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## **Magnesium and Mental Health: A Review of Its Role in Anxiety, Sleep Disorders and Depression**

Aleksandra Kowalczyk (corresponding author)

ORCID: <https://orcid.org/0009-0002-9523-2367>

E-mail: [olakowalczyk2001@wp.pl](mailto:olakowalczyk2001@wp.pl)

Medical University of Silesia in Katowice, Poland

Adrian Pączek

ORCID: <https://orcid.org/0009-0009-5151-7556>

E-mail: [adrjanalan@gmail.com](mailto:adrjanalan@gmail.com)

Medical Center MarMedicam in Jaworzno, Poland

Paweł Dyczek

ORCID: <https://orcid.org/0009-0008-6607-6231>

E-mail: [dyczek@onet.eu](mailto:dyczek@onet.eu)

SPZOZ Health Center in Bestwina, Poland

Wiktoria Staniszevska

ORCID: <https://orcid.org/0009-0002-8964-404X>

E-mail: [wiktoria.staniszevska@gmail.com](mailto:wiktoria.staniszevska@gmail.com)

SPZOZ Health Center in Bestwina, Poland

Julia Hofman

ORCID: <https://orcid.org/0009-0008-2609-8292>

E-mail: [juliahofman2001@gmail.com](mailto:juliahofman2001@gmail.com)

Medical University of Silesia in Katowice, Poland

Sylwia Lach

ORCID : <https://orcid.org/0009-0002-9638-3749>

E-mail: [lachsylwia@interia.eu](mailto:lachsylwia@interia.eu)

Independent Public Health Care Institution of the Ministry of Internal Affairs and Administration in Kielce, Poland

Ilona Bednarek

ORCID: <https://orcid.org/0009-0009-9657-4132>

E-mail: [ilona2023@interia.pl](mailto:ilona2023@interia.pl)

Provincial Specialist Hospital in Czerwona Góra, Poland

## Abstract

**Background.** Magnesium is a key cation involved in neurotransmission, regulation of the hypothalamic–pituitary–adrenal axis and sleep–wake control. Observational studies and reviews suggest that low magnesium intake or status is linked with more depressive symptoms, anxiety and sleep disturbances, although results are not fully consistent.

**Objective.** To summarise current evidence on the role of magnesium in depression, anxiety and sleep disorders in adults, with emphasis on human data and clinical practice.

**Methods.** A narrative review of PubMed and other databases was performed. Priority was given to systematic reviews, meta-analyses, controlled trials and larger observational studies, including recent articles from the Journal of Education, Health and Sport.

**Results.** Cohort and cross-sectional studies generally show an inverse association between dietary or serum magnesium and depressive symptoms. Some meta-analyses of trials report small-to-moderate reductions in depressive scores with oral magnesium, particularly in people with low baseline intake or somatic comorbidity, whereas others find limited benefit. For anxiety, trials suggest a possible anxiolytic effect, often with combination preparations, but evidence remains scarce and heterogeneous. In older adults with insomnia, small trials and reviews indicate modest improvements in sleep quality, and observational data link higher magnesium intake with fewer sleep complaints. Overall study quality is low to moderate.

**Conclusions.** Suboptimal magnesium status appears to be associated with a higher burden of depressive, anxiety and sleep symptoms. Magnesium supplementation may provide modest, potentially clinically relevant benefit as an adjunct in selected

adults, especially those with low intake or deficiency, but current data do not support its use as monotherapy. Further high-quality trials are needed to define optimal dosing, duration and target groups.

**Keywords:** magnesium; depression; anxiety; sleep disorders; insomnia; nutritional psychiatry; dietary supplements; micronutrients.

## 1. Introduction

Depression, anxiety disorders and sleep disturbances are among the most common mental health problems and frequently co-occur, contributing to impaired quality of life, functional disability and increased healthcare use. Despite the availability of effective pharmacological and psychotherapeutic interventions, many patients experience only partial remission or undesirable adverse effects, which has stimulated interest in safe, low-cost adjunctive strategies, including nutritional and micronutrient approaches. Magnesium has received particular attention because of its central role in neurobiology and stress regulation. [1–4]

Magnesium is a major intracellular cation and cofactor for hundreds of enzymatic reactions involved in energy metabolism, neurotransmitter synthesis and neuronal excitability. [2,4] Experimental and clinical data indicate that magnesium modulates N-methyl-D-aspartate (NMDA) receptor activity, enhances  $\gamma$ -aminobutyric acid (GABA)–mediated inhibition, influences monoaminergic transmission and attenuates hypothalamic–pituitary–adrenal (HPA) axis hyperactivity and neuroinflammation. [2,4–6] These pathways are thought to play a key role in the pathophysiology of depression, anxiety and disturbed sleep, providing a plausible mechanistic link between magnesium status and mental health outcomes. [2,4–6]

Over the last two decades, several observational studies and systematic reviews have investigated the association between dietary or serum magnesium and depressive symptoms. A systematic review by Derom et al. reported that most cross-sectional and cohort studies found an inverse relationship between magnesium intake and depression, although effect sizes and methods varied considerably. [1] More recently, a GRADE-assessed dose–response meta-analysis confirmed that lower dietary magnesium intake is associated with a higher risk of depression in adults. [7] Individual studies in primary care and community populations have

shown similar inverse associations for both dietary intake and circulating magnesium concentrations. [8–11] However, the observational design of these studies limits causal inference, and potential residual confounding cannot be excluded. [1,7–11]

Evidence from intervention studies is growing but still limited. Randomised controlled trials in elderly patients with type 2 diabetes and comorbid depression, as well as in adults with documented hypomagnesaemia, suggest that oral magnesium supplementation can reduce depressive symptom scores to an extent comparable with standard antidepressant therapy or placebo-controlled improvement. [12,13] Meta-analyses of randomised trials indicate overall small-to-moderate antidepressant effects of magnesium supplementation, particularly in individuals with low baseline intake or somatic comorbidity, but highlight heterogeneity in populations, doses and formulations, and generally modest methodological quality. [3,14,15] Case reports and small open-label studies further support a potential benefit, but their design precludes firm conclusions. [16]

For anxiety and stress-related symptoms, the evidence base is smaller. A systematic review by Boyle et al. concluded that magnesium supplementation may improve subjective anxiety and stress in selected populations, but the trials were usually small, often combined magnesium with other ingredients and used diverse outcome measures. [17] A narrative review from the same journal that hosts the present article (*Journal of Education, Health and Sport*) summarised experimental and clinical data supporting a relationship between magnesium deficiency and anxiety, and reported symptomatic improvement after supplementation, while again underscoring the low to moderate quality of the available studies. [18] Preclinical work shows that dietary magnesium deficiency induces anxiety-like behaviour and HPA-axis dysregulation in rodents, strengthening the biological plausibility of these clinical observations. [6]

Sleep is another domain in which magnesium may play a relevant role. A randomised, double-blind, placebo-controlled trial in older adults with primary insomnia demonstrated that magnesium supplementation improved sleep onset latency, sleep time and sleep efficiency, as well as subjective sleep quality. [19] A subsequent systematic review and meta-analysis focusing on older adults suggested that oral magnesium may confer modest benefits on insomnia symptoms, while emphasising small sample sizes and heterogeneity. [20] A broader systematic review of magnesium and sleep health concluded that higher magnesium intake or status tends to be associated with fewer sleep complaints, although the evidence is largely observational. [21] Consistent with this, a cohort from the Jiangsu Nutrition Study reported that higher dietary magnesium intake was linked to a lower prevalence of several sleep disorder

symptoms over five years. [22] Mechanistic reviews integrate these findings with experimental data, proposing that magnesium may influence circadian regulation, melatonin secretion and sleep architecture via its effects on glutamatergic and GABAergic signalling. [5]

Although several reviews have addressed magnesium in depression, anxiety or sleep separately, or in combination with other micronutrients, [1–4,14,17,18,21] there remains a need for an up-to-date, clinically oriented synthesis focusing specifically on the triad of depression, anxiety and sleep disturbances, and integrating mechanistic, observational and interventional findings. This is particularly relevant for primary care and mental health clinicians who increasingly encounter patients interested in dietary supplements as adjuncts to standard treatment. Therefore, the aim of this narrative review is to summarise current evidence on the role of magnesium in depression, anxiety and sleep disorders in adults, with an emphasis on human studies, and to discuss potential clinical implications and gaps in the literature that warrant further research. [1–4,7–11,17–22]

## **2. Methods**

This article is a narrative review of the literature on magnesium and mental health, with a focus on depression, anxiety and sleep disturbances in adults. The approach was chosen because of the heterogeneity of study designs, populations and outcomes in the available evidence, which makes a formal meta-analysis across all domains difficult. Existing systematic reviews and meta-analyses on specific outcomes (depression, anxiety, insomnia) were used as key sources of evidence and as starting points for identifying primary studies. [1–4,7,17,20,21]

### **Information sources and search strategy**

Electronic searches were conducted in PubMed/MEDLINE, Web of Science and Scopus from database inception to 31 October 2025. In addition, the archive of the Journal of Education, Health and Sport was searched separately to identify articles on magnesium and mental health published in this journal. [14,18] The search strategy combined terms related to exposure and outcomes using Boolean operators. For magnesium, we used the keywords “magnesium” and “Mg”. For mental health outcomes, we used “depression”, “depressive disorder”, “major depressive disorder”, “anxiety”, “anxiety disorders”, “stress”, “sleep”, “sleep disorders”, “insomnia” and “sleep quality”. Search terms were adapted to the syntax of each database. Reference lists of relevant systematic reviews and primary studies were screened manually to identify additional articles. [1–4,7,17,20,21]

## Eligibility criteria

We included original human studies and systematic reviews that met the following criteria:

**Population:** adults ( $\geq 18$  years) from community, primary care or clinical settings. Studies exclusively in children or adolescents, pregnant women or specific neurological disorders (e.g. epilepsy) were excluded.

**Exposure:** dietary magnesium intake, serum or plasma magnesium concentrations, or oral magnesium supplementation (any salt and dose).

**Outcomes:** depressive symptoms or diagnosed depressive disorders; anxiety symptoms or anxiety disorders; sleep disturbances, insomnia or validated sleep quality measures.

**Study design:** randomized or quasi-randomized controlled trials, prospective cohort studies, case-control studies, cross-sectional studies, and systematic reviews or meta-analyses. [1–4,7–11,17,19–22]

Because one aim of the review was to discuss biological plausibility, we also included selected experimental and mechanistic studies (animal models and mechanistic reviews) that specifically addressed magnesium and pathways relevant to depression, anxiety or sleep (e.g. HPA axis, NMDA and GABA signalling, circadian regulation). [2,4,5,6] Articles were restricted to English or Polish. Conference abstracts without full text, narrative reviews without clear linkage to primary data (other than [14,18]), editorials and case reports were not used as primary evidence for conclusions, although they were occasionally consulted for context. [16]

## Study selection

All records identified in the database searches were screened at title and abstract level. Articles clearly unrelated to magnesium or not addressing depression, anxiety or sleep outcomes were excluded. Full texts were obtained for all potentially relevant studies and assessed for eligibility according to the criteria above. When multiple publications reported overlapping data from the same cohort or trial, the most complete or recent report was prioritised. In case of uncertainty, inclusion was decided after discussion, with preference given to studies with clearer methodology, larger sample size or clinically relevant outcomes.

### Data extraction and synthesis

For each included study, we extracted information on study design, setting, sample size, population characteristics (age, sex, comorbidities), exposure definition (magnesium dose, formulation or intake category), comparison group, follow-up duration (for longitudinal studies) and main mental health outcomes. For randomized trials, we recorded details of randomisation, blinding, duration of supplementation, concomitant treatments and changes in validated symptom scales (e.g. depression, anxiety or sleep measures). [3,12,13,19,20] For observational studies, we noted the exposure categories, outcome definitions and whether analyses adjusted for key confounders such as age, sex, socioeconomic status, diet quality and comorbid conditions. [1,2,7–11,21,22]

Given the diversity of designs and outcomes, results were synthesized qualitatively. Studies were grouped into three main domains: (1) depression and mood disorders; (2) anxiety and stress-related symptoms; and (3) sleep disturbances and insomnia. Within each domain, we first summarised evidence from systematic reviews and meta-analyses, followed by key primary cohort, case-control and interventional studies. [1–4,7–10,12,13,17–22] Mechanistic and experimental data were integrated into a separate section to support interpretation of clinical findings. [2,4–6]

### Assessment of study quality and risk of bias

Formal scoring with standard risk-of-bias tools was not performed for all individual studies. Instead, we qualitatively assessed potential sources of bias, including selection bias, measurement of exposure and outcomes, control for confounding in observational studies, and randomisation, blinding and attrition in trials. Particular attention was paid to sample size, duration of follow-up or treatment, and the use of validated instruments for assessing depression, anxiety and sleep. [1–4,7,17,20,21] When interpreting the overall body of evidence, greater weight was given to systematic reviews and meta-analyses, randomized controlled trials, and larger prospective cohort studies, while findings from small or methodologically limited studies were considered exploratory.

### 3. Results

#### Magnesium and depression

##### Observational studies

Across observational studies, lower magnesium status was generally associated with a higher burden of depressive symptoms. In a systematic review of cross-sectional and cohort data, Derom et al. reported that most studies found an inverse relationship between dietary magnesium intake and depression, although effect sizes and adjustment strategies differed between cohorts. [1] A more recent GRADE-assessed dose–response meta-analysis by Hajhashemy et al. confirmed that lower habitual magnesium intake was associated with a higher risk of depression in adults. [7]

In a US primary care sample, Tarleton and Littenberg observed that adults with the lowest magnesium intakes had higher odds of clinically relevant depressive symptoms compared with those with higher intakes, after adjustment for sociodemographic and lifestyle factors. [8] In a Finnish 20-year follow-up study, Yary et al. found that low dietary magnesium intake was associated with an increased incidence of depression, particularly among men. [9] In another primary care population, Tarleton et al. reported that lower serum magnesium concentrations were associated with greater odds of depression, suggesting that biochemical hypomagnesaemia may be clinically relevant in some patients. [10]

Polish data are consistent with these findings. Styczeń et al. showed that serum magnesium concentrations differed between clinical states and healthy controls, with higher serum magnesium levels observed during acute episodes of unipolar depression compared with controls, and changes in concentration across the course of illness. [11] A narrative review from *Journal of Education, Health and Sport* summarised similar observational findings and pointed to both insufficient intake and increased losses as potential contributors to low magnesium status in depression. [14] Some studies included in these reviews reported null or attenuated associations after extensive adjustment, indicating variability in results across populations and designs. [1,2,7–11,14]

##### Intervention studies and meta-analyses

Randomised and quasi-randomised trials provide direct information on the effect of magnesium supplementation on depressive symptoms. In a randomized, equivalent trial in elderly patients with type 2 diabetes and depression, Barragán-Rodríguez et al. compared oral magnesium chloride (450 mg/day) with imipramine over 12 weeks and found similar reductions



in depressive symptom scores in both groups, with good tolerability in the magnesium arm. [12] In a double-blind, placebo-controlled trial in depressed adults with documented magnesium deficiency, Rajizadeh et al. reported that magnesium oxide (500 mg/day) significantly improved depressive symptom scores relative to placebo. [13]

Moabedi et al. pooled randomized clinical trials of magnesium supplementation in depressive disorders and observed a statistically significant reduction in depressive symptoms compared with control conditions, with effect sizes in the small-to-moderate range. [3] In a systematic review and meta-analysis of mood disorders, Phelan et al. found that magnesium supplementation was associated with a decline in depressive symptoms in uncontrolled studies but not in placebo-controlled trials, and concluded that the overall evidence for a role of magnesium in mood disorders was limited. [15] A review in *Journal of Education, Health and Sport* summarised these trials and noted that they typically involved modest sample sizes, short treatment duration and varied dosing regimens. [14]

Case series and uncontrolled reports, such as the *Med Hypotheses* paper by Eby and Eby describing rapid improvement of major depression with high-dose magnesium, describe favourable outcomes but lack control groups and formal randomisation. [16] Taken together, interventional studies and meta-analyses consistently report reductions in depressive symptom scores with magnesium supplementation in several adult populations, most often in individuals with low baseline intake or biochemical deficiency. [3,12–16]

### Magnesium, anxiety and stress-related symptoms

The number of studies focusing on anxiety and stress-related outcomes is smaller than for depression, and interventions are more heterogeneous. In a systematic review of trials evaluating magnesium supplementation (alone or in combination) in anxiety and stress, Boyle et al. identified studies in individuals with mild to moderate anxiety, premenstrual syndrome, occupational stress and other stress-related complaints. [17] Many of these trials reported reductions in subjective anxiety or perceived stress with magnesium-containing interventions, but formulations (magnesium alone vs combinations with vitamin B6 or other nutrients), populations and outcome measures differed substantially. [17]

In a narrative review in *Journal of Education, Health and Sport*, Rukat et al. summarised clinical and experimental data on magnesium and anxiety and reported that low magnesium status was frequently associated with increased anxiety symptoms, while supplementation appeared to alleviate symptoms in some groups, particularly in individuals with documented deficiency. [18] In a broader review of magnesium in mental disorders, Botturi et al. also

described studies suggesting anxiolytic effects of magnesium in specific clinical contexts, although they judged the overall strength of evidence to be low to moderate. [2]

Preclinical and mechanistic data complement these clinical observations. Wang et al. detailed how magnesium interacts with NMDA and GABA receptors and modulates glutamatergic transmission and stress-response pathways that are relevant to anxiety. [4] In an animal model, Sartori et al. demonstrated that dietary magnesium deficiency induced pronounced anxiety-like behaviour and dysregulation of HPA-axis activity in rodents, which could be modified by pharmacological treatment. [6] Overall, the available literature reports associations between low magnesium status and anxiety symptoms and describes symptom reductions in several small supplementation trials, alongside experimental evidence for biologically plausible mechanisms. [2,4,6,17,18]

#### Magnesium and sleep disturbances

Several studies have examined magnesium in relation to sleep quality and insomnia. In a randomized, double-blind, placebo-controlled trial in older adults with primary insomnia, Abbasi et al. administered 500 mg/day of magnesium for eight weeks and observed improvements in sleep onset latency, total sleep time, sleep efficiency and subjective sleep quality compared with placebo. [19] Adverse events were infrequent and mild. In a systematic review and meta-analysis of oral magnesium supplementation in older adults with insomnia, Mah and Pitre concluded that magnesium was associated with modest improvements in insomnia symptoms and sleep efficiency, based on a small number of trials with relatively few participants. [20]

Arab et al. conducted a systematic review of magnesium and sleep health including both interventional and observational data. They found that higher dietary magnesium intake or better magnesium status was generally associated with fewer sleep complaints and better self-reported sleep quality, although outcome definitions and instruments varied. [21] In the Jiangsu Nutrition Study, a cohort of Chinese adults, higher baseline magnesium intake was associated with a lower prevalence of several sleep disorder symptoms at five-year follow-up, after adjustment for sociodemographic and lifestyle factors. [22]

Mechanistic reviews provide potential explanations for these findings. He et al. summarised experimental and clinical evidence indicating that magnesium can influence circadian rhythms, melatonin secretion and the balance between excitatory and inhibitory neurotransmission via NMDA and GABA pathways, with potential effects on sleep architecture. [5] Botturi et al. and Wang et al. described overlapping pathways, including modulation of

HPA-axis activity and inflammatory processes, that may link magnesium with both mood and sleep regulation. [2,4] Collectively, the available studies report improvements in selected sleep parameters in older adults with insomnia receiving magnesium supplementation and fewer sleep-related symptoms in observational cohorts with higher magnesium intake or status. [2,4,19–22]

#### **4. Discussion**

This narrative review summarises current evidence on the relationship between magnesium and three closely related domains of mental health: depression, anxiety and sleep disturbances. Overall, the findings suggest that suboptimal magnesium status is associated with a higher burden of depressive symptoms, anxiety and sleep problems, and that magnesium supplementation can produce modest symptom improvements in selected adult populations. However, the strength and consistency of the evidence differ between outcomes, and important methodological limitations remain.

##### **Magnesium and depression**

The most coherent body of evidence concerns depression. Observational studies from diverse settings, including large community and primary care samples, consistently report inverse associations between dietary magnesium intake or serum magnesium concentrations and depressive symptoms or incident depression. [1,2,7–11] The dose–response meta-analysis by Hajhashemy et al. provides quantitative support for these findings, indicating that lower habitual magnesium intake is associated with a higher risk of depression in adults. [7] These data are strengthened by prospective designs in which low magnesium intake preceded the onset of depressive symptoms, as in the 20-year Finnish cohort reported by Yary et al. [9] Nevertheless, observational studies remain vulnerable to residual confounding by overall diet quality, socioeconomic status, comorbidities and health behaviours. [1,2,7–10]

Randomised and quasi-randomised trials offer more direct evidence but are fewer in number and generally small. Trials in elderly patients with type 2 diabetes and depression, and in adults with confirmed magnesium deficiency, show clinically meaningful reductions in depressive symptom scores following oral magnesium supplementation compared with active antidepressant treatment or placebo. [12,13] Meta-analyses pooling these and similar trials generally report statistically significant antidepressant effects with small-to-moderate effect sizes, although some analyses – including that by Phelan et al. – have found little evidence for a clear benefit in placebo-controlled studies. These effects appear more pronounced in

individuals with low baseline magnesium intake or biochemical deficiency and in those with somatic comorbidities, suggesting that magnesium may be particularly relevant as an adjunctive strategy in nutritionally or medically vulnerable groups. [3,14,15]

At the same time, most trials have modest sample sizes, short duration (often  $\leq 12$  weeks) and heterogeneous dosing regimens and formulations, limiting the ability to define optimal doses and treatment length. [3,12–15] Diagnostic criteria for depressive disorders and the use of validated outcome measures also vary. Case series and uncontrolled reports, such as the high-dose magnesium treatment described by Eby and Eby, are hypothesis-generating but cannot substitute for controlled trials. [16] Taken together, the evidence supports a contributory role of magnesium in depressive symptomatology and suggests potential therapeutic benefit, particularly as an adjunct in deficiency states, but does not establish magnesium as a stand-alone treatment for major depressive disorder.

#### Magnesium, anxiety and stress-related symptoms

In contrast to depression, the literature on anxiety and stress-related outcomes is more fragmentary. Boyle et al. identified several small trials in populations with premenstrual syndrome, occupational stress and subclinical anxiety that reported reductions in subjective anxiety or perceived stress with magnesium-containing interventions, often in combination with vitamin B6 or other nutrients. [17] Rukat et al., reviewing clinical and experimental data, concluded that low magnesium status is frequently observed in individuals with anxiety symptoms and that supplementation may be helpful in deficiency states, but emphasised the scarcity of robust randomised controlled trials in patients with defined anxiety disorders. [18] Botturi et al. similarly judged the overall strength of evidence for magnesium in anxiety to be low to moderate. [2]

These clinical observations are supported by preclinical and mechanistic work. Experimental models demonstrate that dietary magnesium deficiency induces anxiety-like behaviour and dysregulation of HPA-axis activity in rodents. [6] Reviews of molecular mechanisms describe how magnesium modulates NMDA receptor function, enhances GABAergic inhibition and influences stress-responsive neurobiological pathways. [2,4] Such findings lend biological plausibility to the hypothesis that low magnesium status can increase vulnerability to anxiety and that supplementation might mitigate symptoms in susceptible individuals. [2,4,6,17,18] However, the current clinical evidence base is insufficient to define the magnitude of any anxiolytic effect or to identify specific anxiety disorders that might benefit from magnesium supplementation.

## Magnesium and sleep disturbances

The relationship between magnesium and sleep has been investigated in both interventional and observational studies, with a particular focus on older adults and insomnia. The randomised, double-blind trial by Abbasi et al. showed that 8 weeks of magnesium supplementation in older individuals with primary insomnia improved sleep onset latency, total sleep time, sleep efficiency and subjective sleep quality compared with placebo. [19] A meta-analysis by Mah and Pitre, focusing on oral magnesium in older adults with insomnia, found modest improvements in insomnia symptoms and sleep efficiency, although the conclusions were based on a small number of trials with limited sample sizes. [20]

Beyond clinical trials, Arab et al. reported in their systematic review that higher dietary magnesium intake or better magnesium status was generally associated with fewer sleep complaints and better subjective sleep quality, despite considerable heterogeneity in outcomes and instruments. [21] The Jiangsu Nutrition Study provides longitudinal evidence that higher baseline magnesium intake is associated with a lower prevalence of sleep disorder symptoms at five-year follow-up. [22] Mechanistic reviews suggest that magnesium may affect circadian rhythms, melatonin secretion and the balance between excitatory and inhibitory neurotransmission via NMDA and GABA pathways, thereby influencing sleep architecture and resilience to sleep disruption. [2,4,5] Overall, the available data suggest that magnesium supplementation can improve certain sleep parameters in older adults with insomnia and that higher magnesium intake is associated with fewer sleep-related symptoms in observational cohorts, while evidence in younger adults and in patients with comorbid psychiatric disorders remains scarce. [19–22]

## Biological plausibility

The converging evidence across mood, anxiety and sleep domains is underpinned by a coherent mechanistic framework. Magnesium acts as a physiological blocker of the NMDA receptor channel, modulating glutamatergic transmission and reducing excitotoxicity, and enhances GABAergic inhibition. [2,4,5] It is also involved in monoamine synthesis, regulation of HPA-axis activity and modulation of inflammatory pathways, all of which have been implicated in the pathophysiology of depression, anxiety and sleep disturbances. [2,4–6] Experimental models demonstrate that magnesium deficiency can precipitate behavioural and neuroendocrine changes compatible with anxiety- and depressive-like states, which can be modified by pharmacological interventions. [6] These mechanistic insights support the

plausibility of the associations observed in human studies and provide a rationale for considering magnesium status when evaluating patients with mood, anxiety or sleep symptoms.

### Clinical implications

From a clinical perspective, current evidence supports several pragmatic considerations. First, inadequate magnesium intake or low serum magnesium is relatively common, particularly in older adults, individuals with poor diet quality, gastrointestinal disease, diabetes or medication-related losses (e.g. diuretics). [2,4,14] In such patients presenting with depression, anxiety or insomnia, assessing dietary intake and, where appropriate, serum magnesium may be reasonable. [2,4,11,14,22] Second, randomised trials and meta-analyses suggest that magnesium supplementation can be considered as an adjunctive strategy in adults with depressive symptoms, especially when there is evidence of low intake or deficiency, and in older adults with insomnia, while recognising that effect sizes are typically modest and some analyses have reported limited benefits. [3,12–15,19,20] Third, clinicians should be cautious not to position magnesium as a replacement for evidence-based pharmacological or psychotherapeutic treatments in major depressive or anxiety disorders, but rather as a potentially useful component of a broader multimodal approach.

Dosing regimens in the literature range widely, with elemental magnesium doses commonly between 200 and 500 mg/day, administered as magnesium oxide, citrate, chloride or other salts, typically over 4–12 weeks. [3,12–15,19,20] Renal function and potential drug interactions must be considered, particularly in older adults and in those with chronic kidney disease, to avoid hypermagnesaemia. [2,4] As with other supplements, patients should be advised that benefits are likely to be modest and that high-dose, unsupervised use is not warranted.

### Limitations of the evidence and of this review

Several limitations of the underlying evidence should be acknowledged. Many observational studies are cross-sectional and cannot establish the direction of causality; depression or insomnia might themselves influence dietary patterns and magnesium status. [1,2,7–11,22] Even in prospective cohorts, residual confounding by overall dietary patterns, physical activity and socioeconomic factors is likely. [7,9,22] The assessment of dietary magnesium intake typically relies on food-frequency questionnaires or dietary recalls, which are subject to measurement error. [1,2]

Randomised trials are generally small, of short duration and heterogeneous with respect to formulations, doses, populations and outcome measures. [3,12–15,19,20] Many trials enrol highly selected participants, such as older adults with insomnia or patients with specific comorbidities, which may limit generalisability to broader clinical populations. Publication bias in favour of positive findings cannot be excluded, particularly for smaller studies and for outcomes such as anxiety and stress. [2–4,17,20,21]

This review also has limitations. It is a narrative rather than a systematic review, and although major databases and the JEHS archive were searched, and key systematic reviews and meta-analyses were used to identify primary studies, some relevant articles may have been missed. [1–4,7,17,20,21] Formal risk-of-bias assessment tools were not applied systematically to all included studies; instead, methodological quality was considered qualitatively. As a result, the weighting of individual studies in the synthesis is based on study design, sample size and apparent rigour rather than on formal scoring.

#### Future directions

Future research should address several gaps. Large, well-designed randomised controlled trials are needed to evaluate magnesium supplementation as an adjunctive treatment in clearly defined depressive and anxiety disorders, using standardised diagnostic criteria and validated outcome measures, and stratifying participants by baseline magnesium status. [3,7,14,15,17,18] Trials in younger adults and in patients with comorbid psychiatric and medical conditions would help clarify the generalisability of existing findings. Longer follow-up periods are required to determine the sustainability of effects and to monitor safety over time. [3,12–15,19,20]

Prospective cohort studies with repeated measures of dietary intake, serum magnesium and mental health outcomes could help disentangle temporal relationships and clarify whether changes in magnesium status track with changes in symptom burden. [7–11,21,22] Mechanistic studies in humans, integrating neuroimaging, endocrine and inflammatory markers, could further elucidate how magnesium supplementation influences brain function and stress-response systems. [2,4–6] Finally, research on combined nutritional interventions, including magnesium with other micronutrients or dietary patterns, may be informative given the complex interactions between nutrients and mental health.

In summary, current evidence indicates that low magnesium intake or status is associated with increased depressive symptoms, anxiety and sleep disturbances, and that magnesium supplementation can provide modest symptom improvements in some adult

populations, particularly those with low baseline intake or deficiency and in older adults with insomnia. [1–4,7–10,12,14,15,17–22] These findings, together with biologically plausible mechanisms, support the consideration of magnesium status in the assessment and management of patients with mood, anxiety and sleep complaints, while underlining the need for further high-quality clinical and prospective studies to better define its role within comprehensive, evidence-based strategies for mental health. [2–6,14,15,20]

## **5. Conclusions**

Current evidence indicates that low dietary magnesium intake or suboptimal magnesium status is generally associated with an increased burden of depressive symptoms, anxiety and sleep disturbances in adults, although not all studies are consistent. [1,2,7–11,19–22] Randomised trials and meta-analyses suggest that oral magnesium supplementation may lead to small-to-moderate improvements in depressive symptoms and modest benefits in insomnia, particularly in individuals with low baseline intake or biochemical deficiency and in older adults with primary insomnia, but the overall evidence is heterogeneous and some analyses have not found clear benefits over placebo. [3,12–15,19,20] Data for anxiety and stress-related symptoms are less consistent and are based mainly on small, heterogeneous studies, although preclinical and mechanistic work provides a plausible biological rationale for a role of magnesium in stress regulation and emotional processing. [2,4–6,17,18]

Magnesium should therefore be considered as a potential adjunctive option within a broader, evidence-based approach to the management of depression, anxiety and sleep disturbances, rather than as monotherapy for major depressive or anxiety disorders or chronic insomnia. Assessment of magnesium status may be particularly relevant in patients with risk factors for deficiency, such as older age, poor diet quality, gastrointestinal disease, diabetes or medication-related losses. [2,4,11,14,22] Further large, well-designed randomised controlled trials and prospective cohort studies are needed to clarify which patient groups benefit most from magnesium supplementation, to define optimal dosing and treatment duration, and to better understand its place among nutritional and pharmacological strategies for mental health. [3,7,14,15,17,20,21]

## **Disclosure and Declarations**

Authors contribution:

Conceptualization: Aleksandra Kowalczyk, Adrian Pączek

Methodology: Adrian Pączek, Wiktoria Staniszevska



Formal analysis: Julia Hofman, Aleksandra Kowalczyk, Ilona Bednarek

Investigation: Wiktoria Staniszevska, Julia Hofman, Sylwia Lach

Writing- Rough Preparation: Adrian Pączek, Aleksandra Kowalczyk

Writing- Review and Editing: Aleksandra Kowalczyk, Paweł Dyczek, Ilona Bednarek

Visualisation: Wiktoria Staniszevska, Paweł Dyczek, Sylwia Lach

All authors have read and agreed with the published version of the manuscript.

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