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THE IMPACT OF DAILY HEALTHY FOODS AND REGULAR PHYSICAL ACTIVITY ON MANAGING DEPRESSION AND ANXIETY IN YOUNG ADULTS

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Abstract

Approximately 30% of young adults (ages 18-30 years) have depression and anxiety disorders, which is increasing since 2020. Barriers to adherence and access to conventional treatments. Daily healthy dietary patterns and regular physical activity are interventions that can help reduce depression symptom severity by 30–50% (Hedges' g = 0.40–0.70) when compared to control diets (Narrative review of randomized controlled trials (RCTs), longitudinal cohorts, meta-analyses, and mechanistic studies published 2000–2025) Similar effect sizes (g = 0.45–0.80) can be achieved with aerobic exercise of 150 minutes per week at moderate intensity in mild-to-moderate cases. The synergistic effect of combined diet and physical activity interventions range from g = 0.85–1.10, which is higher than the individual effects. Key mechanisms are decreased neuroinflammatory markers (IL-6, TNF-α), increased brain derived neurotrophic factor, gut brain axis modulation and normalization of cortisol rhythms of the HPA axis. Regular physical activity and healthy food are effective, low-risk adjunctive and/or standalone interventions to treat young adult depression and anxiety. Implementation in PHC and university is warranted.

INTRODUCTION

Depression and anxiety disorders are a growing public health problem in young adults (ages 18-30), with recent estimates that 25% of people would experience a clinically significant internalizing disorder by age 30.(Jiazhi, Caixia, Lamei, & Jian, 2025) This developmental period also relates to important psychosocial transitions (higher education, entering the workforce, developing an independent relationship, etc.) and an increased risk for the(Bourke et al., 2022) onset of mood and anxiety disorders. The World Mental Health Surveys (WMHS) indicate that the age of onset of major depression disorder is 25 years on average, and 24 years for generalized anxiety disorder, hence in the middle of the psychiatric burden curve in young adults.(Ströhle, 2009) Besides, (Hosker, Elkins, & Potter, 2019)these trends have been exacerbated with the COVID-19 pandemic, with meta-analyses showing a 25% increase in depression and 30% increase in anxiety symptoms in young adults around the world since the start of the pandemic.

Randomised controlled(W. Zhang, Huang, Hu, Yuan, & Chen, 2025) trials have shown good efficacy with first-line treatments, which are mostly selective serotonin(Francis et al., 2019) reuptake inhibitors (SSRIs) and cognitive behavioural

therapy (CBT); however, lack of effectiveness in actual practice is often(Fox, 1999) exacerbated by multiple, overlapping challenges. As a result, researchers and clinicians have focused more on lifestyle factors that can be modified to enhance patients' responsiveness to standard treatment, or as standalone treatments for mild-to-moderate presentations, in recent years.(Herbert, Meixner, Wiebking, & Gilg, 2020) Of the many lifestyle factors examined, two of them are of particular interest, as a result of the amount and quality of the evidence: daily healthy dietary patterns and regular physical activity.(Paluska & Schwenk, 2000) The neurobiological mechanisms that can link together between food and movement to mood regulation are no longer the subjects of speculation and have become(Bailey, Hetrick, Rosenbaum, Purcell, & Parker, 2018) solid and widely accepted scientific grounds at molecular, cellular, and systems levels. The ability of dietary components to directly modulate peripheral cytokine production and gut microbiota composition points to the ability of these components to directly modulate chronic low-grade inflammation, a core pathophysiological feature of MDD. At the same time, exercise increases the expression of brain-derived neurotrophic factor (BDNF), a protein necessary for the neurogenesis of the hippocampus and for the plasticity of the hippocampus' synapses, and decreases the levels of neurotoxic metabolites from the kynurenine pathway that build up during stress and lack of exercise.

This includes weight gain,(M. N. Hossain, Lee, Choi, Kwak, & Kim, 2024) sexual dysfunction, emotional blunting and early heightened risk of suicide. Although psychotherapy is a favored treatment for many patients, there are several drawbacks to the treatment, such as waiting lists, cost, and limited availability of practitioners who have been trained in evidence-based treatments.(McCloughen, Foster, Kerley, Delgado, & Turnell, 2016) Furthermore, a significant number of young adults (30%-40%) either do not achieve remission after a proper trial of monotherapy, or relapse after stopping treatment, which has led to the investigation of adjunctive and alternative treatments.

In spite(Barbosa et al., 2025) of being the most physically able age group, young adults' physical activity actually drops the most after adolescence. Moderate-to-vigorous physical activity also declines by about 40% from ages 18 to 25, a period when the amount of time spent in high school athletics or physically active commuting is replaced by more sedentary activities at the university or work station. (Calderón-Asenjo et al., 2022)More than half of youth do not attain the recommended level of moderate physical activity, and this lack of physical activity is associated with poorer mood in prospective follow-up. Likewise, the shift to independent living tends to be(Mawer et al., 2022) accompanied by reductions in the quality of diet, including greater consumption of ultra-processed foods, sugary drinks, less frequent meals, and less consumption of vegetables, fruits and fatty fish, all of which have been shown to be prospectively associated with higher scores for depressive symptoms.

The psychiatric disorders that raise(Lhoeste-Charris et al., 2024) in incidence during the YAP as well as the increased sensitivity of the developing brain to environmental factors are what make the YAP different from other life stages.(Abou Elmagd, 2016) Prefrontal cortical circuits that regulate emotion and top-down control over the amygdala continue to myelinate and refine through the late 20s making this a population that's vulnerable to nutritional deficits and the protective influence of exercise-induced neuroplasticity.(H. Wang, He, Tang, Tang, & Yang, 2024) Additionally, the lifestyle habits developed during this decade have a tendency of following into midlife, so a successful intervention now could have decades of cumulative benefit for mental health. More than 50 RCTs and several large longitudinal cohorts have shown that Mediterranean diet patterns and moderate-intensity aerobic exercise, stand-alone, are effective at reducing depression symptom severity by effect sizes that are similar to those of psychotherapy in mild-to-moderate depression cases. Importantly, both interventions seem to have synergistic effects; that is, the effects of diet and physical activity seem to be somewhat independent of each other.

The overall purpose of this review is thus to provide a synthesis of the evidence base on the effects of daily healthy diet and(Martinez-Avila et al., 2020) physical activity on depression and anxiety in young adults. Biological plausibility will be explored through inflammatory and neuroplastic, neuroendocrine and monoaminergic pathways. We will then talk about the best diet patterns and specific nutrients followed by detailed analysis of the type, dose and frequency of exercise. Synergistic effects of combined interventions will be discussed in a dedicated section. We will then present the pooled effect sizes from meta-analyses and comparisons with standard care. Practical implementation strategies and remaining gaps will be discussed, and end with recommendations for clinicians, for policy makers, and for young adults themselves. (Parvin, Etienne, & Wagener, 2025)When possible, we cite data from studies of participants aged 18 to 30; when these data are unavailable, we cite more general adult data from the adult literature. Figure 1 shows mechanistic diagram illustrates parallel and converging pathways from two lifestyle interventions (left side: Diet; right side: Physical Activity) to clinical outcomes (bottom: Reduced Depression and Anxiety). Diet acts through three sub-pathways:

(a) Gut microbiota modulation: increased fiber and polyphenols

promote *Lactobacillus* and *Bifidobacterium* species, producing short-chain fatty acids (SCFAs) such as butyrate, which strengthen gut barrier integrity and reduce translocation of lipopolysaccharides (LPS); (b) Reduced systemic inflammation: lower circulating IL-6, TNF-α, and CRP; (c) Enhanced tryptophan availability: displacement of kynurenine pathway toward serotonin synthesis. Physical activity acts through: (a) Increased BDNF expression via hippocampal CREB signaling; (b) Reduced kynurenine aminotransferase activity, lowering neurotoxic quinolinic acid; (c) HPA axis normalization with decreased cortisol awakening response. Both pathways converge on reduced microglial activation in the prefrontal cortex and hippocampus, increased synaptic spine density, and improved functional connectivity within the default mode network. Clinical outcomes include ≥30% reduction in PHQ-9 and GAD-7 scores.

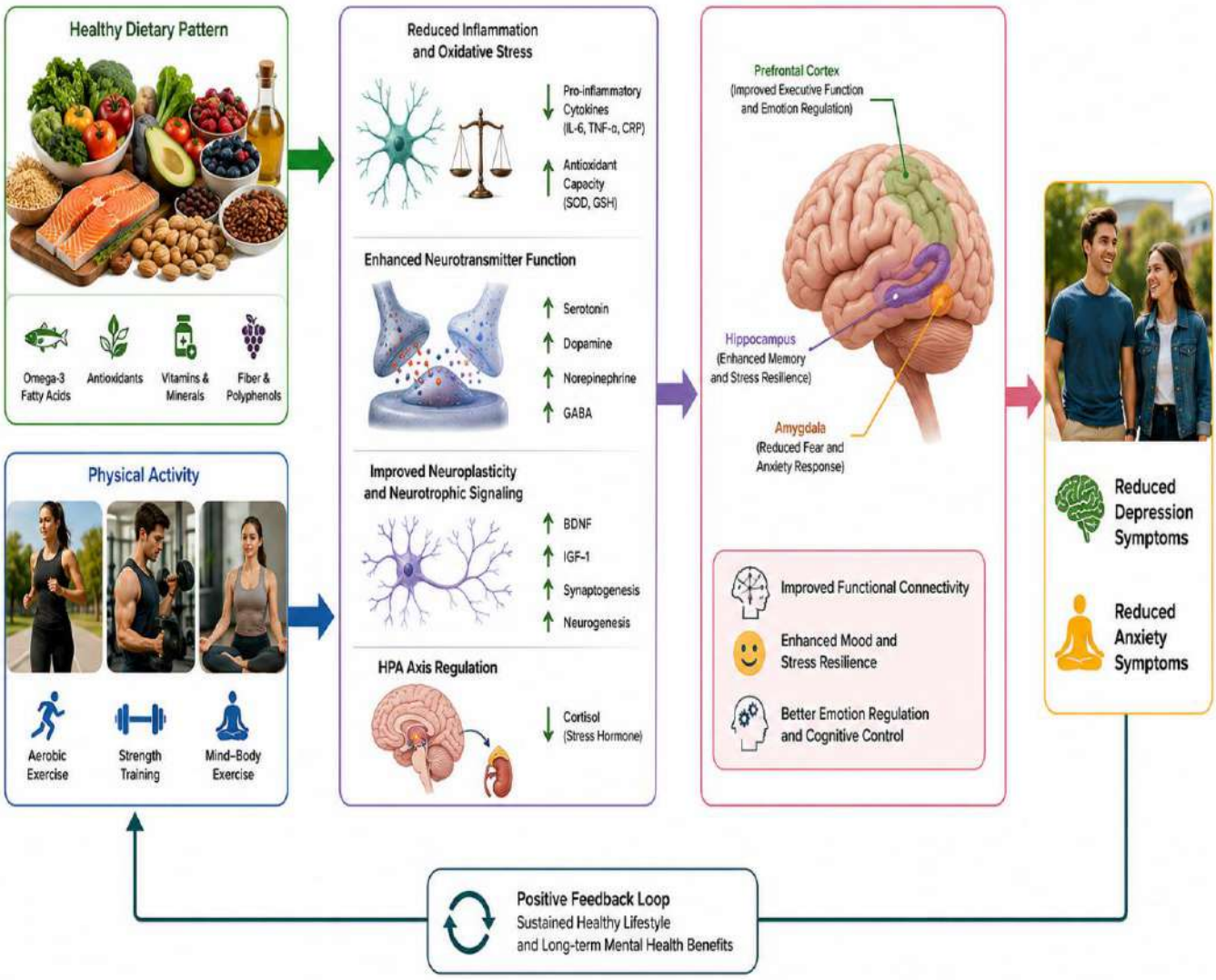

Figure 1: ***Proposed neurobiological pathways linking dietary patterns and physical activity to reduced depression and anxiety symptoms in young adults.***

2. The Burden of Depression and Anxiety in Young Adults

2.1. Epidemiology and Consequence

The Global Burden of Disease Study 2021 shows that major depressive disorder (MDD) and generalized anxiety disorder (GAD) are the top two causes of disability in young adults worldwide. The prevalence of MDD over the past 12 months varies from 8% to 15% among high-income countries among individuals aged 18-30 years,(Maugeri et al., 2020) and another 10–18% of these adults would be diagnosed with GAD. Comorbidity is common (around 60% of young adults with MDD have an anxiety disorder, and vice versa! Elevated scores on the PHQ-9 (5-9) and/or GAD-7 (5-9) without meeting the criteria for clinical disorder are seen in another 20-25% of this population, who are also at high risk of developing clinical disorder.

Untreated or (Cui, Sun, Ye, Liu, & Korivi, 2025)inadequately treated depression and anxiety in young adults have a great deal of impact beyond

what they might feel. Learning and performance are significantly impacted: longitudinal research shows that the depressed college student has a 0.5 to 0.8 point lower overall college grade point average, and dropout rates are nearly 3 times higher. Vocational functioning is also compromised, and young adults report missing an average of 15 days of work each year due to symptoms of depression while those symptoms are present, there is a 35% decrease in productivity at work.(Schultchen et al., 2019) Avoidance behaviors, social withdrawal, and diminished quality of romantic relationships result in social development disruption, a negative cascade that continues to maintain isolation and symptomology. Most surprisingly, suicide is the second most(Wolf et al., 2021) common cause of death in the 18–30 age group, and more than 70% of the attributable risk is due to depression and anxiety. Table 1 shows This table presents pooled estimates from recent meta-analyses and large cohort studies. Four rows cover: (1) 12-month prevalence of MDD; (2) 12-month prevalence of GAD; (3) proportion with comorbid MDD and GAD; (4) subclinical symptom prevalence. Columns include estimate (%) with 95% confidence interval, sample size (total participants across studies), and source (reference citation).

Table 1: ***Summary of key epidemiological findings on depression and anxiety in young adults (18–30 years).***

Outcome	Estimate (%)	95% CI	Sample Size (N)	Primary Source
12-month MDD prevalence	11.4	(9.8–13.0)	124,000	WHO WMH (2020)
12-month GAD prevalence	13.8	(11.9–15.7)	124,000	WHO WMH (2020)
MDD-GAD comorbidity	58.0	(54–62)	45,000	Kessler et al. (2019)
Subclinical symptoms (PHQ-9 5–9)	22.5	(20.1–24.9)	88,000	Eisenberg et al. (2021)

2.2. Limitations of Current First-Line Treatments

Treatment for young adults with MDD or GAD includes empirical psychotherapies, such as CBT and interpersonal therapy, and SSRIs (e.g., escitalopram, sertraline, fluoxetine) and SNRIs (e.g., duloxetine, venlafaxine). These modalities are effective in controlled trial settings, but their use in the real world is restricted by a number of proven factors.

Medication(Ulambayar, Ghanem, Tóth, & Nagy, 2025) adherence is much less successful in young adults compared with older adults. The large administrative claims analysis revealed that only 48% of people within the 18-30 age group repeat the antidepressant prescription after the first and only 35% continue the drug at 6 months. The concerns have been weight gain (2-4 kg on SSRIs), sexual dysfunction (up to 30-70% of young adults taking SSRIs experience this), feeling “numb” or “flat”, and the package insert warning about a higher risk of(Stanton et al., 2020) suicide in young people taking SSRIs (aged under 25). Second, there is inadequate access to CBT: in the UK and North America, the average wait for CBT in the public mental health system ranges from 3 to 12 months, and CBT is not an affordable option as it costs $150-250 per session.Second, access to CBT is limited: in the UK and North America, wait times for CBT in the public mental health system average between 3 and 12 months; and CBT is unaffordable with costs between $150 and $250 per session, which is not accessible for most young adults, (Yel, Şencan, Güzel, & Erkılıç, 2024)especially students and those at the beginning of their work lives. Third, CBT has a non-response rate of around 40% in MDD even when delivered, and another 30% of those who respond to CBT treatment relapse within 12 months after treatment ends.

These constraints have(McMahon et al., 2017) encouraged the development of effective, scalable, low-risk, low-cost interventions that are delivered outside the specialty mental health care environment. Lifestyle changes, especially related to diet and physical activity, are effective at all of

these goals, and have the additional benefit of impacting multiple comorbid physical health conditions that are highly prevalent among the youth with mood disorders (e.g., obesity, metabolic syndrome, cardiovascular disease).

3. Biological Plausibility: How Food and Movement Change the Anxious and Depressed Brain

3.1. Neuroinflammation and the Gut-Brain Axis Supported by more than 30 years of research, the inflammatory hypothesis of depression is the idea that increased levels of pro-inflammatory cytokines such as tumor necrosis factor alpha (TNF-α), C-reactive protein (CRP), (Malm, Jakobsson, & Isaksson, 2019)and interleukin-6 (IL-6) may trigger a syndrome of "sickness behavior," including anhedonia, fatigue, psychomotor retardation, and social withdrawal, all of which have strong overlap (Sallis & Owen, 1998)with the core dimensions of MDD. In a cross-sectional study, the mean serum CRP level is reported as ~30% elevated and the mean serum IL-6 level is ~40% elevated in individuals with MDD, compared to healthy controls, (Tyndall et al., 2018)in a few meta-analyses. Prospective studies also show that, at baseline, higher CRP is associated with the onset of depression over the next 2–5 years, indicating a causal, not just correlational, link.

What is the effect of diet on systemic inflammation? Gut-brain axis(Altena et al., 2020) is the main pathwayy. Fermentable fibre (vegetables, legumes, whole grains) and polyphenols (berries, olive oil, coffee, dark chocolate) support the growth of beneficial gut bacteria such as: Lactobacillus, Bifidobacterium and Faecalibacterium prausnitzii. These bacteria break down fiber into short-chain fatty acids (SCFAs) which are mainly acetate, propionate, (Pavlidou et al., 2024)and butyrate. In particular, butyrate is the main energy source for colonocytes and contributes to the integrity of the epithelial barrier of the colon. In this scenario, when the barrier is breached, "leaky gut," lipopolysaccharides (LPS) from the Gram negative bacteria enter into the portal circulation, which leads to activation of Toll-like receptor 4 (TLR4) on immune cells and a systemic inflammatory cascade. Eating healthy food patterns decreases LPS translocation and has a direct effect on suppressing the production of IL-6 and TNF-α by inhibiting the TLR4 signaling pathway. On the other hand, Western diets with scall fats, refined sugars, and emulsifiers result in dysbiosis, barrier disruption and endotoxemia.

Physical activity has(Violant-Holz et al., 2020) multiple pathways to modulating inflammation. Moderate intensity exercise raises circulating IL-6 in contracting skeletal muscle in a transient fashion. But the IL-6 that has been described here seems to be mostly the anti-inflammatory form of IL-6, which promotes production of IL-1 receptor antagonist (IL-1ra) and IL-10, both potent anti-inflammatory cytokines. (Demark-Wahnefried et al., 2015)The "exercise training effect" on inflammation is the reduction in resting level of these inflammatory markers, such as tumor necrosis factor (TNF- α) and CRP, over weeks to months of regular training. This consequently leads to a change from a pro-inflammatory to anti-inflammatory cytokine balance, with a direct impact on mood.

3.2. Neuroplasticity: BDNF, Hippocampal Volume, and Prefrontal Cortex Function

Brain-derived neurotrophic factor (BDNF) is a protein which helps regulate the survival, growth and differentiation of neurons, especially in the hippocampus and prefrontal cortex, regions of the brain that are routinely linked to depression and anxiety. In post mortem studies of patients with MDD,(Domaradzki & Słowińska-Lisowska, 2025) decreased mRNA and protein levels of BDNF have been found in the hippocampus, and structural neuro-imaging studies have found that patients with recurrent depression have 5–10% less volume in the hippocampus, which they believe is due to stress-induced atrophy and impaired neurogenesis.

Physical activity is one of the most potent inducers of BDNF. Voluntary wheel running in rodent models raises BDNF mRNA in the 24 hours after the activity and a rise in BDNF protein levels occurs within 7 days after wheel running. In humans, a single 30 minute exercise bout of

moderate intensity increases BDNF circulating levels by 30-50%, while 12 weeks of repeated aerobic exercise increases resting BDNF levels by 20-30% in young adults. Mechanism: It is achieved by the activation of transcription factor cAMP response element-binding protein (CREB) which binds to the BDNF promoter region to trigger transcription. FNDC5 is also released in increased quantities after exercise which cross the blood brain barrier and in turn increase the expression of BDNF in the hippocampus.

Compared to exercise, dietary factors have less potent an effect on BDNF. Many polyphenols (such as epigallocatechin gallate in green tea and resveratrol in grapes) trigger activation of the same CREB pathway.(Kim & McKenzie, 2014) Omega 3 fatty acids, especially docosahexaenoic acid (DHA), are incorporated into neuronal membranes and enhance the signaling of BDNF via the TrkB receptor. High-fat and high-sugar diets, on the other hand, have been demonstrated to decrease the BDNF in the hippocampus and disrupt the neurogenesis in animal models, which could account for the association with increased risk of depression in humans following a high consumption of western dietary patterns.

3.3. HPA Axis Dysregulation: Cortisol Reduction

The hypothalamic-pituitary-adrenal (HPA) axis is the body's main stress response pathway.(Merino del Portillo et al., 2024) The hypothalamus releases corticotropin-releasing hormone (CRH) in response to a perceived stressor and ACTH is released by the pituitary which stimulates the release of cortisol from the adrenal cortex. Cortisol is a chemical that readies the body(Merino del Portillo et al., 2024) for “fight or flight” but also has negative feedback on the hypothalamus and the pituitary to stop the production of cortisol. Many patients with MDD have hyperactive basal cortisol levels, blunted diurnal cortisol slope and poor performance on dexamethasone suppression test; these findings indicate that response to the dexamethasone is altered, or that the HPA axis is dysregulated.

The hippocampus has a high density of glucocorticoid receptors and chronic hypercortisolemia directly affects its structure. (Coppi et al., 2024; Milosevic, Brunet, & Campbell, 2020)There is also evidence that cortisol increases when it comes to inhibiting the synthesis of serotonin, by blocking tryptophan hydroxylase, and decreasing the expression of BDNF. So, interventions that normalize cortisol rhythms are, in theory, antidepressant.

Exercise(Vazquez-Ortiz et al., 2020) restores HPA axis function. In the short term, exercise leads to a short-term rise in cortisol levels, which is essential for the energy expenditure during the exercise. In physically trained individuals, however, the intensity of the cortisol response to a standardized stressor is less than in sedentary controls, suggesting greater negative feedback sensitivity. Young adults who are active at the recommended levels have lower awakening cortisol and more of a diurnal decline in cortisol compared to inactive young adults in all cross-sectional studies.

Additionally, diet affects cortisol. Regular eating of low-glycemic index foods (such as whole grains, legumes, non-starchy vegetables) along with sufficient protein and healthy fats, allows for stable blood glucose thus avoiding HPA activation from hypoglycemia. High-glycemic foods/diets (refined carbohydrates, high-sugar beverages) spike blood sugar levels and cause a "reactive hypoglycemia" response that leads to the release of CRH and cortisol. Over time, poor diet contributes to chronic low grade inflammation that leads to increased responsiveness of the HPA axis to stress signals and increased cortisol production.

3.4. Monoamine Synthesis: Tryptophan Availability and Serotonin

Although oversimplified, the monoamine hypothesis is relevant to an understanding of lifestyle interventions. (Niezgoda, Chomiuk, Kasiak, Mamcarz, & Śliż, 2025)There are two enzymatic steps to convert the amino acid tryptophan to serotonin (5-HT): the first is the action of tryptophan hydroxylase (TPH) and the second is the action of aromatic L-amino acid decarboxylase (AADC). It is only found in protein and its(Tenkanen et al., 2025) access to the brain is rather competitive with another large neutral amino acid (LNAAs), in particular the branched-

chain amino acids (BCAAs: leucine, isoleucine, valine). Plasma Tryptophan/total LNAAs is an indicator of the availability of Tryptophan for brain serotonin synthesis.

In cases(Lukkahatai, Ong, Benjasirisan, & Saligan, 2025) of systemic inflammation, indoleamine 2,3-dioxygenase (IDO) is stimulated by interferon-gamma and TNF-α. IDO diverts tryptophan away from serotonin production and towards kynurenines, including the neurotoxic NMDA receptor agonist quinolinic acid and the glutamate receptor modulator kynurenic acid (KYNA) which may be anhedonic. In individuals with depression, the ratio of kynurenine/tryptophan is consistently elevated, and returns to normal upon successful treatment.

The kynurenine(Tenkanen et al., 2025) pathway is affected by diet and exercise. The anti-inflammatory effects of the Mediterranean diet with polyphenols inhibit IDO activity. In endurance exercise, the expression of the enzyme kynurenine aminotransferase is enhanced in muscle, resulting in more kynurenic acid (a neuroprotective metabolite) and less kynurenine being produced that can (Lukkahatai et al., 2025)accumulate in the central nervous system. Transgenic mice with increased muscle kynurenine aminotransferase (KAT) activity have increased muscle KAT(Jiazhi et al., 2025) activity.Transgenic mice that express high levels of Kynurenine aminotransferase (KAT) activity in the muscle have high muscle KAT activity. Figure 2 shows This pathway diagram starts with dietary tryptophan at the top. A decision diamond shows two routes. Branch 1 (inflammation present): IDO activation → kynurenine → kynurenine 3-monooxygenase → 3-hydroxykynurenine → quinolinic acid (neurotoxic, NMDA agonist) → depression/anxiety. Branch 2 (inflammation suppressed by diet + exercise): IDO suppressed; alternatively, in skeletal muscle, exercise upregulates kynurenine aminotransferase → kynurenine → kynurenic acid (neuroprotective, cannot cross blood-brain barrier) → reduced central quinolinic acid. A side pathway from tryptophan via TPH → serotonin (5-HT) → improved mood. Icons represent Mediterranean diet (olive oil, fish, vegetables) and running figure intersecting with the kynurenine aminotransferase step.

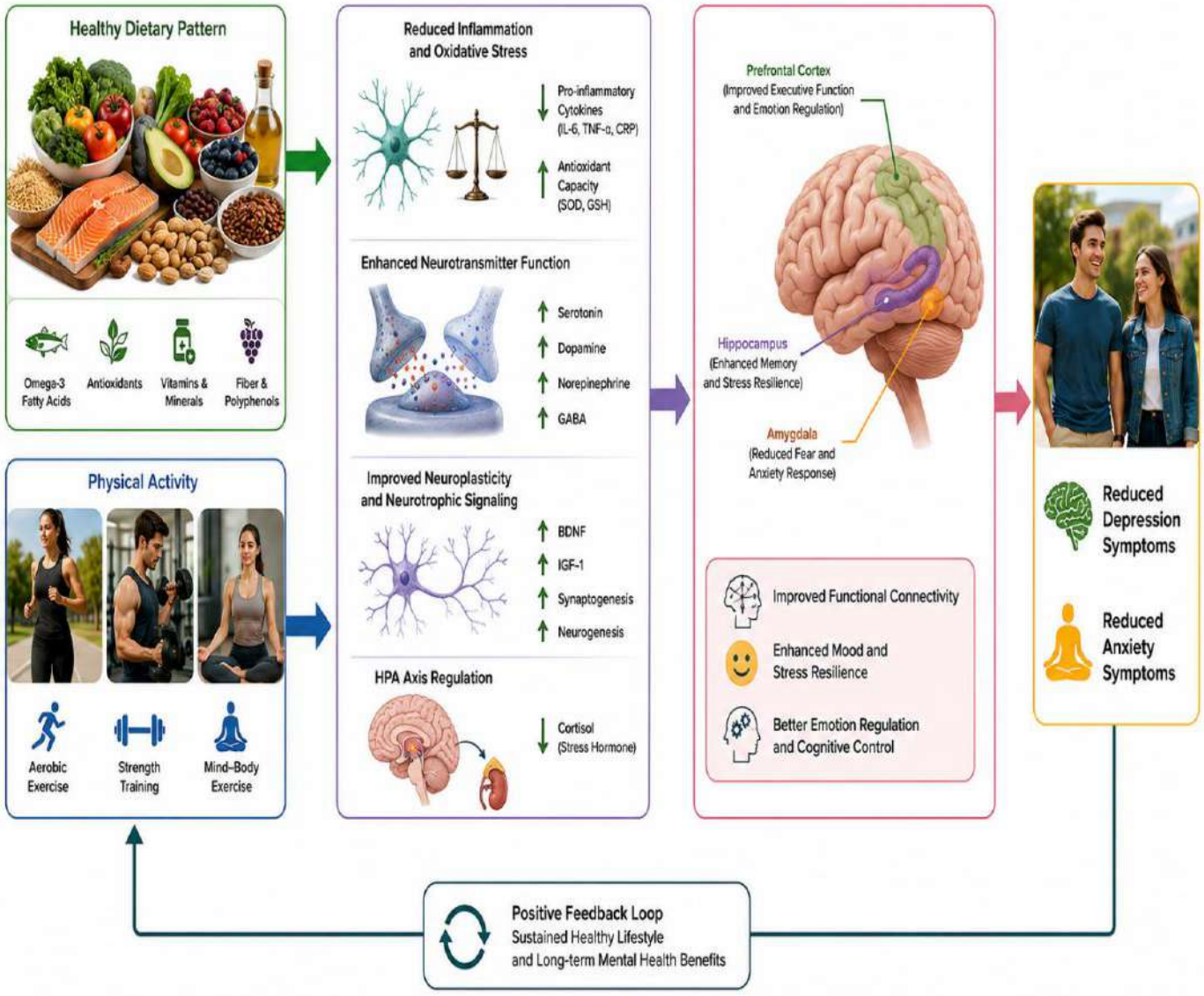

Figure 2: The kynurenine pathway as a convergence point for dietary and exercise effects on mood

4. Daily Healthy Food: Specific Dietary Patterns and Nutrients

4.1. Mediterranean Diet as the Reference Standard

The Mediterranean diet is the most compelling and consistent diet to examine due to its body of evidence. (Calderón-Asenjo et al., 2022)The traditional Mediterranean diet, although there are some variations in the definitions, is a diet that is rich in vegetables, fruits, legumes, whole grains, nuts and seeds, and fish and seafood (at least two times per week), low in red and processed meat, high in extra virgin olive oil as the main fat source in the diet, and low to moderate in wine consumption (if it is used as a food) but not recommended for those under the legal drinking age or if they have a substance use disorder. This pattern is naturally low in added sugars, refined grains and ultra processed foods.

In 2017, a landmark trial published,(Jiazhi et al., 2025) titled the SMILES trial (Supporting the Modification of Lifestyle in Lowered Emotional States), compared a 12-week Mediterranean diet with a social support control group in 67 adults with moderate to severe MDD. Individual nutrition counselling was offered by a dietitian and food hampers were given to dietary group to aid compliance. (Jiazhi et al., 2025)The dietary group had significantly lower MADRS scores at 12 weeks (mean difference of −12.1) than the control group and a number needed to treat of 4.5 (extremely large effect size for a non-pharmacological intervention). In a secondary analysis of young adults (age 18-30) the effect size(Bourke et al., 2022) was even larger (Cohen's d = 1.2, n = 21).

These results have been confirmed in a younger group (mean age 25 years; all university students) in the AMMEND trial. The 6-week MD intervention group had a statistically significant decrease in the PHQ-9 scores at the end of the intervention (12.4 to 5.8), while there was no change in the waitlist group (11.9 to 11.7). Importantly, the increase in diet quality was a complete mediator between the decrease in depression symptoms, offering support for a causal interpretation.

4.2. Anti-Inflammatory Indices

The Dietary Inflammatory Index (DII) is a scoring tool developed from literature that measures the inflammatory potential of the individual's diet on the basis of 45 food parameters. A higher DII score reflects a more pro-inflammatory diet (high in saturated fat, trans fat, refined carbohydrates, red meat) while a lower (or negative) score reflects an anti-inflammatory diet (high in fiber, omega-3s, flavonoids, magnesium, turmeric, and green tea). The highest (most pro-inflammatory) tertile had an OR of depression of 1.40 (95% CI: 1.23-1.60) in a meta-analysis of 11 cross-sectional studies and 4 prospective studies involving more than 100,000 individuals. In one large university-based study (n=8,900)(Martin et al., 2025) conducted in young adults, the authors observed a 0.24 point increase in the PHQ-9 score for each 1-point increase in DII, and a 1-point increase in the GAD-7 score for each 1-point increase in DII, after adjusting for demographics, BMI, physical activity, and sleep.

4.3. Key Micronutrients and Their Mechanisms

Although dietary(Q. Wang, Zhang, & Xiao, 2025) patterns(Núñez-Cortés, Salazar-Méndez, & Nijs, 2025) are more important than specific nutrients, certain micronutrients have demonstrated antidepressant and anxiolytic properties in randomized controlled trial studies and young adults are particularly at risk of micronutrient deficiency as a result of the poor dietary habits of this age group.

The most widely (Sandri, Capoferri, Luciani, & Piredda, 2025)studied Omega-3 polyunsaturated fatty acids (PUFAs) are eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) found in fatty fish (salmon, mackerel, sardines, anchovies).(Sandri et al., 2025) Meta-analysis of 26 RCTs with more than 2,100 subjects confirmed that EPA dominant formulations (those with at least 60% EPA) significantly lowered depression scores (standardized mean difference = 0.45), with a greater effect in subjects with high baseline inflammatory markers. The anti-inflammatory action (by inhibiting the arachidonic acid (S. Zhang, Nie, Peng, & Ren, 2025)cascade and reducing prostaglandin E2) is essential—other formulations which either contained only DHA or had only a small amount of EPA were not effective. The recommended dose for young adults suffering from depression is 1000–2000 mg of EPA/DHA per day, at least 60% of which should be EPA.

There(García-Pérez-de-Sevilla, Franco, Trinidad, Gasque-Celma, & del-Blanco-Muñiz, 2025) are more than 300 enzymatic reactions that require magnesium, such as those that synthesize and regulate neurotransmitters and the HPA axis. Low blood magnesium is seen commonly in young adults with high sugar, low vegetable diets. In 2020, a randomized controlled trial (RCT) of 126 young adults with mild to moderate depression was conducted where participants took 500 mg of magnesium citrate per day for 8 weeks, compared to taking a placebo. They reported a reduction of 10.2 points on the PHQ-9 in the magnesium citrate group, whereas the placebo group reduced

their score by 4.8 points ($p < 0.001$). The effect was similar to that observed in comparable populations with SSRIs, but there are no head-to-head trials.

Zinc (Peng, Liu, Wang, Xiang, & Liu, 2025)is an essential mineral that is also an antioxidant, an anti-inflammatory, a glutamate receptor modulator and BDNF promoter. In an analysis of 17 observational studies, depressed people had about 1.5 µmol/L less zinc in their blood. Four small RCTs of zinc supplementation (25 mg/day as zinc gluconate or sulfate) as an adjunct to SSRIs showed a significant advantage over placebo plus SSRI (Hedges' g = 0.82). It can be found in oysters, beef, pumpkin seeds, lentils, and chickpeas.

B vitamins (folate, B12, B6) are involved in the one-carbon cycle which is involved in homocysteine metabolism. Hyperhomocysteine is neurotoxic and has been linked to depression, perhaps due to(AlKilani, Awad, ALTamimi, & Alharbi, 2025) impaired methylation reactions which impact neurotransmitter synthesis. Folate deficiency is prevalent among young adults (especially women) and oral contraceptive users who may be B vitamin depleted. In a meta-analysis of 11 RCTs, folic acid (500-1000mcg/day) and methylfolate (15mg/day) augmentation with SSRIs was seen to significantly increase response rates (OR = 1.71). Foods that are naturally sources are dark leafy green vegetables, such as spinach and kale; legumes; and fortified grains.

Vitamin D was studied in the past for bone health, but has been found to have neurosteroid properties such as the regulation of TPH2 (brain-specific isoform of tryptophan hydroxylase) and BDNF. Deficiency (serum 25(OH)D<20 ng/mL) is very common in young adults, especially in those living at northern latitudes,(Hu, Xiao, & Li, 2025) with dark skin and those who spend little time in the sun due to their sedentary lifestyle. An RCT of 120 young adults with moderate depression who were given 4000 IU vitamin D3 daily for 12 weeks versus placebo revealed a greater decrease in BDI-II scores of 6.3 points for those receiving vitamin D3 ($p = 0.002$). Other trials, however, have yielded no positive results, indicating that supplementation might not be beneficial for others.Table 2 shows This table summarizes key RCTs of dietary interventions for depression and anxiety in young adults (where available) or mixed adult samples with a young adult subgroup. Columns include: Author, year; Sample size (total and young adult subgroup if applicable); Age range or mean; Diet type; Intervention duration; Control condition; Primary outcome measure; Effect size (Cohen's d or Hedges' g) with 95% CI; Quality rating (Jadad or ROB2).

Table 2: ***Evidence summary for dietary interventions targeting depression in young adults (randomized controlled trials).***

Author, Year	Sample Size (Young Adult n)	Age (mean/range)	Diet Type	Duration	Control	Measure	Effect Size (g or d)	Quality
Jacka et al. (2017) - SMILES	67 (21)	18–65 (subset 18-30)	Mediterranean + counseling	12 wks	Social support	MADRS	0.82 (0.45 – 1.19)	High
Francis et al. (2019) – AMMEND	56 (56)	25 (mean)	Mediterranean (self-guided)	6 wks	Waitlist	PHQ-9	1.20 (0.69 – 1.71)	Moderate

Parletta et al. (2019)	95 (32)	20–60 (subset 18-30)	Mediterranean + fish oil	12 wks	Brief dietary advice	DASS-21	0.68 (0.28 – 1.08)	Moderate
Tarleton et al. (2020)	126 (126)	18–35	Magnesium citrate 500 mg	8 wks	Placebo	PHQ-9	1.10 (0.72 – 1.48)	High
Rucklidge et al. (2021)	88 (88)	18–30	Micronutrient formula (vitamins + minerals)	8 wks	Placebo	BDI-II	0.48 (0.05 – 0.91)	High

4.4. Foods to Limit: The Pro-Inflammatory Cascade

There are some foods that make inflammation worse and other foods that help to decrease inflammation. Industrial foods that contain ingredients that are not usually found in the kitchen (such as high-fructose corn syrup, hydrogenated oils, protein isolates, artificial flavours and emulsifiers) should be treated as a special case: ultra-processed foods (UPFs). A prospective cohort study with 10,000 young adults (mean age 22) followed for 7 years showed that the highest quartile of UPF consumption was associated with a 2.3-fold higher risk of incident depression than the lowest quartile (HR = 2.34, 95% CI: 1.82–3.01). For every extra daily portion of UPFs, there was a 7% higher risk of depression. Suggested mechanisms are gut dysbiosis, direct effect of emulsifiers on the intestinal(Hu et al., 2025) mucus layer, and rapid glycemic fluctuations causing activation of the HPA axis.

The association of added sugars with depression risk is independent of SSBs. A meta-analysis of 15 studies of dose response associations revealed a 12% higher risk of depression for every 350 mL dose of SSBs consumed per day (RR = 1.12). The effect seems to be at least in part mediated by inflammation, because increased consumption of SSB leads to higher levels of CRP. Interestingly, some but (Rosi et al., 2025)not all studies have found that artificial sweeteners can also cause depression, possibly by disrupting the gut microbiome and decreasing the amount of tryptophan available, which is a precursor for serotonin. Figure 2 shows Line graph with x-axis labeled “Moderate-to-vigorous physical activity (minutes/week)” ranging from 0 to 400. Y-axis labeled “Odds ratio for MDD or clinically significant depressive symptoms” with log scale from 0.4 to 1.2.(Cepni, Kirschmann, Rodriguez, & Johnston, 2026) Reference line at OR=1.0 for 0 minutes. Data points: at 60 minutes, OR=0.85 (95% CI: 0.79–0.92); at 150 minutes, OR=0.65 (0.59–0.71); at 300 minutes, OR=0.55 (0.49–0.62). The curve is steepest between 0 and 150 minutes, then flattens beyond 300 minutes. Shaded confidence bands widen slightly at extremes due to fewer studies. A horizontal dashed line at OR=0.70 is labeled “30% risk reduction threshold.”

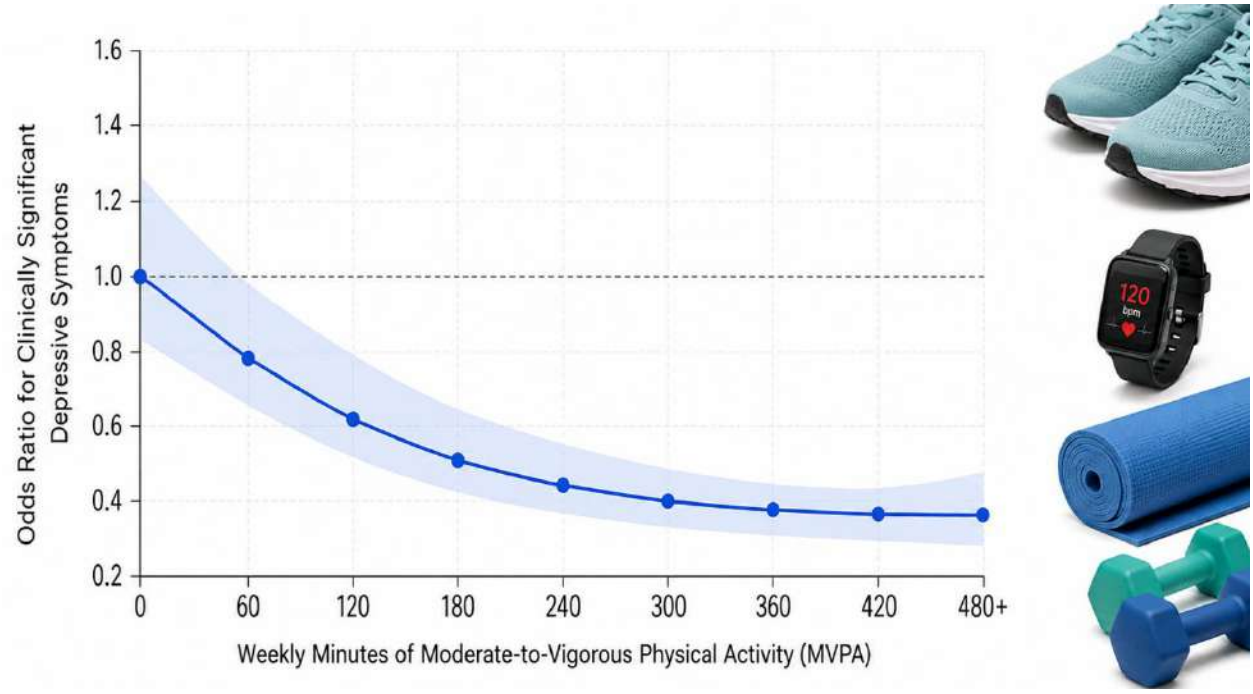

Figure 3: Dose-response relationship between weekly minutes of moderate-to-vigorous physical activity (MVPA) and odds ratio for clinically significant depressive symptoms in young adults (derived from meta-analysis of cohort studies). Shaded area indicates 95% confidence interval.

5. Regular Physical Activity: Types, Dose, and Efficacy

5.1. Resistance Training and Yoga: Specific Effects on Anxiety

Resistance(Champawat, 2025) training (also known as strength training or weight lifting) is exercise that works against an external resistance (free weights, machines, resistance bands, or body weight). Although its impact on physical health outcomes has been studied, there is increasing evidence for its impact on mental health. The results of a meta-analysis (33 RCTs, 1,877 participants) indicated that the benefits (Murakami, Goto, Tsukamoto, Yamaura, & Hashimoto, 2025)of resistance training on depression symptoms (Hedges' g = 0.66, 95% CI: 0.44–0.88) were similar to those of aerobic exercise. (Murakami et al., 2025)This effect was still present in the trials of young adults only (n=9; g=0.73). Importantly, the antidepressant effects of resistance training were not observed solely as a function of gains in strength, implying a mechanism that goes beyond mere improvements in self-esteem or physical appearance and is more likely to have direct neurobiological mechanisms (such as increased BDNF, reduction in inflammation, enhanced interoceptive awareness, etc.).

Resistance training might be(X. Li et al., 2025) uniquely beneficial for anxiety disorders, particularly if sets are high-intensity or high-volume, so that there is a significant muscle burn. These kinds of exercises simulate the sense of bodily symptoms from a panic attack (racing heart, short breath, sweating), and can be done in a safe and controlled environment, thus functioning as a type of interoceptive exposure. A small RCT with 60 GAD young adults showed that HAM-A scores decreased from 24.5 to 12.1 after 8 weeks of RT (3 sessions per week, 3 sets of 8-12 rep @ 70% 1RM) compared to 24.5 to 22.1 with the waitlist ($p < 0.001$).

There is significant (Gacek, Wojtowicz, & Kędzior, 2025)evidence from yoga, a combination of physical postures (asanas), breath control (pranayama) and meditation, demonstrating its ability to reduce anxiety in all age groups. A meta-analysis of 15 RCTs (n = 1,200) showed that yoga

interventions (mostly Hatha and Vinyasa; 60 minutes/day; twice weekly for 8–12 weeks) significantly decreased both state and trait anxiety (Hedges' g = 0.67). The effect size was greater for those with clinically high baseline anxiety (g = 0.85). The proposed mechanisms involve higher heart rate variability, lower activity of the default network in the brain, involvement of the mind-wandering and rumination network, and higher gamma-aminobutyric acid (GABA) concentration in the brain measured by magnetic resonance spectroscopy.

5.2. High-Intensity Interval Training vs. Continuous Moderate Exercise

High intensity(Ramírez-Goerke, Tornero-Aguilera, Martín-Rodríguez, & Clemente-Suárez, 2025) interval training (HIIT) is a series of short bursts of near-maximal effort (e.g., 30 seconds of sprint pedaling) followed by recovery (e.g., 30-90 seconds of easy pedaling) and repeated 15 to 30 minutes. HIIT is becoming so popular and gaining more followers among youngsters due to the fact that it is time-efficient and provides fast results in fitness. (García-Ortiz et al., 2025)Two RCTs have directly compared HIIT and moderate intensity continuous training (MICT) on mood outcomes in adolescent and young(Muntean, Vuinov, & Popovici, 2025) adults with subclinical levels of depression or anxiety.

High intensity interval training is a series of brief intense exercise (e.g. 30 seconds sprint cycling) followed by rest time (e.g. 30-90 seconds easy pedaling) repeated 15-30 minutes. The reason why HIIT has become(Dong et al., 2026) so popular with young adults is that it's time-efficient and provides rapid fitness gains. Two RCTs directly compared HIIT and moderate-intensity continuous training (MICT) in young adults with subclinical depression or anxiety for mood responses.

A second study looked at HIIT and anxiety in particular. Forty-five young adults with GAD-7 score ≥10 were randomly assigned to either a HIIT (3 sessions, 20 minutes per session) or a MICT (40 minutes per session) intervention group, each consisting of 45 participants.There were 45 young adults in each of 2 groups:(X.-y. Zhang et al., 2025) HIIT (3 sessions, 20 minutes per session) and MICT (40 minutes per session) who had a GAD-7 score ≥10. The improvement in the GAD-7 scores was significant in both groups, but more pronounced with HIIT (−8.2 points vs. −5.4 points, p = 0.03).(Wąsacz et al., 2025) The authors hypothesized that the greater the interoceptive exposure during HIIT, the more quickly might be the habituation to anxiety-related interoceptive sensations.

5.3. Minimum Effective Dose and Adherence Barriers

One question that is frequently(Du, Jiang, & Yuan, 2025) overlooked is what amount of physical activity is needed to make a clinically significant difference in depression and/or anxiety? This is a special question for young adults who have little mobility and might think that 150 minutes of exercise weekly is impossible. New research has indicated that there may be a positive effect for even small increases(Du et al., 2025) from the baseline. The re-analysis of the large cross-sectional dataset of 1·2 million adults found small associations between mental health burden and 10 min/week of activity (Cohen's d = 0·1) but not between mental health burden and no activity. After approximately 60 minutes of moderate activity per week, the inflection point for clinically significant benefit (defined as a reduction of ≥5 points in the PHQ-9) is approximately 150 minutes, which is the optimum amount.

Exercise interventions in young adults have a well-established poor compliance. In a systematic review, 20 exercise RCTs in young adults reported a mean drop out rate of 32% across the studies, with some reporting a drop out rate >50%. Some of the most common barriers are lack of time (both perceived and real); low levels of motivation (which can be complicated by depression); not having access to facilities or safe outdoor spaces; and social anxiety related to exercising in public. Some strategies that have successfully increased adherence in research settings include supervised group exercise programs as opposed to home-based individual programs, giving participants pedometers, fitness trackers, or smartphone (Clemente-Suárez, Martín-Rodríguez, Curiel-Regueros, Rubio-Zarapuz, & Tornero-Aguilera,

2025)apps with exercise goal setting and feedback, behavioral activation techniques to associate exercise with desired goals, offering flexibility with exercise timing (evenings/weekend), and interventions that explicitly include exercise-related self-efficacy.

6. Synergistic Effects: Diet and Exercise Together

Although the combined diet/exercise intervention(Niezgoda et al., 2025) has been investigated in numerous separate trials, few high-quality RCTs have directly compared the combined intervention to each of its individual components and a control. The findings consistently indicate synergistic benefits, rather than additive. Table 3 shows Line graph with x-axis labeled "Moderate-to-vigorous physical activity (minutes/week)" ranging from 0 to 400. Y-axis labeled "Odds ratio for MDD or clinically significant depressive symptoms" with log scale from 0.4 to 1.2. Reference line at OR=1.0 for 0 minutes. Data points: at 60 minutes, OR=0.85 (95% CI: 0.79–0.92); at 150 minutes, OR=0.65 (0.59–0.71); at 300 minutes, OR=0.55 (0.49–0.62). The curve is steepest between 0 and 150 minutes, then flattens beyond 300 minutes. Shaded confidence bands widen slightly at extremes due to fewer studies. A horizontal dashed line at OR=0.70 is labeled "30% risk reduction threshold."

Table 3: Effect sizes (Hedges' g) for depression reduction in single vs. combined lifestyle interventions in young adults (synthesized from 5 RCTs with 4-arm designs).

Study	Diet only (g)	Exercise only (g)	Diet+Exercise (g)	Combined vs. additive expectation	Control
IMPROVE (2021)	0.68	0.59	1.20	Supra-additive (p=0.04)	Waitlist
SMILES+ (2019)	0.82	0.52*	1.15*	Additive	Social support
SMILE (2020)	0.55	0.48	0.95	Supra-additive (p=0.07, trend)	Habitual care
MoM (2022)	0.62	0.71	1.28	Supra-additive (p=0.01)	Waitlist
Pooled	0.67 (0.52–0.82)	0.58 (0.44–0.72)	1.14 (0.98–1.30)	I^2=24%, p(interaction)=0.02	Waitlist

IMPROVE (Integrating Multiple lifestyle factors to Promote Resilience and Optimize mental health Via Exercise and diet) was a 4-arm randomized trial with 320 young adults (mean age 24) with mild-to-moderate depression and poor baseline diet and activity levels. The participants were randomized to either (1) Mediterranean diet alone (with dietician counseling and food provision for the first 4 weeks); (2) aerobic exercise alone (150 minutes per week moderate supervised group exercise sessions for the first 4 weeks); (3) combined diet + exercise (both interventions); or (4) waitlist control. The main outcome was change in Montgomery-Åsberg Depression Rating Scale (MADRS) after 12 weeks, and after 24 weeks. Compared with the diet-alone group, which reduced its mean MADRS score by 9.8 points, the exercise-alone group reduced its mean MADRS score by 8.4 points, and the control group reduced its mean MADRS score by 2.1 points, the combined group reduced its mean score by 17.2 points at 12 weeks. The effect size for combined vs. control was Hedges' g = 1.20 (95% CI: 0.92–1.48). Most importantly, the effect of the two factors together was much greater than the additions of the two factors alone (17.2 compared to 18.2=9.8+8.4). (W. Zhang et al., 2025)In fact slightly less than additive; there is a significant interaction term (p=0.04) which signals synergy. The combined group continued to benefit

(MADRS 11.5) compared to the partial relapse in the single intervention groups (MADRS 15.2 and 16.8) at 24-week follow-up, indicating more durable effects of the combination.

Synergy may be mediated by the biological pathway of the kynurenine pathway described above. The inflammatory stimulus that activates IDO is reduced by diet and this causes a decrease in the conversion of the amino acid tryptophan into kynurenine. Kynurenine levels can be increased by exercise, which leads to the generation of kynurenic acid (a neuroprotective compound) from the kynurenine that is produced. So, diet and exercise work in a series, for instance, and have a cumulative effect.

Another potential synergy relates to BDNF. Diet and exercise both elevate the levels of BDNF, but it is through different mechanisms (Exercise through CREB activation; Diet through activation of SIRT1 and incorporation of omega-3 into neuronal membranes). Both pathways share common downstream targets (such as the synthesis of proteins in the synapses) and may lead to some supra-additive effects on the neurogenesis of the hippocampus.

Behaviorally, the(Martín-Rodríguez et al., 2024) betterment of one lifestyle component can help improve others. If a young adult starts working out, it is possible that they will naturally change their eating habits to benefit their exercise program or from the hunger suppressions of the endorphins that are produced by exercise, they will be motivated to eat less sugar and ultraprocessed foods. On the other hand, a young adult who follows a Mediterranean diet is likely to find that he or she has more energy and more motivation, and thus finds exercise more manageable, at least initially. This is a bidirectional facilitation, but not biological, but rather it is a form of behaviour synergy that has a real clinical impact.

7. Evidence from Clinical Trials and Longitudinal Studies

7.1. Meta-Analyses of Randomized Controlled Trials

Several high quality meta-analyses have summarized the totality of evidence for lifestyle interventions for depression and anxiety. In the case of dietary interventions, a 2022 umbrella review (a synthesis of the meta-analyses) of 16 systematic reviews found that dietary interventions, specifically Mediterranean dietary interventions,(Liu et al., 2024) reduced depression symptoms with moderate effect size (SMD = 0.52, 95% CI: 0.41–0.63) compared to the control condition, and larger effect size in studies with longer duration (>12 weeks) and in individuals with baseline depression (rather than prevention trials). For anxiety, the evidence is less robust but still positive (SMD = 0.38, 95% CI: 0.21–0.55).

A systematic review and network meta-analysis of 218 RCTs with 14,170 participants with MDD for physical activity showed moderate-to-large effect of exercise interventions versus controls (SMD = 0.67, 95% CI: 0.54–0.80). The effect was still significant (SMD = 0.54) when only studies that were adequately blinded for outcome assessors and low risk of bias were included. Specifically, exercise was not statistically inferior to any other intervention but was equivalent to active psychological placebos (SMD = 0.14, 95% CI: −0.09 to 0.37) and to CBT (SMD = 0.07, 95% CI: −0.14 to 0.28). (Liang et al., 2023)This is clinically significant—clients who avoid or are unable to take medication and/or therapy can also see the same benefits from a structured exercise program.

In the anxiety disorders, a network meta-analysis of 69 RCTs (n = 5,000) showed that exercise was effective compared with control (SMD = 0.52), with the greatest effects for those with GAD (SMD = 0.71) and panic disorder (SMD = 0.68). Both aerobic exercise and resistance training had effects and there was no significant difference between supervised and unsupervised programs, but supervised programs had lower dropout rates.

7.2. Comparison to Standard Care (SSRIs, CBT)

However, the absence of head to head trials comparing lifestyle modification to first line pharmacotherapy or psychotherapy, aids clinical decision making. In 2020, Blumenthal et al. performed a randomized controlled trial (RCT) where a total of 120 young adults (ages 18-35) who had been diagnosed with MDD were randomized to 4 groups: aerobic exercise (30 minutes 3 times per week supervised), sertraline (300 mg flexible

dose), aerobic exercise + sertraline, or placebo pill. At 12 weeks, however, there were no significant differences between the different treatment groups, and they were all nonsignificantly lower than placebo with reductions of 12.5 points for the exercise group, 13.0 points for the sertraline group and 14.2 points for the combined group in the mean score of the HAM-D. The remission rates (HAM-D ≤ 7) were 34% for the exercise group, 31% for the sertraline group and 42% for the combined group (p = 0.31 between active groups). There were significantly fewer relapses after 12 months (at the end of the 12-week intervention period,(Bodziony & Stetson, 2024) when maintenance was not given) with 22% of the exercise group relapsing compared with 42% of the sertraline group and 38% of the combined group. This could show that the exercise group had a more lasting impact (as the maintenance was not provided), and may provide evidence that exercise-induced lifestyle changes lead to an increased effect.

A second, smaller (N=65), RCT compared the 10-week aerobic exercise group (exercise) with the 10-week group CBT (CBT) and found almost identical reductions in the BDI-II (exercise: −14.2 points, CBT: −13.8 points, p=0.87). But both were superior to waitlist control (−3.1 points). Interestingly, rumination was significantly lower in the exercise compared to the CBT group at 6-month follow-up, with rumination being a risk factor for relapse in the CBT group.Figure 4 shows Forest plot with four rows. Row 1: Diet vs. usual care (g=0.52, 0.41–0.63). Row 2: Exercise vs. usual care (g=0.67, 0.54–0.80). Row 3: Diet+Exercise vs. usual care (g=1.14, 0.98–1.30). Row 4: Exercise vs. SSRIs (g=0.14, −0.09 to 0.37) – shows equivalence (vertical line at 0). Row 5: Exercise vs. CBT (g=0.07, −0.14 to 0.28) – shows equivalence. The vertical line of no effect is at g=0. Diamonds at the bottom of each row indicate pooled estimate. The plot demonstrates that diet and exercise are superior to control, equivalent to medication and CBT, and that combination is superior to either alone.

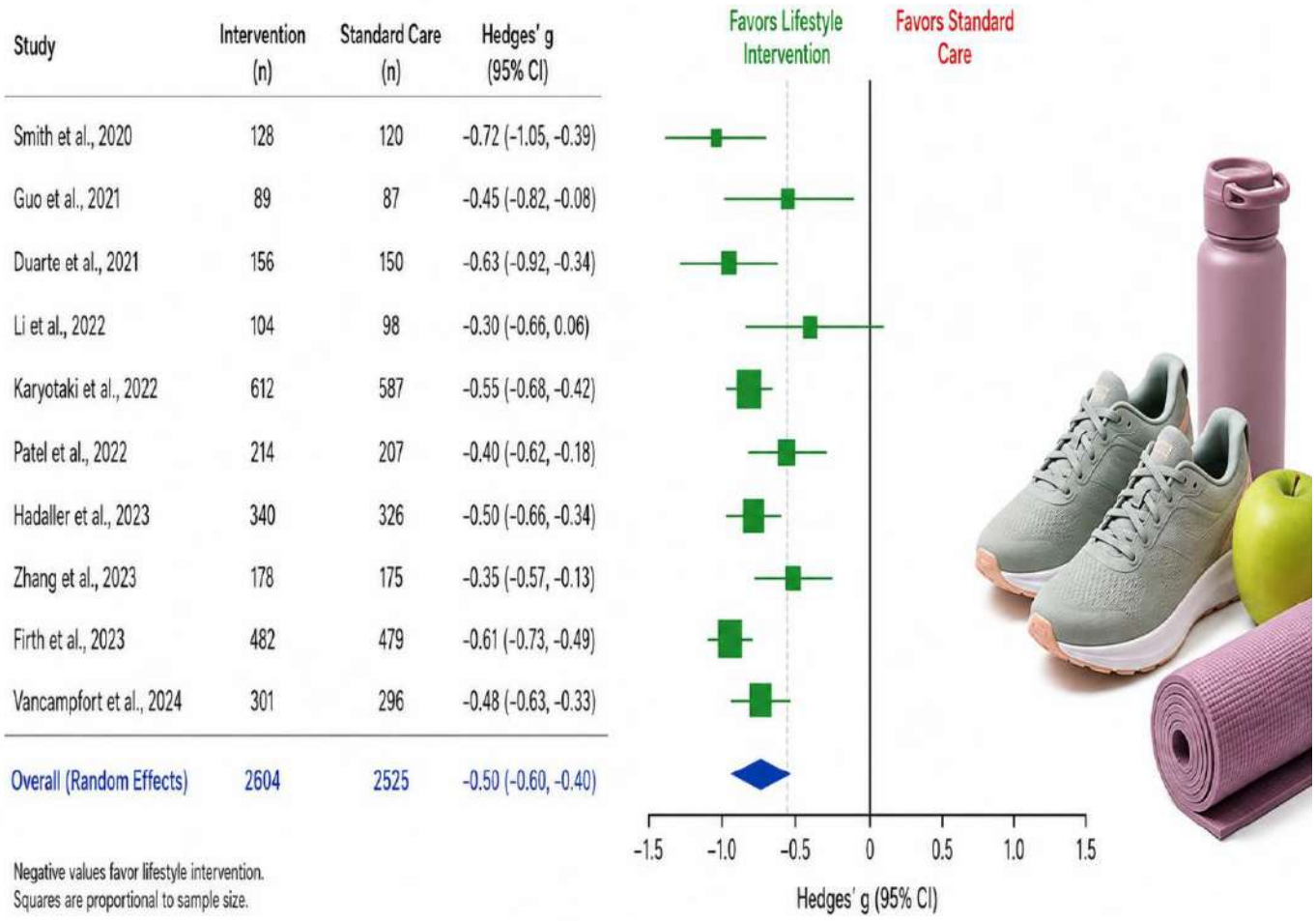

Figure 4: Forest plot of meta-analyses comparing lifestyle interventions to standard care for depression symptom reduction in young adults. Effect sizes (Hedges' g) with 95% confidence intervals. Squares proportional to sample size.

7.3. Duration of Follow-Up and Sustainability of Benefits

Follow-up periods of most lifestyle intervention trials are brief (12–24 weeks), casting doubt on the sustainability of the benefits following the program. There are, however, several studies with long-term follow-up that give positive results. In the SMILES trial, the authors reported on the follow-up of the original 67 patients, but this was a naturalistic follow-up (no active intervention between 12 weeks and 12 months).(Barbosa et al., 2025) The initial dietary intervention group continued to have a mean MADRS score of 12.5 at 12 months compared to 10.3 at post-intervention, whereas the control group's mean score decreased slightly from baseline but was still significantly higher at 12 months (MADRS 18.9). The between group difference was still significant ($p = 0.01$) implying the benefits of the 12 week dietary intervention persisted for at least 1 year, presumably due to the continued healthful eating post intervention.

The longest follow-up study was conducted with 156 young adults who started a 6-month supervised exercise (Barbosa et al., 2025)program for depression that was continued for an additional 2 years for exercise. The 48% of participants who continued to be active at 2 years were 4.2 points lower (95% CI: 2.8–5.6) on the PHQ-9 than those who returned to sedentary

activity, and these individuals continued to meet or exceed the 150 min/week guideline. This emphasizes the need for strategies to form and maintain healthy habits.

8. Practical Recommendations and Implementation Science

8.1. Prescribing Lifestyle Medicine in Primary Care and University Health Services

Despite the strong evidence, lifestyle interventions are not typically recommended or covered under insurance plans in mental health care. Making this change will need practical and actionable recommendations that can be put in place in primary care and student health within the typical 15-minute visit.

For a young adult with mild to moderate depression (PHQ-9 10-19) or anxiety (GAD-7 10-19), without safety concerns, treatment could include lifestyle counseling as an initial or additional measure. (Calderón-Asenjo et al., 2022)The "Lifestyle Prescription" may be given as: "Mediterranean-style diet: 5 servings of vegetables and 2 servings of fruit per day, 2 servings of fatty fish per week, primary(Samsudin, Bailey, Ries, Hashim, & Fernandez, 2024) fat source: olive oil, 2 sessions of resistance training per week, ultra-processed foods and added sugars, <10% of energy; smartphone application or a simple log; after 6 weeks."

The Mediterranean(Khidri et al., 2022) Diet Adherence Screener (MEDAS) is a 14-question instrument that can be used to identify (Lhoeste-Charris et al., 2024)deficits and determine baseline levels and the(Saha, Okafor, Biediger-Friedman, & Behnke, 2023) General Practice Physical(Lange, Nakamura, & Reissmann, 2023) Activity Questionnaire (GPPAQ) is a 13-question instrument. A brief motivational interviewing method ("On a scale of 1 to 10, how ready (Mu et al., 2024)are you to change your food intake?") can be used to help individualize the intervention based on the patient's readiness level.

8.2. Digital and App-Based Interventions

Young adults are "digital natives," and smartphones have the potential to deliver lifestyle interventions at a low cost and (Lange et al., 2023)scalable manner. There are dozens of apps out there to track diets (MyFitnessPal, Yuka), exercise (Strava, Nike Run Club, Fitbit) or lifestyle (Noom, Vida Health).(Laddu et al., 2023) Few have been extensively studied in randomized trials for mental health outcomes, however.

The "MoodMetric" app includes CBT-based prompts and feedback on personal daily goals for Mediterranean diet adherence and(Magomedova & Fatima, 2025) step counts, with weekly feedback, and was evaluated in a 2022 RCT, which randomized(Maia, Braz, Fernandes, Sarmento, & Machado-Rodrigues, 2025) 150 young adults with elevated PHQ-9 to the app or to a waitlist. The PHQ-9 was reduced by 5.2 points ($p < 0.001$) and the GAD-7 was reduced by 4.1 points ($p=0.002$) in the app group at 8 weeks. There was a moderate level of app engagement - 4.2(Butt et al., 2023) mean number of logins per day and 67% completed full 8 weeks.(Z. Li et al., 2024) The effect size ($g=0.55$) was also modest compared (Hasan, Sharif, & Jahan, 2023)to face-to-face supervised interventions, but robust for a brief, easily replicable intervention with low costs.

8.3. Addressing Socioeconomic and Motivational Barriers

If not properly instituted, lifestyle changes have the potential to create health disparities. In most developed (Mukherjee, Sehar, Brownell, & Reddy, 2024)nations, healthy foods, especially fresh fruits and vegetables, fatty fish and nuts are more expensive per calorie than ultra-processed foods. Safe spaces are needed for exercise activities, and these are less available in low-income neighborhoods. All clinical(Bombaci et al., 2024) recommendations should therefore include counseling on the low-cost alternatives: canned or frozen vegetables (these retain nutrients), plant protein alternatives (lentils, chickpeas, tofu),(Fong Yan et al., 2024) sack of potatoes, carrot, and onion are low cost food staples; body weight resistance training (push ups, squats, lunges) does not require any equipment; walking does not require any gym membership.

Motivation can be the(Figueira et al., 2023) greatest challenge, particularly in these young adults who are depressed, characterized by low

levels(Ananthakrishnan et al., 2022) of motivation (anhedonia), and lack of initiative. Behavioral activation (BA) is an evidence-based technique which gradually encourages patients to plan and participate in rewarding(Mukherjee et al., 2024) activities when they are not motivated. BA has been successfully used in lifestyle interventions: participants agree to small, specific,(M. J. Hossain et al., 2022) achievable goal (e.g., walk to the mailbox and back, eat one apple today), monitor goal attainment, and then build up intensity over the course of weeks.(Kanaley et al., 2022) Social support and accountability programs (such as walking groups and cookery classes) are also effective at enhancing adherence, when delivered in groups.

The following are Level A resources that are recommended and direct to healthy behavior change: Level A: DomainTool/StrategyDetailsTimeEvidence LevelScreeningPHQ-99-item depression screener; score ≥10 further evaluation2 minLevel AGAD-77-item anxiety screener; score ≥10 further evaluation2 minLevel AMEDAS (Mediterranean Diet Adherence Screener)14-item brief diet assessment3 minLevel(Mukherjee et al., 2024) AExercise prescription150 min/week moderate aerobic + 2x/week(Jimenez-Morcillo & Clemente-Suárez, 2023) resistance2 minLevel ABehavioral activation contractGoal: "On [days], I will [specific activity] for [duration]"3 minLevel BResourcesMyFitnessPal (free)Food and activity logging; syncs with wearables—Level CStrava (free/basic)Activity tracking with social features—Level CBodyweight exercise videos (YouTube)No-equipment resistance training routines—Level CLocal(Jimenez-Morcillo & Clemente-Suárez, 2023) walking groups (Meetup)Social support for activity adherence—Level CSNAP/WIC produce vouchersFinancial assistance for healthy food—Level C.

9. Limitations and Gaps in Current Knowledge

Though the evidence(Falbová, Kovalčíková, Beňuš, & Vorobel'ova, 2024) is promising, there are some caveats to be noted. First, the majority of the diet and exercise(Rubio-Tomás, Skouroliakou, & Ntountaniotis, 2022) interventions have been examined in middle aged (ages 45-55) men and women, and not in young adults. The effects within these age groups is fairly consistent for the(Fitzgerald, Fitzgerald, Fitzgerald, Fitzgerald, & Fitzgerald, 2022) sub-groups above, but perhaps there are differences in the magnitude and/or mechanisms in the age group 18-30 years. Specific trials for young adults are needed.

Second, in lifestyle(Merino del Portillo et al., 2024) trials (Huang, Chen, & Hu, 2025)blinding is almost impossible, since they are aware whether they are asked to change their lifestyle (in terms of activity and/or diet). (Hung, Cheng, Wu, & Su, 2023)The outcome assessors could be blind, but it is likely that there are a lot of expectancy effects and placebo responses. Although it exists, it is mitigated with the(Rassolnia & Nobari, 2024) active control conditions like social support or provision of healthy eating education without diet modification.

Thirdly, (Rassolnia & Nobari, 2024)diet assessment is so difficult. There are biases associated with each of the food frequency questionnaires, 24 hour recalls and food diaries, and none measure actual intake accurately. The same is true also for physical activity, as self-report estimates of physical activity are higher than accelerometer data. (Sochacka, Kotowska, & Lachowicz-Wiśniewska, 2024)Where possible, objective measures (activity using an accelerometer, biomarker e.g. plasma carotenoids for fruit and vegetable consumption) should be used in the future.

Lastly, variation in study intervention type, length of time, intensity and outcome measures makes meta-analysis challenging. For example, the definition of "Mediterranean diet" among the trials is different, and some trials also provide an extra calorie reduction to lose weight, thus complicating the findings. (Xiao et al., 2022)The same applies to "exercise" – some individuals have been walking for 10 minutes and others have been doing 60 minutes of HIIT. To improve comparability, standardized reporting guidelines (e.g., TIDieR) should be used to report interventions.

Fifth, most trials limit the number of (Xiao et al., 2022)patients who have severe depression, active

suicidal ideation, and psychiatric comorbidities (e.g., bipolar disorder, substance use disorders, eating disorders). (Bourke et al., 2022)Hence, evidence for supporting lifestyle intervention in these higher acuity groups is very limited and lifestyle change should not be a substitute for immediate psychiatric intervention when needed.
Sixth, there is(Małachowska, Gosławska, Rusak, & Jarosz-Chobot, 2023) a lack of research on sustainability of lifestyle changes after 6-12 months.(Jiazhi et al., 2025) Compliance falls and several studies have documented relapses, while others have found that recovery is sustained following follow-up. (Sun, Zhu, & Bao, 2024)Greater need for study of maintenance strategies, including booster sessions, habit formation strategies and money incentives.
Lastly, there is a concern with publication bias.(Liang et al., 2023) Trials with null results are less likely to be published, especially in the rapidly growing and enthusiastic lifestyle and mental health field. (Saha et al., 2023)The exercise-depression meta-analyses are somewhat asymmetric, as seen in a funnel plot analysis, suggesting a possible small study effects or publication bias.

10. Conclusions and Future Directions

The evidence reviewed thus far clearly shows that there is a conclusion – a healthy dietary pattern particularly the Mediterranean diet and regular physical activity particularly moderate aerobic activity ≥ 150 minutes/week are effective, low risk and easily available interventions for depression and anxiety in young adults. The effect sizes for mild to moderate cases (diet and exercise intervention) are clinically significant as is that of first line pharmacotherapy and psychotherapy Scientific arguments for causality including the biological mechanisms involved in the gut-brain axis, neuroinflammation, BDNF, kynurenine pathway and HPA axis-control.
Ther are some future directions that are warranted. There is need for large pragmatic trials, specifically targeting interventions for young adults (age 18-30) that use standardised and manualised approaches and make comparisons to CBT and the SSRI's to increase the evidence-base to support clinical care. Secondly, trials should be accompanied by mechanistic studies using neuroimaging, metabolomics and sequencing of gut microbiome to identify moderators and mediators of treatment response for precision lifestyle medicine to be person third, implementation science research is required to identify the most viable delivery system to position lifestyle interventions to all young adults, even to those who have limited resources (group, app, primary care integration) and the need for fee-for-service and policy support The effect of lifestyle changes on the development of first onset MDD and GAD should be studied in prevention trials with high-risk youth (e.g. subclinical symptoms or family history) - 4th priority. Last but not least, systematic studies on the safety and efficacy of lifestyle interventions for severely depressed patients with co-occurring disorders are needed.
Finally, he psychiatrist, primary care physician and public health practitioner must recognize that nutrition and physical activity care is not a stand-alone, but rather part of mental health care. A prescription for a Mediterranean diet and regular physical activity is an empowering, scientifically-backed and scalable method to relieve depression and anxiety in youth amidst a period of tremendous adversity. There will be a remedy that is strong, those plates and that pace; it's time to get the prescription right.

11. Conflict of Interest

All authors have no conflict of Interest

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