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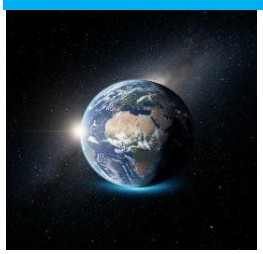

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Dietary Patterns and Supplementation in Depression and Anxiety: A Comprehensive Literature Review

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Abstract

Introduction and purpose: Depression and anxiety frequently co-occur and cause substantial functional burden. Nutrition - especially diet quality and supplementation - has been studied as a modifiable factor. This comprehensive literature review summarised evidence on dietary patterns and selected supplements in relation to depressive and anxiety symptoms.

Material and methods: A narrative synthesis was performed using 32 articles (systematic reviews/meta-analyses, randomised trials, observational and mechanistic papers). The scope covered diet quality and dietary patterns, whole-of-diet interventions, gut–brain axis concepts, and supplementation with omega-3 fatty acids, B vitamins, magnesium, vitamin D, zinc, and probiotics.

Results: Prospective evidence generally linked higher diet quality with lower depression risk. Whole-diet intervention trials showed mixed results, with several studies reporting improvements in depressive outcomes; anxiety outcomes were less consistent. Mechanistic literature supports plausibility through inflammation/oxidative pathways, serotonergic neurotransmission, and diet–microbiota interactions. Among supplements, evidence is most developed for omega-3 fatty acids (dose-response signals for anxiety) and magnesium (benefits in trials and meta-analysis), while findings for vitamin D, B vitamins, zinc and probiotics vary across studies.

Conclusions: Diet quality improvement and selected supplements may offer adjunctive benefits for some individuals, but effects are heterogeneous and should not be overgeneralised. Nutritional strategies should be integrated with evidence-based mental health care, with timely clinical assessment and standard treatments used when indicated.

Keywords: Depressive Disorder; Anxiety; Diet; Dietary Supplements; Omega-3 Fatty Acids; Magnesium

1. INTRODUCTION AND PURPOSE

Depression and anxiety disorders are frequently discussed as distinct diagnoses, but in clinical reality they often overlap and reinforce each other. Many patients experience a mixture of low mood, anhedonia, fatigue, sleep disruption, cognitive difficulties, and persistent worry or physiological tension, and the resulting burden is rarely confined to “mental” symptoms alone. Because of this complexity, interest has grown in modifiable factors that may support prevention, symptom reduction, and long-term resilience. Diet has become a particularly attractive candidate: it is a daily exposure, closely linked to metabolic and inflammatory health, and strongly shaped by routines that can either stabilize or destabilize wellbeing over time.

The literature provided for this review reflects a broader shift in nutritional psychiatry. Earlier discussions often focused on isolated nutrients, but more recent work increasingly emphasizes

diet quality and dietary patterns as a more realistic lens - capturing what people actually do rather than what they take in a capsule. In prospective evidence, healthier patterns and higher diet quality are generally associated with lower depression risk, although interpretation is cautious because bidirectionality and confounding remain major challenges in observational research [5,23].

It is also worth noting that supplement use is common in the general population, including among people with mental health conditions. A large population-based survey examined folic acid and vitamin supplement use among adults with diagnosed depression and anxiety and reported measurable differences in supplement-taking patterns compared with those without these conditions [29].

In parallel, mechanistic papers describe plausible biological routes connecting diet to depression and anxiety-related symptoms, including immune-inflammatory processes, oxidative balance, serotonergic neurotransmission, and gut–brain axis pathways influenced by diet–microbiota interactions [4,10,15,25]. Alongside whole-diet approaches, targeted supplementation remains widely used and studied; however, the evidence base is uneven, and effects appear to vary depending on baseline status, dose, population characteristics, and outcome domains [1,12,13,17,21,22,31,32].

Aim of the study: The aim of this comprehensive literature review is to integrate evidence from the provided full-text articles on diet quality and dietary patterns, whole-of-diet interventions, mechanistic frameworks, and selected supplements investigated in relation to depressive symptoms and anxiety outcomes, while maintaining a clinically responsible interpretation of what this evidence can and cannot support.

2. MATERIAL AND METHODS

This comprehensive literature review was based exclusively on the systematic reviews and meta-analyses, randomized controlled trials, observational studies, and narrative/scoping reviews addressing diet quality and dietary patterns, whole-of-diet interventions, relevant biological mechanisms (including inflammation, neurotransmission, and gut–brain axis concepts), and dietary supplementation (B vitamins, omega-3 fatty acids, magnesium, vitamin D, zinc, probiotics, and broader nutraceutical approaches) in relation to depression and/or anxiety outcomes. For each article, core descriptive details were extracted (study type,

population, exposure or intervention characteristics, outcome measures, and key findings). Because the included studies were heterogeneous, results were synthesized narratively and organized thematically in subsequent sections. No additional database search beyond the provided set was performed; accordingly, the review should be interpreted as a structured synthesis of a predefined evidence set rather than a de novo systematic review.

3. DIET QUALITY AND DIETARY PATTERNS IN DEPRESSION AND ANXIETY

3.1. Why "diet quality" matters more than single foods

A recurring message across the provided literature is that diet is rarely best understood as a single component acting in isolation. In real life, people do not eat "a nutrient"; they eat patterns, routines, and combinations of foods that accumulate into a broader dietary environment. This is exactly why many authors emphasize diet quality and dietary patterns rather than one "superfood" or one supplement as the primary unit of analysis. When diet quality improves, multiple exposures change at once - micronutrient density, fiber intake, fatty acid balance, and the overall proportion of minimally processed foods - making it biologically plausible that the downstream impact could be broader than what any single nutrient might achieve [4,23].

This pattern-based approach is particularly visible in prospective evidence. In a systematic review and dose–response meta-analysis of prospective studies, higher diet quality and healthier dietary patterns were generally associated with a lower risk of depression outcomes over time, although interpretation is necessarily cautious because observational evidence can never fully eliminate confounding and reverse causality [5]. A separate systematic review similarly reported an overall association between diet quality/dietary patterns and depression in adults, while also showing that results vary across cohorts, dietary assessment tools, and depression outcome definitions [23].

In practical terms, these findings support a clinically intuitive idea: dietary patterns may operate as a kind of "background exposure." They might not instantly transform symptoms, but they can shape the physiological and behavioral conditions under which mood symptoms emerge, persist, or improve. At the same time, these papers also encourage restraint in interpretation. Depression can reduce motivation, disrupt routines, and change appetite or food choices, so poorer diet quality may sometimes reflect the illness trajectory rather than drive it [5,23]. Importantly, the relationship between diet and mental health is also visible within clinical groups. In an observational analysis comparing adults with and without depressive and anxiety

disorders, diet quality was poorer in participants with current disorders, and the lowest diet quality was reported in those with comorbid depression and anxiety; disorder chronicity and severity were also linked with less favorable diet quality scores [6].

3.2. Mediterranean-type patterns and the broader "protective pattern" concept

Within the pattern-based literature, Mediterranean-style dietary patterns appear repeatedly as a reference point - less as a strict prescription and more as a recognizable cluster of features (greater emphasis on plant foods, overall nutrient density, and more favorable fat quality). The provided work discussing Mediterranean diet and depression places this pattern in the broader context of diet quality and depression-related outcomes, reinforcing how often this pattern emerges in epidemiological discussions [18]. Importantly, this does not mean that the Mediterranean diet is "the answer," but rather that it functions as a useful model for what "higher diet quality" tends to mean in many studies [5,18].

3.3. Anxiety: evidence is present, but the picture is less uniform

Compared with depression, diet–anxiety evidence is often harder to synthesize into one simple conclusion. Anxiety is not measured in a single uniform way: studies may focus on clinical anxiety disorders, anxiety symptoms, perceived stress, or mixed emotional distress. A scoping review mapping the field emphasizes that a meaningful literature exists, but it is heterogeneous in exposures, outcomes, and study contexts - making strong generalizations more difficult [3]. A mechanistic review focusing on diet-regulated anxiety similarly supports the idea that diet could plausibly influence anxiety-related states, but it also frames anxiety biology as multi-pathway and context-dependent, which aligns with why effects may vary across studies [7].

This matters for how we write about anxiety in a review: rather than forcing a single "diet reduces anxiety" narrative, it is more accurate to treat anxiety outcomes as potentially sensitive to baseline context (stress load, sleep disruption, comorbid depression, and physiological arousal). That framing remains consistent with the literature you provided and avoids overstating certainty where the evidence is mixed [3,7].

3.4. Special contexts: celiac disease and the reality of medically indicated diets

The relationship between diet and mental health becomes especially complex in conditions where diet is not merely a preference but a medical requirement. In adult celiac disease, a gluten-free diet is necessary, yet anxiety and depression remain relevant clinical issues. The study focusing on adults with celiac disease on a gluten-free diet highlights that mood and

anxiety symptoms are still present and clinically meaningful in this population [2]. This perspective is useful beyond celiac disease, because it reminds us that a dietary change can be simultaneously therapeutic, demanding, and psychologically burdensome - depending on nutritional adequacy, restriction-related stress, and daily-life constraints [2].

4. WHOLE-OF-DIET INTERVENTIONS: FROM ASSOCIATION TO ACTION

Observational data can suggest that diet quality and depressive outcomes travel together, but intervention trials ask the more clinically actionable question: if we help people change their diet, do symptoms improve? Whole-of-diet interventions are especially important because they mirror real eating behavior - patterns and routines - rather than a single nutrient added on top of an unchanged baseline diet [26].

In the systematic review of randomized controlled trials of whole-of-diet interventions, seventeen trials met eligibility criteria; all reported depression outcomes and ten reported anxiety outcomes. Compared with control conditions, almost half (47%) of the included studies observed significant effects on depression scores in favor of the dietary intervention, while the remaining studies reported null effects [26]. The review also identified practical features more commonly present in effective interventions: they tended to use a single delivery mode, employ a dietitian, and were less likely to recommend certain restrictive or "cardiac-style" recommendations (e.g., reducing red meat intake, selecting leaner meat products, or following a low-cholesterol diet) [26].

This is an unusually useful detail for a review article because it translates evidence into a realistic implementation message: success may depend not only on what the diet is "on paper," but also on how the intervention is delivered and whether it feels coherent and doable for participants. It also highlights a limitation emphasized by the authors: few trials specifically targeted individuals with clinical depression, which restricts how confidently results can be generalized to clinical psychiatric populations [26].

5. BIOLOGICAL MECHANISMS LINKING DIET TO DEPRESSION AND ANXIETY

5.1. Inflammation and oxidative balance as plausible bridges

Mechanistic work in the provided set makes a consistent point: depression (and anxiety-related distress) is not typically explained by a single pathway, and diet may influence several systems that are repeatedly discussed in relation to depressive symptomatology. The mechanistic review by Marx and colleagues outlines biologically plausible links between diet and depression through inflammation as well as oxidative and nitrosative stress, among other pathways [4]. Importantly, this literature does not require an "either/or" view in which diet is either the cause of depression or irrelevant. Instead, diet can be framed as one contributor that shapes vulnerability, symptom persistence, and potentially treatment responsiveness in certain contexts [4,8].

5.2. Gut–brain axis: diet–microbiota interactions as a mediator pathway

Another dominant mechanistic theme is the gut-brain axis. The review by Luna and Foster emphasizes that diet can shape microbiota composition and function and that microbiota-related pathways may influence behaviors relevant to anxiety and depression [15]. A psychiatry-focused review similarly highlights the emerging role of gut microbiota in mood and behavior and frames diet as a central upstream factor in this system [25]. When these are read together, a coherent interpretation emerges: dietary patterns may influence mental health partly through immune signaling, microbial metabolites, and gut-related pathways that interact with stress responsiveness and affective regulation [15,25].

5.3. Neurotransmission: serotonergic pathways in the nutritional context

Within the provided material, serotonergic neurotransmission is presented as another plausible interface between diet and depressive states. The review on diet and serotonergic neurotransmission discusses how dietary factors may influence serotonin-related systems relevant to depression [10]. Again, the most responsible interpretation is not reductionist ("food equals serotonin equals mood fixed"), but contextual: nutritional status and diet composition help shape the biochemical environment in which neurotransmission occurs [4,10].

6. DIETARY SUPPLEMENTATION IN DEPRESSION AND ANXIETY: WHAT THE EVIDENCE SHOWS

6.1. B vitamins: a clearer signal for stress than for depression or anxiety

The systematic review and meta-analysis of B vitamin supplementation is valuable because it separates outcomes rather than bundling everything into "mood." Across included trials, supplementation showed a significant benefit for stress, while the effect on depressive symptoms did not reach significance and there was no effect on anxiety [1].

Evidence from experimental supplementation also exists at the trial level. In a double-blind study, one month of high-dose vitamin B6 supplementation reduced self-reported anxiety and showed a trend toward reduced depression, while vitamin B12 showed only trend-level changes [16].

This is an important "expectation-setting" point for the paper: even for widely used supplements, measurable benefits may cluster in specific symptom domains (e.g., stress) rather than in depression or anxiety outcomes as such [1].

6.2. Omega-3 fatty acids: dose–response evidence for anxiety symptoms

Omega-3 supplementation has one of the most quantitatively detailed analyses in the collected data: a dose–response meta-analysis of randomized trials assessing anxiety symptoms. In 23 trials including 2189 participants, each additional 1 g/day of omega-3 supplementation was associated with a moderate decrease in anxiety symptoms (SMD −0.70, 95% CI −1.17 to −0.22; GRADE low) . The non-linear dose–response analysis suggested the greatest improvement at 2 g/day (SMD −0.93, 95% CI −1.85 to −0.01) and reported that doses below 2 g/day did not affect anxiety symptoms in the modeled relationship. The same meta-analysis reported no increase in adverse events (OR 1.20, 95% CI 0.89 to 1.61; GRADE moderate) [13].

These findings are clinically interesting for two reasons. First, they imply that dose may meaningfully influence whether an effect is detectable in anxiety outcomes. Second, they also force humility: the authors emphasize uncertainty and limitations (including substantial heterogeneity and low certainty), which means omega-3 should be framed as potentially helpful for some individuals rather than universally anxiolytic [13]. Trial-level evidence also exists in the provided set connecting omega-3 supplementation to anxiety-related outcomes and inflammatory measures in a high-stress population (medical students), which supports the broader plausibility of an effect in certain contexts [20]. Omega-3 has also been tested in major depression trials, illustrating sustained clinical interest in affective-disorder populations [14].

6.3. Magnesium: consistent signals across a randomized trial and meta-analysis

Magnesium is another supplement with comparatively strong support across the found data. In an open-label, randomized, crossover clinical trial in adults with mild-to-moderate depression

symptoms, magnesium chloride supplementation (248 mg elemental magnesium/day) was associated with meaningful improvements: PHQ-9 depression scores improved during magnesium treatment (−4.3 points, 95% CI −5.0 to −3.6) but not during control (−0.1, 95% CI −0.9 to +0.7), yielding a net benefit of −4.2 points. Anxiety outcomes measured by GAD-7 also improved during magnesium supplementation (−3.9 points, CI −4.7 to −3.1) while worsening during control (+0.8, CI +0.02 to +1.6), with a net benefit of −4.7 points (CI −6.0 to −3.3) [22]. This trial-level signal is reinforced by a systematic review and meta-analysis of randomized clinical trials in adults with depressive disorder, where magnesium supplementation significantly reduced depression scores (SMD −0.919, 95% CI −1.443 to −0.396) [17]. In addition, magnesium is discussed in relation to subjective anxiety outcomes in the provided literature, which aligns with the trial's finding that anxiety scores may shift alongside depressive symptoms in some settings [24]. Overall, magnesium emerges as a supplement where the evidence base - while still requiring careful interpretation - contains both controlled-trial signals and quantitative synthesis [17,22,24].

6.4. Vitamin D: mixed overall results, potentially stronger in clinically depressed subgroups

Vitamin D is represented by two meta-analyses and a trial-level paper in this review. In one meta-analysis, vitamin D supplementation had no overall effect on depressive symptoms (SMD −0.14, 95% CI −0.33 to 0.05), though heterogeneity was substantial; importantly, subgroup analysis suggested a moderate significant effect in participants with clinically significant depressive symptoms or depressive disorder (2 studies: SMD −0.60, 95% CI −1.19 to −0.01), while those without clinically significant depression showed a small, non-significant effect [31]. Another meta-analysis similarly found no significant reduction in depression after vitamin D supplementation overall, noting that many trials included individuals with low levels of depression and often sufficient baseline vitamin D, as well as variability in dosing and duration [32].

Taken together, these findings support a careful, non-sensational conclusion: vitamin D is biologically plausible and widely relevant, but the clinical signal is not uniform, and baseline characteristics appear to matter when interpreting supplementation outcomes [11,31,32].

6.5. Zinc and other supplement categories: plausible signals, but narrower evidence

Zinc supplementation is included through a randomized clinical trial in major depression in the sources, indicating that zinc has been investigated as an adjunctive nutritional strategy in

depressive disorders [12]. Broader supplement-focused reviews (including dietary and plant-based/nutritional supplements) reinforce that the evidence base is heterogeneous across compounds and populations, and that effects should not be assumed to generalize from one supplement class to another [9,19,27,28].

6.6. Probiotics: an evidence area aligned with the gut–brain model, still variable in practice

A review of probiotics in depressive and anxiety disorders reflects ongoing interest in microbiome-focused interventions and their potential relevance to mood and anxiety outcomes [21]. However, microbiome-related work across the provided set repeatedly implies that variability in strains, dose, duration, and outcome measurement makes "one-line conclusions" difficult. The most consistent message is that probiotics are conceptually aligned with gut–brain axis mechanisms, but the clinical literature remains developing and must be interpreted with attention to heterogeneity [15,21,25].

7. PRACTICAL INTERPRETATION AND CLINICAL INTEGRATION

Taking into account the analyzed literature as a whole, the most protective and clinically useful approach is the integrated approach. Diet quality and dietary patterns show consistent associations with depression risk in prospective evidence, and whole-of-diet intervention trials provide "proof-of-concept" that dietary change can improve depression outcomes in some contexts [5,23,26]. Mechanistic literature strengthens plausibility by outlining pathways through inflammation/oxidative balance, neurotransmission, and diet–microbiota interactions [4,10,15,25].

At the same time, supplementation evidence varies substantially by nutrient and by outcome domain. B vitamins show clearer effects for stress than for anxiety or depressive symptoms; omega-3 shows dose–response findings for anxiety symptoms but with important uncertainty; magnesium shows robust trial-level and meta-analytic signals; vitamin D findings are mixed overall but may be more favorable in clinically depressed subgroups [1,13,17,22,31,32]. These patterns point toward an interpretation that is both realistic and patient-centered: nutritional strategies can be meaningful, but they are not uniformly effective for everyone, and they are not interchangeable across supplement categories. [9,19,27,28]. R

reviews focused on vitamin B12 discuss a possible relationship between B12 status and depressive symptoms and suggest that early supplementation may be relevant in some contexts, including potential benefit alongside antidepressant treatment; however, the same review emphasizes that B12 should not be viewed as a stand-alone treatment for depression [30].

This is where a responsible clinical message becomes important - especially for a paper intended to be readable and useful. Nutritional strategies can increase agency, support physical comorbidity management, and contribute to long-term resilience. Yet none of the provided evidence base supports positioning diet or supplements as a universal standalone alternative in persistent, severe, or functionally impairing presentations. Instead, the literature fits best with a stepped model: use nutrition to strengthen foundations and support recovery, while ensuring that individuals who need timely clinical assessment and established treatments receive them without delay. This approach avoids extremes - neither dismissing nutrition nor overselling it - and is consistent with the overall tone of the included reviews and trials [8,26,27].

8. CONCLUSIONS

Overall, the evidence in this review points in a fairly consistent direction: people with better diet quality and healthier dietary patterns tend to report fewer depressive symptoms and a lower risk of depression, while findings for anxiety are more mixed and harder to generalize. When diet is tested as an intervention, whole-of-diet trials suggest that changing eating patterns can improve depressive outcomes in some settings, but results are not uniform and clearly depend on factors such as the population studied and how well people are able to follow the program.

The mechanistic literature helps explain why nutrition keeps coming up in this field, linking dietary exposures to pathways involving inflammation and oxidative balance, serotonergic neurotransmission, and gut–brain interactions. In supplementation studies, the most convincing signals in the provided articles relate to omega-3 fatty acids (including dose-related effects on anxiety symptoms) and magnesium (supported by both trials and meta-analyses). For vitamin D, B vitamins, zinc and probiotics, the picture is more variable, which suggests these approaches are best considered selectively rather than as “one-size-fits-all” solutions.

In practice, nutrition works best as supportive care: improving diet quality and, in some cases, using targeted supplements may help some people feel better and build longer-term resilience. At the same time, these strategies should not be used to delay clinical assessment or evidence-

based treatment when symptoms are persistent or severe - nutrition fits most safely as a complement within standard care, not a replacement.

Disclosure

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