlled subset [2,14]. The strongest individual RCT remains a 2017 double-blind placebo-controlled trial in depressed patients with documented magnesium deficiency, where magnesium supplementation significantly improved depression status compared with placebo [2]. Evidence for adults with anxiety and stress is weaker but has accumulated supportive RCTs, including the high-dose vitamin B6 study by Field and colleagues, the modified-microbiota proline-metabolism study by Mayneris-Perxachs and colleagues, and the magnesium–B6 stress trial by Pouteau and colleagues [2, 15, 51].

Mechanistically, the magnesium–B6–GABA link is plausible [5, 7, 15]. Mood-disorder symptoms may reflect impaired GABAergic inhibition, an imbalance that increases as serum and intracellular magnesium fall and as PLP-dependent GAD65 synthesis of GABA becomes substrate-limited. Restoration of the magnesium pool and intracellular PLP is therefore a reasonable target for subclinical mood symptoms, although a primary treatment role is not supported by the available evidence [7, 40].

Suggested structure–function claim: Magnesium and vitamin B6 support normal psychological and nervous-system function; supplementation may help maintain mood balance where dietary intake is inadequate.

Evidence tier: Level 1a (mixed) and 1b (positive RCT in deficiency).

7.3 Migraine — moderate evidence at supraphysiological doses

The clinical literature on magnesium and migraine is one of the oldest and most consistent in the nutraceutical field. Magnesium has been investigated as both acute treatment (intravenous or oral) and prophylactic supplement (daily), with consistent reporting of efficacy in systematic reviews. The protocol for the most recent Cochrane review on magnesium supplementation for migraine prophylaxis frames the central question around episodic and chronic migraine and the equity-related effect of age, sex and economic status, underscoring unresolved questions about dose, formulation and target population [10,52].

Mechanistically, NMDA-receptor blockade by magnesium attenuates the cortical spreading depression thought to drive the migraine aura, while the improvement in mitochondrial oxidative phosphorylation offered by magnesium-ATP repletion reduces neuronal hyper-excitability [8, 38, 53]. Meta-analyses report that intravenous magnesium results in significant relief within the first 24 hours and that oral magnesium significantly reduces the frequency and intensity of migraine attacks [53]. The American Academy of Neurology guideline (since archived, retained as a historical document) classified magnesium as "probably effective" for episodic migraine prevention, alongside riboflavin and MIG-99 feverfew [9]. A 2025 narrative review on magnesium and migraine in *Nutrients* reaffirmed the plausibility of the mechanism and the persistence of the evidence base despite methodological heterogeneity [10]. A 2015 mechanistic review on pathophysiological targets for non-pharmacological treatment of migraine mapped magnesium's NMDA-blockade, mitochondrial support and serotonin-modulating actions onto the major current targets of migraine-non-pharmacological-intervention study [54].

A critical caveat applies for MAG Complex. The successful migraine trials typically administer 400–600 mg of elemental magnesium per day, in some cases as magnesium dicitrate (600 mg/d) [8]. The approximate 121–156 mg of elemental magnesium provided by MAG Complex is below half the doses that have demonstrated efficacy. No own-product data exist, and the migraine benefit cannot be transferred.

Suggested structure–function claim: Magnesium has been investigated as a supportive nutrient for migraine prophylaxis; consult a clinician for migraine-specific dosing.

Evidence tier: Level 1a with respect to magnesium as a class; level 5 ("not-yet-tested") for the multi-salt product.

7.4 Irregular heartbeat — physiological support, not a prevention claim

Magnesium and potassium are essential to cardiac ion-channel behaviour and myocardial electrical stability; deficiency of either ion predisposes the heart to ectopic activity and to ventricular and atrial arrhythmia [4, 14, 18]. The evidence base for oral magnesium and prevention of new-onset atrial fibrillation is largely null in meta-analysis: a 2023 systematic review and meta-analysis found no statistically significant effect of oral magnesium prophylaxis on postoperative or medical AF incidence.

By contrast, intravenous magnesium has therapeutic roles in selected arrhythmias (improving rate control in rapid atrial fibrillation, reducing ventricular arrhythmias in cardiac-surgery and acute-coronary-syndrome settings) and oral magnesium modestly improves vascular function in randomised trials. These IV and supra-physiological settings cannot be transferred to a routine oral nutraceutical.

Suggested structure–function claim: Supports normal cardiac electrical activity, electrolyte balance and muscular function.

Evidence tier: Mechanism = level 4; AF prevention = level 1a null; IV therapy endpoints = level 1a positive.

7.5 Maintenance of blood magnesium — careful wording required

Serum magnesium is tightly regulated and represents a small fraction of total-body magnesium; ionised magnesium may be a more responsive marker, although data on direct clinical translation are limited [3, 13]. An oral supplement increases dietary magnesium intake, may help correct inadequate intake and may contribute to the homeostatic flux between extracellular and bone/intracellular compartments — but cannot, in itself, "maintain serum magnesium" for every consumer in the absence of an underlying deficiency.

Suggested structure–function claim: Provides multiple dietary sources of magnesium to help meet daily magnesium requirements and support whole-body magnesium homeostasis.

Evidence tier: Level 1b for ionised-magnesium kinetics; level 4 for clinical interpretation in non-deficient populations.

7.6 Digestive health — dose- and form-dependent

Poorly absorbed magnesium salts osmotically retain water in the intestinal lumen and improve stool passage. Double-blind randomised placebo-controlled trials in adults have shown that:

- A natural mineral water rich in magnesium and sulphate significantly increased complete spontaneous bowel movements and improved stool consistency compared to placebo in a 3-week parallel-group study of adults with functional constipation, an effect attributable to the osmotic retention of water and electrolytes in the intestinal lumen [55].
- Magnesium oxide, as monotherapy or in head-to-head comparison with senna, is effective for chronic constipation and transit-related symptoms, with benefit emerging from week two in placebo-controlled trials [29].

The mechanism that confers the laxative effect, however, is identical to the mechanism that produces adverse effects in non-target populations — dose-dependent osmotic diarrhoea, abdominal cramping and intestinal gas. Gastrointestinal effects must therefore be portrayed as dose-dependent and dose-intended rather than as a generic benefit.

Suggested structure–function claim: Certain magnesium salts may support normal bowel regularity, particularly at prudent bowel-targeted doses.

Evidence tier: Level 1b for chronic-constipation outcomes; level 4 for general "digestive health" extrapolation.

7.7 Sleep quality — promising but low-certainty evidence

The underlying rationale for magnesium in sleep is that magnesium modulates NMDA-receptor-mediated excitation, supports GABAergic inhibition via the PLP-dependent GAD65 pathway, and reduces nocturnal cortisol and sympathoadrenal activity by stabilising autonomic balance [5,7,15]. A 2021 systematic review and meta-analysis of three RCTs in 151 older adults with insomnia found a pooled reduction in sleep-onset latency of 17.36 minutes (95 % CI −27.27 to −7.44, p = 0.0006) after oral magnesium supplementation, although the limited number of trials, the moderate-to-high risk of bias across studies, and the heterogeneity of dosing and timing meant that the GRADE certainty was assessed as "low to very low" [56]. In the cited trials, oral magnesium preparations of 320, 500 and up to 729 mg of elemental magnesium per day were administered, generally as magnesium oxide or magnesium citrate, in 8-week courses [56]. The 2024 *Sleep Medicine: X* RCT of magnesium-L-threonate for adults with self-reported sleep problems improved sleep quality and daytime functioning but tested a salt form not present in MAG Complex.

Suggested structure–function claim: Magnesium may help support normal sleep quality, particularly where intake or status is inadequate.

Evidence tier: Level 1a meeting but with low-to-very-low GRADE certainty; level 4 for any quantitative benefit claim.

7.8 Thyroid health — weak, associative evidence only

Observational evidence associates low serum magnesium with an increased prevalence of positive antithyroid antibodies and hypothyroidism. A 2018 cross-sectional study linked severely low serum magnesium to higher odds of positive anti-thyroglobulin antibody and hypothyroidism; a 2020 systematic review and meta-analysis of trace-element status reported similar associations; and a 2023 cross-sectional analysis of multiple circulating blood metals confirmed the qualitatively similar inverse association between magnesium and indices of thyroid dysfunction [57,58]. None of these data sets includes a randomised trial demonstrating that magnesium supplementation improves thyroid function, and a 2026 broad endocrine review frames magnesium's role as modulatory rather than therapeutic [59].

Suggested structure–function claim: Magnesium contributes to normal cellular energy metabolism, broadly relevant to endocrine physiology; specific thyroid benefits of the product are not established. Consumers on levothyroxine should take MAG Complex several hours apart from thyroid medication.

Evidence tier: Level 3 (associative) for thyroid observation; level 1b for the levothyroxine interaction.

7.9 Target populations and product positioning

Table 6: Recommended consumer sub-populations and the supporting evidence for each.

Consumer sub-population	Why MAG Complex is appropriate	Strength of evidence
Healthy adults with low dietary magnesium intake	Provides multiple dietary magnesium salts, addresses possible inadequacy	Mechanism + pharmacokinetic level
Adults under chronic stress with self-reported low magnesium	Magnesium + PLP combine to support stress-related metabolic demand	Phase-IV RCT positive for combination [5]
Older adults at risk of inadequate intake and reduced absorption	Magnesium + potassium cohort targets; low opioid risk profile	Mechanism + cohort evidence
Adults with self-reported constipation dominated by slow transit	Osmotic magnesium support via oxide component	Double-blind RCTs

Adults with documented depressed status secondary to deficiency	Magnesium repletion and PLP coadjuvancy	DB-RCT positive in deficiency subset
Adults on levothyroxine therapy	Co-incident electrolyte and PLP support, with timing caution	ThyroMag trial interaction signal [30]
Adults at risk of inadequate potassium intake	Provides supplementary K with alkalinising citrate	Dose-response meta-analyses [11,33]
Patients on potassium-sparing diuretics, ACE inhibitors, ARBs	**Avoid because of additive potassium risk**	Level 4 + mechanism
Individuals with moderately severe renal impairment (eGFR < 30 mL/min/1.73 m²)	**Use only under medical supervision**	Mechanism
Pregnant and lactating women	May support the elevated RDA during pregnancy; consult clinician	Life-stage recommendation [2,14]

7.10 Strength-of-evidence summary

Table 7: Strength-of-evidence summary for the eight proposed benefits and two additional outcomes.

Proposed benefit	Best available evidence	Evidence level	Recommended positioning
Nerve and muscle function	Mechanistic reviews; cryptogenic cramp RCTs (mixed)	1a (against cramp); 1b (positive nocturnal RCT); mechanism = 4	Primary structure–function rationale
Mood	Meta-analyses (mixed); DB-RCT in deficiency	1a / 1b	Cautious supportive wording
Migraine	Systematic review + dose-response meta-analysis at higher doses	1a	Supportive research area; not a treatment claim
Cardiac rhythm	Mechanistic reviews; null meta-analysis for AF prophylaxis	1a (null) + mechanism	Contributes to normal cardiac electrical activity
Blood magnesium	Ionised-magnesium kinetics; homeostasis reviews	1b / 4	Supports intake and homeostasis only
Digestive health	Double-blind RCTs of magnesium oxide, magnesium-rich mineral water	1b	May support bowel regularity; dose-dependent
Sleep	One meta-analysis + small RCTs; low certainty	1a (low–very-low GRADE)	May support sleep-related physiology
Thyroid	Cross-sectional/observational only	3	Not a principal claim; note levothyroxine timing
Bone (potassium citrate)	Randomised trials + alkaline potassium meta-analyses	1a / 1b	Supports bone-related physiology at larger K doses than product delivers
Cardiovascular (potassium)	DASH pattern; WHO/Aburto meta-analysis; cohort evidence	1a / 2a (stroke)	Not transferable at product dose

7.11 Defensible label-claim framework

A central requirement of any nutraceutical monograph is to define the wording that can be supported by the cited evidence and the wording that should be avoided. The following table sets out the structure–function statements defensible for MAG Complex and the corresponding non-defensible extensions.

Table 8: Defensible versus non-defensible label-claim framework under FSSAI permitted statements.

Benefit area	Defensible structure–function statement	Statement to AVOID	Why to avoid
Nerve/muscle	"Supports normal neuromuscular function and muscle relaxation"	"Cures restless-leg syndrome/fibromyalgia"	Disease-treatment claim not authorised
Mood	"Supports normal psychological function"	"Effective treatment for clinical depression"	Level-1a evidence mixed; product not tested
Migraine	"Magnesium has been investigated as a supportive nutrient"	"Clinically proven to prevent migraine"	Trials at higher doses; no product data
Cardiac rhythm	"Supports normal cardiac electrical activity"	"Prevents atrial fibrillation/arrhythmia"	Meta-analysis null for AF prevention
Blood magnesium	"Provides dietary sources of magnesium for normal intake"	"Normalises serum magnesium in deficiency"	Serum insensitive; product not tested
Digestive health	"Certain magnesium salts may support bowel regularity"	"Relieves IBS / colitis / IBD"	Indication-specific; not authorised
Sleep	"May support normal sleep physiology"	"Clinically proven insomnia treatment"	Low GRADE certainty; product not tested
Thyroid	"Supports normal cellular energy metabolism broadly relevant to endocrine physiology"	"Improves thyroid function / replaces levothyroxine"	No randomised evidence; levothyroxine-interaction caution

8. Safety, tolerability, interactions, special populations, quality and regulatory considerations

8.1 Safety of oral magnesium

Oral magnesium supplementation is generally well tolerated in healthy individuals with normal renal function. The principal adverse effect is dose-dependent osmotic diarrhoea, driven chiefly by the less-absorbed salts in the formulation (most notably magnesium oxide and the phosphate tribasic). The US Institute of Medicine has set a tolerable upper intake level of 350 mg/day of supplemental magnesium (in addition to dietary magnesium) for healthy adults, on the basis of the dose–response curve for osmotic diarrhoea [2]. The EFSA has set a lower UL of 250 mg/day for magnesium added to food and beverages, including in supplements, and notes that at the proposed maximum use levels of magnesium citrate malate, that UL is exceeded [26]. The approximated ~121–156 mg of elemental magnesium in MAG Complex lies comfortably below both US and EFSA ceilings.

8.2 Renal impairment and electrolyte maintenance

Both magnesium and potassium should be used under medical supervision in people with impaired renal function. Reduced urinary excretion can lead to hypermagnesaemia and hyperkalaemia, with potentially severe cardiac and neuromuscular consequences. Potassium-containing products warrant particular caution in patients taking potassium-sparing diuretics (amiloride, triamterene), ACE inhibitors, angiotensin-

receptor blockers, heparin, trimethoprim or any other agent that raises serum potassium. In the presence of limb or peripheral oedema, palpitation, or recent diagnostic confirmation of elevated serum potassium or creatinine, consumers should seek medical advice before use.

8.3 Drug-supplement interaction framework

Magnesium can reduce the absorption of certain medicines, particularly antibacterials (tetracyclines, fluoroquinolones), bisphosphonates (alendronate, risedronate), iron salts and L-thyroxine (the last referenced in Section 8.4 below). The standard guidance is to separate dosing by 2–4 hours where these interactions are clinically relevant. The Canadian Family Physician administrative-spacing table provides the following recommended separations when magnesium or other di-/trivalent cations are co-administered with interacting medications.

Table 9: Recommended cation-/drug-dosing separations based on the Canadian Family Physician 2023 spacing table.

Medication class	Examples	Take cation BEFORE medication	Take cation AFTER medication	Notes
Bisphosphonates	Alendronate, risedronate, etidronate	30 min–2 h	30 min–2 h	Earlier separation recommended before medication
Thyroid medications	Levothyroxine, desiccated thyroid	Ideally 4 h (some studies 2 h)	Ideally 4 h	Aligns with ThyroMag trial finding
Fluoroquinolone antibiotics	Ciprofloxacin	2 h	6 h	
Fluoroquinolone antibiotics	Moxifloxacin	4 h	8 h	
Fluoroquinolone antibiotics	Levofloxacin, delafloxacin	2 h	2 h	
Tetracycline antibiotics	Doxycycline, minocycline, tetracycline	2 h	3–4 h	Doxycycline and minocycline may be taken with milk or food

The spacing for levothyroxine aligns with the ThyroMag trial interaction datum showing magnesium aspartate and, to a lesser extent, magnesium citrate reduce thyroxine absorption (Section 8.4). No data are available on direct interactions between MAG Complex and warfarin, antiepileptics or oral antidiabetics; mechanistic plausibility is low but cannot be excluded [60–62].

8.4 Levothyroxine interaction

The 2025 ThyroMag open-label randomised crossover trial by Attinger and colleagues reported that co-administration of either magnesium citrate or magnesium aspartate with levothyroxine (1 mg single dose, liquid formulation) reduced total thyroxine exposure over the next 24 h compared with levothyroxine given alone. Magnesium aspartate significantly reduced thyroxine AUC by 12 % (geometric mean ratio 0.88, 95 % CI 0.81–0.95, p = 0.002) and significantly reduced Cmax by 7 % and increased Tmax by 17 %; magnesium citrate reduced thyroxine AUC non-significantly by 7 % (GMR 0.93, 95 % CI 0.86–1.01, p = 0.076), with smaller changes in Cmax and Tmax. Because both citrate and aspartate salts are present in MAG Complex, a temporal separation of at least 4 hours is recommended between MAG Complex and

thyroid-hormone replacement. If co-administration cannot be avoided, the trial authors suggest that the citrate component would be the better-tolerated option, as its effect on thyroxine absorption was smaller [30].

8.5 Vitamin B6 safety

At nutritional doses (1.9 mg/day, the EU NRV), the safety profile of vitamin B6 is excellent. The historical safety concern about pyridoxine neuropathy relates to prolonged high-dose (typically gram-level) supplementation with pyridoxine specifically; this risk does not extend to pyridoxal-5′-phosphate at reference-intake levels. Wilson and colleagues' 2019 review of disorders of vitamin B6 metabolism highlights that pyridoxine-responsive disorders provide insights into the safety profile of PLP and the dosing thresholds at which chronic high-dose exposure produces sensory peripheral neuropathy in normal subjects [41,42].

8.6 Special populations

Table 10: Recommendations for special populations (pregnancy, lactation, paediatric, older adults, renal impairment).

Population	**Recommendation**	**Cautions**	Evidence level
Pregnancy	May be considered as adjunct to dietary intake, particularly in second and third trimesters where hypomagnesaemia is common	Clinician supervision; standard UL applies from age 9 upward [2]	Mechanism + correlation in pregnancy
Breastfeeding	Maternal intake supports breastmilk mineral content; standard UL applies	Clinician supervision	Mechanism + life-stage intake data [2]
Paediatric (< 9 y)	No UL established for pre-school children; MAG Complex is not recommended as a routine supplement in < 9 y	Magnesium content exceeds life-stage RDA proportionally to body mass	UL data absent [2]
Paediatric constipation (clinical scenario)	Magnesium oxide at clinical doses (>300 mg/d) is indicated in functional chronic constipation	Use only under paediatric supervision	Kubota 2020 RCT [29]
Older adults	Most appropriate cohort because of likely reduced intake and polypharmacy-related urinary losses	Standard UL applies; check eGFR	Mechanism + cohort data
Moderate renal impairment (eGFR 30–60 mL/min/1.73 m^2)	Reduced renal excretion of magnesium and potassium; use only under clinician supervision	Monitor serum magnesium and potassium	Mechanism

Severe renal impairment (eGFR < 30 mL/min/1.73 m²)	Avoid or use only with specialist endorsement	High risk of accumulation	Mechanism
Concomitant levothyroxine	Separate MAG Complex from levothyroxine by minimum 4 h	Reduces thyroxine exposure	ThyroMag RCT [30]
Concomitant potassium-sparing diuretic, ACEi, ARB	Avoid MAG Complex	Additive hyperkalaemia risk	Mechanism

8.7 Quality, chromatography and stability

Analytical surveys of marketed multivitamin/mineral supplements show that measured magnesium and other mineral contents deviate from declared label values — most ingredients, magnesium included, exceed the label because manufacturers add overages to compensate for expected shelf-life losses so that products remain label-compliant to the end of shelf life [63]. Storage conditions — particularly temperature and humidity — are recognised determinants of the chemical stability of solid dosage forms, so accelerated stability testing and an ongoing stability-monitoring programme are important for establishing and verifying shelf life [64]. The EFSA scientific opinion on magnesium citrate malate notes that the data provided demonstrate a product that is stable throughout its proposed shelf life [26].

From a quality-chemistry standpoint, magnesium salts used in MAG Complex are routinely characterised by ICP-OES for elemental magnesium, by complexometric back-titration with EDTA, and by ion chromatography for the counter-ion. PLP is best characterised by reverse-phase HPLC with fluorescence detection (λex 320 / λem 400 nm) with strict protection from light and humidity. *Chlorella vulgaris* biomass is profiled for protein content, carotenoids, chlorophylls (spectrophotometry) and minerals, and identity is confirmed by 18S rRNA sequencing or species-specific PCR.

The key implication for MAG Complex is that formulation finishing (particle size, excipients, coating, packaging) is a meaningful determinant of stability and that the manufacturer should establish a stability-monitoring programme aligned with FSSAI requirements and shelf-life claims.

8.8 FSSAI regulatory positioning

The Indian Food Safety and Standards Authority recognises magnesium aspartate, chloride, gluconate, phosphate tribasic and oxide, as well as potassium citrate, as permitted mineral sources in health supplements and nutraceuticals, and requires the rationale for ingredient combinations to be supported by scientific and technical evidence [16]. Vitamin B6 in the form of pyridoxal-5′-phosphate is recognised as a permitted source of vitamin B6. *Chlorella vulgaris* is recognised under the 2022 FSSAI regulations for nutraceuticals as a permitted ingredient, subject to specific impurity and labelling limits [12,16]. Structure–function and general well-being claims are permitted when supported by accepted scientific evidence; claims to treat, cure, mitigate or prevent a disease require specific regulatory authorisation. The general US dietary-supplement regulatory framework anchored by the Dietary Supplement Health and Education Act provides an additional reference standard for permitted structure–function labelling [12].

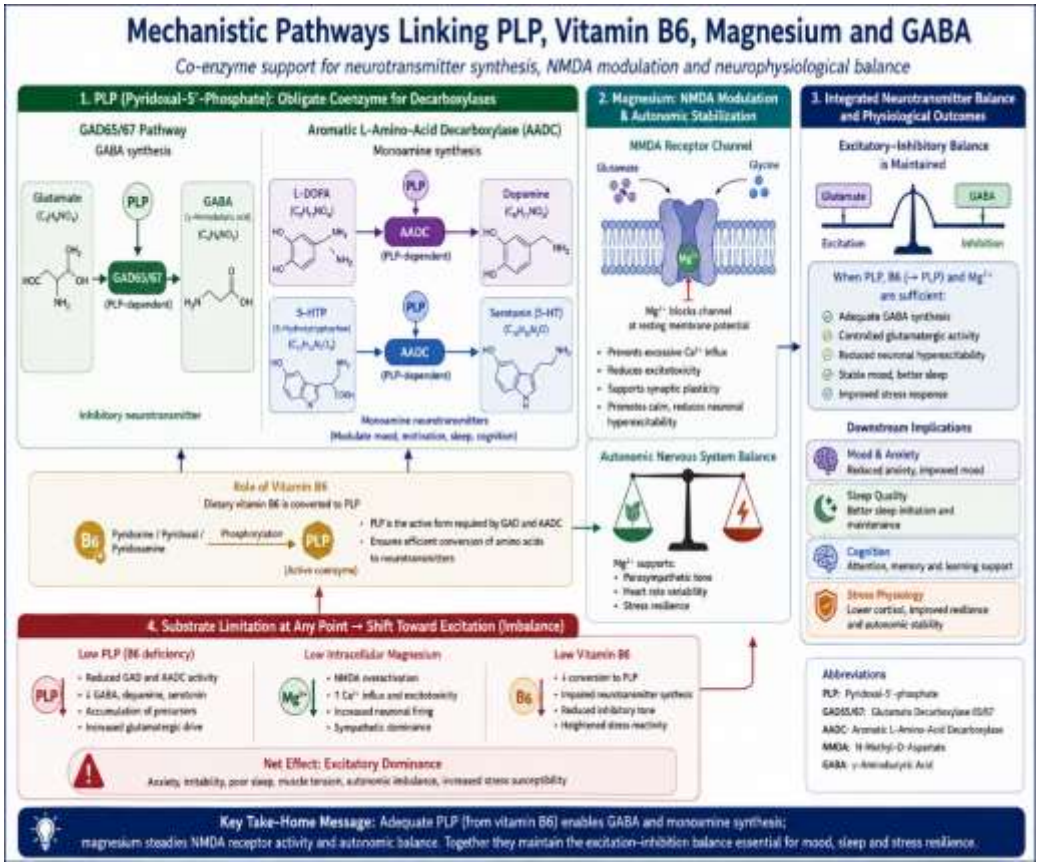

Fig 6: Mechanistic pathways linking PLP, B6, magnesium and GABA (schematic). The diagram shows pyridoxal-5′-phosphate as the obligate coenzyme of GAD65 (which converts glutamate to GABA) and of aromatic L-amino-acid decarboxylase (which converts L-DOPA to dopamine and 5-HTP to serotonin). Magnesium modulates NMDA-receptor activity and stabilises autonomic balance. Together, the three substrates support a balance between excitatory glutamatergic neurotransmission and inhibitory GABAergic signalling, with downstream implications for mood, sleep and stress physiology. Substrate limitation at any point in this network (low PLP, low intracellular magnesium, low B6) shifts the balance toward excitation, producing the phenomenology described in Sections 7.2 and 7.7.

9. Research gaps and future directions

No pharmacokinetic study has characterised the elemental-magnesium delivery and absorption profile of this specific five-salt combination in a placebo-controlled head-to-head design, and no randomised placebo-controlled trial has evaluated the finished MAG Complex product for any endpoint. The bioavailability of trimagnesium phosphate in humans is not established by direct study. The clinical contribution of a 50 mg dose of *Chlorella vulgaris* is unknown. The declared potassium contribution and its percentage of the reference intake require reconciliation with the certificate of analysis [3, 12].

9.1 Comparator landscape — where MAG Complex sits

Table 11: Comparative positioning of MAG Complex against common comparator formulations.

Comparator formulation	Typical elemental Mg per dose	Salt profile	Principal positioning	Where MAG Complex differs
Standard magnesium oxide tablet	240–400 mg	Single high-density inorganic salt	Laxative and inexpensive general magnesium	Glycogen salt load is single-form and poorly absorbed; additive oxide dose in MAG

				Complex is moderated and combined with better-soluble salts
Standard magnesium citrate	200 mg	Single organic salt	Bowel function and general magnesium	Magnesium citrate is present as a sub-component in MAG Complex; equivalence assumed for clinical contexts
Magnesium amino-acid chelate	200 mg	Single organic chelate	Mood, sleep and general supplementation	Magnesium glycinate-class behaviour; MAG Complex adds PLP and elemental coverage
Picosized magnesium chloride	300 mg	Solubilised nanosized $MgCl_2$	Bioavailability focus; ionised-magnesium kinetics	Comparable bioavailability target; MAG Complex needs a similar particle-finish product-specific test
Magnesium glycinate	200–400 mg	Single organic salt	Mood and sleep focus	MAG Complex adds PLP (P5P) as a GABA-axis support partner
Magnesium-L-threonate	144–2000 mg	Single organic salt	Cognitive and sleep focus with BBB penetration claim	Different target; not directly comparable
Potassium citrate	30–60 mEq (~1,170–2,340 mg)	Single potassium citrate	Renal-stone prevention and bone	MAG Complex delivers a sub-therapeutic K citrate dose; cannot substitute for a potassium-citrate supplement at therapeutic dose
Multi-mineral multivitamin	80–100 mg elemental Mg	Combination product	General nutrition	Most multi products do not include P5P or *Chlorella vulgaris*

9.2 Priority research agenda

1. **Analytical confirmation** of elemental magnesium (per salt and per serving), elemental potassium, P5P assay and stability, and identity/microbial/heavy-metal testing for *Chlorella vulgaris*, anchored by the FSSAI compliance framework.
2. **Single-dose and steady-state pharmacokinetics** using the $MgCl_2$-style protocol of Zhan and colleagues, with whole-blood ionised magnesium as the primary endpoint and 24-hour urinary magnesium as a secondary endpoint [13].
3. **Randomised, placebo-controlled, double-blind trials** in defined sub-populations (e.g., working adults with self-reported low intake/stress, post-menopausal women with low bone mass, older adults with insomnia or self-reported muscle cramps). Endpoints should be selected from tier-1 outcome measures available to nutraceuticals: PSS-10 for stress, EQ-5D-5L for quality of life, ISI and PSQI for sleep, blood ionised magnesium for status.
4. **Specific migraine prophylaxis RCT** if the dose is supported by supplemental elemental magnesium escalation, conducted under headache-clinic supervision with MIDAS, HIT-6 and migraine-day count as primary endpoints [8, 9, 52].
5. **Cardiovascular pharmacokinetic/pharmacodynamic study** in mildly hypertensive older adults, with 24-hour ambulatory BP monitoring as the principal endpoint and supplemented by plasma renin and aldosterone.

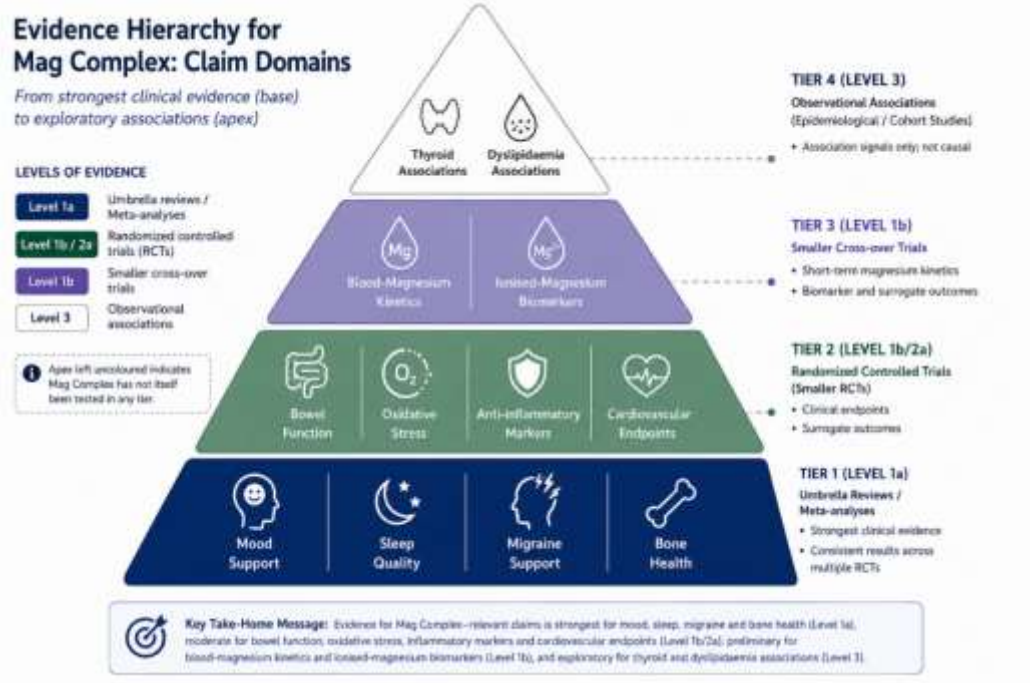

Fig 7: Therapeutic and physiological evidence pyramid for MAG Complex (described). A four-tier pyramid: at the base, mood, sleep, migraine and bone are anchored by umbrella reviews or meta-analyses (level 1a); in the second tier, bowel function, oxidative stress, anti-inflammatory markers and cardiovascular endpoints rest on smaller RCTs (level 1b/2a); in the third tier, blood-magnesium kinetics and ionised-magnesium biomarkers rest on small cross-over trials (level 1b); at the apex, thyroid and dyslipidaemia associations are level-3 observational. The pyramid's apex is left uncoloured to denote that MAG Complex has not itself been tested in any tier.

10. Practice points and clinical takeaways

- **For healthy adults with low dietary magnesium intake (estimated ~30 % of population):** A multi-salt supplement that pairs absorption-friendly organic salts with a clinical-grade oxide sub-component offers an appropriate structure–function support; the entire supplement should remain below the EFSA UL of 250 mg/day supplemental Mg for adults [2,26].
- **For older adults on polypharmacy:** Begin with one serving per day; monitor for osmotic GI symptoms, particularly if the consumer is on a PPI, loop or thiazide diuretic. Coordinate with the treating physician; check eGFR before chronic use [14].
- **For adults on levothyroxine:** Schedule MAG Complex at least 4 h apart from thyroid medication. The ThyroMag trial reports a 12 % thyroxine AUC reduction with magnesium aspartate and a non-significant 7 % reduction with citrate, with the citrate component being the better-tolerated option [30].
- **For adults with self-reported constipation dominated by slow transit:** The magnesium-oxide subcomponent in MAG Complex may support bowel regularity at the standard serving; if no effect is observed at one serving, escalate slowly up to the diarrhoeal threshold, but stay well within the UL [29].
- **For migraine:** Magnesium has class-level RCT evidence for prophylaxis at 400–600 mg of elemental Mg per day; the MAG Complex dose is below this range. Patients seeking migraine-specific prevention should consult a clinician for a higher-dose formulation [8, 9, 52].
- **For mood and stress:** The combination of magnesium and PLP has phase-IV RCT support in deficient adults [5,6]; for individuals taking SSRIs/SNRIs, no direct interaction is expected but co-administration is reasonable.
- **For sleep:** Effect sizes are modest and certainty is low; consider as adjunct rather than primary therapy, and use for ≥8 weeks before judging efficacy [56].
- **For thyroid:** The product should not be used in place of thyroid medication; the section 7.8 wording is the only supportable framing [59].

- **For renal impairment or concomitant K-sparing diuretic / ACEi / ARB:** Avoid or use only under specialist supervision [4].

11. Conclusion

MAG Complex combines five magnesium salts (trimagnesium phosphate, chloride, gluconate, oxide and aspartate) with potassium citrate, pyridoxal-5′-phosphate at nutritional dose, and a small quantity of dried *Chlorella vulgaris* powder. The design is physicochemically coherent: combining low-density soluble salts with high-density insoluble ones balances elemental-magnesium delivery against gastrointestinal tolerability, and the inclusion of potassium and PLP addresses the established magnesium–potassium and magnesium–B6 cross-talks that drive transmembrane excitability, electrolyte balance and the GAD65-mediated GABA synthesis pathway. The strongest evidence supports the role of the product in normal neuromuscular function, electrolyte balance, support of cardiovascular physiology and nutritional contribution to magnesium intake. Meta-analytic evidence supports magnesium in migraine prophylaxis at doses that substantially exceed the ~121–156 mg of elemental magnesium delivered by one serving; magnesium-rich mineral salts for chronic constipation; and potassium and alkaline potassium salts for blood pressure and bone endpoints. Evidence for mood and sleep is positive but heterogeneous and of lower certainty; evidence for a direct thyroid benefit is limited to cross-sectional associations and to the levothyroxine-interaction datum that argues for temporal separation between the product and thyroid medication. Within the FSSAI permitted structure–function framework, defensible label claims are anchored to mechanism and ingredient-class evidence; disease-treatment language is not supportable on the current evidence base [12, 16].

The completion of this review does not certify that the finished product has clinically demonstrated effects. The claims that may be derived safely from the present evidence stream are structure–function statements aligned with nutritional physiology, accompanied by quantitative and temporal guidance (levothyroxine separation, drug-spacing per Section 8.3) where clinical caution is warranted. Manufacturer-initiative steps — analytical confirmation per batch, a single-dose and steady-state pharmacokinetic study with an ionised-magnesium endpoint, a stability-monitoring programme and randomised placebo-controlled trials in defined populations — are required before any claim of clinical efficacy can be transferred from the individual ingredients to MAG Complex itself.

Declarations

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Competing interests. All authors are employees of Biotex Life Solutions Pvt. Ltd., the manufacturer of MAG Complex, the product reviewed in this study. This presents the authors with a conflict of interest, which was disclosed in the review. The conflict of interest was mitigated by evaluating the evidence on a per-ingredient basis according to the study design, using primary and secondary peer-reviewed data, discussing the evidence's lack of presence and heterogeneity, and avoiding any claims on the efficacy of the tested product itself. Authors confirm no additional financial or personal conflicts of interest, which could influence the review's outcome.

Author contributions. Authors conceived the review scope and drafted the ingredient-physiology and mechanism sections, evidence appraisal, formulation-rationale synthesis and manuscript coordination. All authors critically revised the manuscript and approved the final version.

Data availability. Data sharing does not apply to this article: no new datasets were generated or analysed. All sources cited are publicly available and listed in the References.

Ethics approval and consent to participate. Not applicable. This is a narrative review of previously published literature and did not involve human participants, identifiable human data or animals.

Consent for publication. Not applicable.

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Disclaimer: This article is an ingredient-based narrative review that summarises ingredient-level evidence relevant to MAG Complex formulated by Biotex Life Solutions. The findings apply to magnesium, potassium, vitamin B6 and *Chlorella vulgaris* as classes of nutraceutical ingredients, not to the finished MAG Complex product. No claim is made that MAG Complex has been shown in clinical trials to diagnose, treat, cure or prevent any disease. Structure–function statements must remain within the framework of applicable FSSAI regulations and be supported by the certificate of analysis for each production batch, consistent with FSSAI requirements for health supplements and nutraceuticals (and with analogous international frameworks such as the US Dietary Supplement Health and Education Act) [12,17].

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