

Role of Micronutrient Deficiencies in Depression and Anxiety- A Narrative Review

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Abstract: Depression and anxiety are highly prevalent mental disorders with multifactorial etiologies that include biological, psychosocial, inflammatory, endocrine, and nutritional pathways. An increasing amount of original human research indicates that, especially in vulnerable populations, shortages or insufficient consumption of specific micronutrients may contribute to the onset, intensity, or persistence of depression and anxious symptoms. The original research on vitamin D, zinc, magnesium, iron/ferritin, folate, vitamin B12, and general micronutrient sufficiency in connection to anxiety and depression is summarized in this narrative review. poor ferritin, altered zinc status, poor vitamin D status, and insufficient consumption of certain micronutrients are all linked to a higher burden of depressive symptoms, according to observational studies. In certain groups, reduced ferritin may also be correlated with anxiety severity. While the evidence for magnesium, folate, and vitamin B12 is still more inconsistent and frequently indirect in the examined sources, it is strongest for zinc as an adjuvant to antidepressant medication in serious depression and for vitamin D in high-risk older persons with prediabetes. Although the existing data does not support oversimplifying depression and anxiety as solely nutritional diseases, it does encourage micronutrient assessment as part of a more comprehensive biopsychosocial examination of patients with these disorders. Future research should concentrate on clinically significant psychiatric consequences, standardized biomarkers, and populations defined by deficiencies.

Keywords: Depression, Anxiety, Micronutrients, Vitamin D, Zinc, Magnesium, Ferritin, Iron, Folate, Vitamin B12.

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I. INTRODUCTION

Among the most prevalent mental health illnesses are anxiety and depression, which are increasingly recognized as being impacted by interrelated neurochemical, inflammatory, metabolic, and environmental factors. Because a number of micronutrients are involved in neurotransmitter synthesis, methylation, antioxidant defense, mitochondrial energy metabolism, myelination, and immunological modulation, nutrition has emerged as one potentially modifiable component. The biochemical disruptions that emerge from these micronutrient deficiencies may potentially exacerbate mood and anxiety symptoms or lower stress tolerance. (Rajkumar, 2026; Zaromytidou et al., 2022)

It is improbable that the link is universal or linear. Micronutrient status may be indirectly altered by depression and anxiety, which can also deteriorate diet quality, decrease outdoor exercise, disrupt sleep, change gastrointestinal function, and increase inflammatory burden. Because of this, original human research is particularly crucial for separating association from therapeutic benefit. Therefore, this narrative review analyses the function of micronutrient deficiencies in depression and anxiety without depending on fake or

unreliable references and concentrates on original papers found through published sources available in PubMed/PMC-linked records.

II. RATIONALE AND SCOPE

This review addresses seven domains supported by original or primary human studies retrieved for this paper: vitamin D, zinc, magnesium, iron/ferritin, folate, vitamin B12, and overall micronutrient adequacy. The aim is not to claim causality for every nutrient, but to critically summarize what primary studies suggest about risk, symptom severity, and response to supplementation. (Ranjbar et al., 2013a; Zaromytidou et al., 2022)

Because the available research varies in terms of population, biomarker definition, mental phenotype, and study methodology, a narrative approach is necessary. Clinically diagnosed major depression is the subject of some studies; anxiety features or depressive symptom scales in vulnerable but non-psychiatric populations are the subject of others; and some studies concentrate on certain age groups, including older adults or adolescents. (Abdelmoneam et al., 2024a; Sánchez-Villegas et al., 2018).

III. BIOLOGICAL BASIS

Through a number of convergent processes, micronutrients may affect anxiety and depression. Iron is necessary for oxygen transport, myelination, and monoamine synthesis; zinc influences glutamatergic transmission, neuroplasticity, and brain-derived neurotrophic factor; magnesium is involved in NMDA receptor regulation and stress responses; folate and vitamin B12 are essential for one-carbon metabolism and methylation reactions relevant to neurotransmitter function; and vitamin D is involved in neuroprotection, inflammatory modulation, and signaling pathways related to mood regulation. (Rajkumar, 2026; Ranjbar et al., 2013a; Zaromytidou et al., 2022)

These mechanisms support biological plausibility, but plausibility alone is insufficient. The stronger question is whether human studies show that deficiency states or inadequate intake correspond to psychiatric symptoms and whether correction of deficiency improves outcomes. The following sections examine that evidence nutrient by nutrient.

➤ *Vitamin D*

Vitamin D offers some of the most convincing original interventional data for both anxiety and depression among the micronutrients examined. Weekly vitamin D3 supplementation was linked to significantly decreased trait anxiety, state anxiety, and PHQ-9 depression scores at 6 and 12 months in an open-label, randomized controlled research of older persons with prediabetes when compared to controls who just received lifestyle advice. Restoring low vitamin D status may be clinically significant in an elderly population at high risk, as baseline deficiency was prevalent in both groups and the improvement continued over follow-up. (Zaromytidou et al., 2022) This study is significant since it used validated scales to measure anxiety and depression outcomes over a considerable amount of time. However, generalization to all patients with anxiety or depression disorders is limited due to its open-label design, unique metabolic-risk group, and exclusion of people with severe psychiatric diseases. Nevertheless, the experiment offers firsthand, first-hand proof that improving low vitamin D status can lessen the burden of symptoms in at least one susceptible population.

➤ *Zinc*

Zinc has been linked to depressive disorders on several occasions, especially when used as an adjuvant rather than a stand-alone treatment for serious depression. When compared to a placebo plus antidepressants, daily zinc supplementation for 12 weeks significantly reduced Beck Depression Inventory ratings in a randomized clinical trial of patients with serious depression taking SSRIs. By week 12, there was a noticeable improvement. This lends credence to the theory that chronic depression symptoms or an inadequate response to treatment may be caused by a zinc deficit or deficient zinc biology. Because the researchers also tracked dietary consumption and tried to adjust for nutritional confounding, the zinc experiment is noteworthy. However, the study cannot determine if zinc deficiency has a similar therapeutic impact in anxiety disorders because the sample was small and the

focus was on depression rather than anxiety. According to the available data, zinc seems to be more closely associated with improvements in depressive symptoms than with outcomes related to anxiety. (Ranjbar et al., 2013b)

➤ *Magnesium*

The examined literature points to a possible link between anxiety and magnesium, but the direct primary evidence is still weaker than that of zinc or vitamin D. A PubMed-indexed comprehensive review found that while the data is suggestive of a positive impact of magnesium supplementation on subjective anxiety in anxiety-vulnerable groups, it also highlighted the need for stronger randomized trials and low methodological quality. This is not a primary trial in and of itself, but it represents the current status of original human investigations in this field and indicates that the signal is encouraging but not conclusive. (Boyle et al., 2017)

Further evidence is provided by an adolescent study that examined the relationship between depression and ferritin and magnesium shortage; lower ferritin was clearly linked to depression, and magnesium was included in the nutrient profile that was being examined. However, judgments about magnesium should be cautious because the materials gathered here do not offer a robust stand-alone original magnesium-deficiency experiment in clinically diagnosed anxiety or depression. Stronger deficiency-targeted psychiatric research is still required, even if magnesium may be important. (Abdelmoneam et al., 2024b; Boyle et al., 2017)

➤ *Iron and Ferritin*

Because iron is required for brain energy metabolism, myelination, and the production of neurotransmitters like dopamine and serotonin, iron shortage is a scientifically plausible cause of anxiety and depression symptoms. Serum ferritin was shown to be adversely linked with the intensity of anxiety and depression in certain patient groups, according to a Frontiers review of primary data. This finding supports a symptom gradient with worsening iron depletion. Despite being a review, that source explicitly summarizes the results of primary studies and emphasizes ferritin as sometimes a more therapeutically useful test than haemoglobin alone. (Rajkumar, 2026)

Adolescent case-control research that found that depressed students had considerably lower ferritin levels than healthy students and that the likelihood of depression increased when ferritin changed from normal to low provides further direct primary evidence. This suggests that low iron levels may be linked to depressive symptoms before overt severe anemia becomes clinically apparent, although it does not establish causation. Clinically, these results support the need to pay attention to ferritin and iron insufficiency in patients who exhibit symptoms of fatigue, depression, difficulty concentrating, and anxiety, particularly in women and adolescents who are at risk of iron deficiency. (Abdelmoneam et al., 2024a; Rajkumar, 2026)

➤ *Folate and Vitamin B12*

Folate and vitamin B12 are long-standing contenders in the literature on nutritional psychiatry because they are essential to methylation processes and homocysteine metabolism. Folate and vitamin B12 deficiencies have been linked to an increased incidence of depression, according to the publications included for this study; however, supplementation studies have yielded conflicting findings. Folate and vitamin B12 supplementation did not significantly lessen the intensity of depression symptoms in the short term, according to a PubMed-indexed systematic review and meta-analysis, while they may be helpful in longer-term management. These results point to a crucial contrast between supplementing as an instantaneous antidepressant approach and deficiency as a risk signal. Although current evidence from the studies retrieved here does not support overstating their short-term efficacy for depression or anxiety symptom remission, folate and vitamin B12 deficiencies should still be identified and corrected in practice for medical reasons and because they may contribute to neuropsychiatric symptoms. Additionally, there is little primary evidence in the current source collection that expressly links anxiety effects to B12 or folate deficiencies.(Almeida et al., 2015)

➤ *Overall Micronutrient Adequacy*

Sizable prospective cohort research examined whether insufficient consumption of many micronutrients predicted depression in the future. Participants with inadequate intake of four or more micronutrients had a significantly greater risk of incident depression after a median follow-up of 8.5 years compared to those with more appropriate intake; however, the association diminished when repeated dietary evaluations were taken into account. This study is particularly important since it goes beyond cross-sectional connection and implies that over time, cumulative nutritional deficiencies may somewhat increase the risk of depression. Additionally instructive is the attenuation following updated repeated assessments. It suggests that rather than being a constant, independent factor in every person, micronutrient deficiency may be a component of a larger dietary and lifestyle pattern. Nevertheless, the study validates the clinical hypothesis that, in many real-world contexts, cumulative dietary deficiencies may be more significantly linked to depression risk than any one separate vitamin. (Sánchez-Villegas et al., 2018)

Table 1 Comparison of Main Studies

Nutrient/domain	Study type	Population	Main mental health outcome	Key finding
Vitamin D	Randomized controlled study	Older adults with prediabetes	Anxiety and depression	Weekly vitamin D3 reduced STAI-T, STAI-S, and PHQ-9 scores at 6 and 12 months compared with controls
Zinc	Randomized clinical trial	Adults with major depression on SSRIs	Depression	Zinc adjunct therapy significantly lowered Beck depression scores versus placebo by week 12
Iron/ferritin	Case-control study	Adolescent students	Depression	Depressed students had lower ferritin, and low ferritin increased odds of depression
Multiple micronutrients	Prospective cohort	13,983 adults	Incident depression	Inadequate intake of four or more micronutrients was associated with higher depression risk in primary analysis
Folate/B12	Systematic review of randomized trials	At-risk or depressed adults	Depression	Short-term symptom reduction was not clearly demonstrated, though long-term benefit remained possible
Magnesium	Review of human supplementation studies	Anxiety-vulnerable samples	Subjective anxiety	Existing evidence was suggestive, but study quality was poor and confirmation is needed

IV. CLINICAL IMPLICATIONS

A practical clinical approach is preferred above indiscriminate supplementing based on the available data. Micronutrient deficiencies may legitimately be assessed as part of routine evaluation for patients with depression or anxiety who also have fatigue, poor diet quality, restricted eating patterns, gastrointestinal disorders, low sun exposure, menstrual blood loss, malabsorption risk, advanced age, or chronic medical illness.(Rajkumar, 2026; Sánchez-Villegas et al., 2018; Zaromytidou et al., 2022) This is particularly important for low ferritin/iron storage, zinc deficiency, and vitamin D deficiency, where initial research indicates

significant correlations or supplemental benefits.(Abdelmoneam et al., 2024a; Ranjbar et al., 2013a)

On the other hand, personalized and deficiency-informed supplementation is excellent. Presenting vitamin or mineral therapy as an alternative to evidence-based psychiatric care, psychotherapy, physical activity, sleep management, or treatment of medical comorbidities is not supported by the evaluated evidence. Rather, it is important to consider micronutrient correction as a potentially beneficial part of integrated care. (Almeida et al., 2015; Sánchez-Villegas et al., 2018; Zaromytidou et al., 2022)

V. LIMITATIONS OF THE EVIDENCE

Several limitations recur across the available literature. First, many studies are small, observational, or confined to special populations such as elderly adults with prediabetes or adolescent students, reducing external validity. Second, psychiatric outcomes vary from symptom scales to clinical diagnoses, making comparison difficult. Third, biomarker thresholds for deficiency differ across studies, and some nutrients may be measured by intake rather than blood concentrations, which are not equivalent constructs. Reverse causality is another major concern. Depression and anxiety may cause reduced appetite, poorer self-care, lower sunlight exposure, inactivity, and chronic inflammation, all of which can worsen micronutrient status without proving that deficiency came first. Therefore, even strong associations should be interpreted carefully unless supported by well-designed randomized correction trials.

VI. FUTURE DIRECTIONS

Original trials that include subjects with biochemically verified deficits and clinically significant symptoms of anxiety or depression should be given priority in future research. Standardized biomarker cutoffs, sufficiently powered sample sizes, blinded designs, and parallel evaluation of functional, inflammatory, and mental outcomes should all be used in such investigations. Additionally, multi-micronutrient models are required instead of only single-nutrient thinking. Future research should investigate whether combined correction of low vitamin D, ferritin, zinc, folate, or B12 offers greater advantages than standalone treatment in specific subgroups because nutrients interact biologically and deficiencies frequently cluster.

VII. CONCLUSION

The available original human evidence suggests that micronutrient deficiencies may play a contributory role in depression and anxiety, but the strength of evidence differs by nutrient. Vitamin D has notable interventional support for reducing anxiety and depressive symptoms in deficient or high-risk older adults, zinc shows adjunctive benefit in major depression, low ferritin appears associated with depressive burden and possibly anxiety severity, and broad micronutrient inadequacy may modestly increase future depression risk.

The most defensible clinical position is that micronutrient deficiencies should be screened for and corrected when present, especially in vulnerable populations, while continuing to manage depression and anxiety through comprehensive, evidence-based psychiatric and medical care.

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