**Role of Nutrition on Mood and Mental Health for Adolescent Girls: A Review**

**ABSTRACT**

Girls face substantial risk of facing mental health disorders like anxiety and depression throughout their adolescence because this developmental phase brings major psychological and physical changes. A rising number of chronic psychiatric illnesses impacts approximately 31.03% of the population according to studies while female individuals experience greater prevalence rates. Three primary lifestyle changes which include increased stress levels together with diminished sleep quality and reduced social networking and unhealthy eating practices drive this mounting trend.

Researchers now show that dietary choices play an essential part in supporting emotional response as well as cognitive functioning. The mental health improvement triggered by key nutrients such as iron together with omega-3 fatty acids and zinc alongside vitamins B6, B12 and folate has been proven through research studies. The adoption of Mediterranean-style eating patterns contributes to reducing the occurrence of mental health diseases. This investigation examines how mental health effects adolescent girls based on their diet quality assessment through the Index of Nutritional Quality (INQ). Research seeks to demonstrate the role of better nutrition in protecting adolescents from psychological conditions and mood disturbances among vulnerable groups.

**Keywords:** Nutrition, Mood, Mental Health, Adolescent Girls and Adolescence

1. **INTRODUCTION**

Adolescent psychological disorders are a significant global public health concern. These illnesses lead to despair, anxiety, and stress in many people worldwide (Abbasalizad *et al*., 2018). Depression is a chronic and recurring condition that can bring disability and self-destruction if not treated (Slavich *et al*., 2018). Anxiety and other mental problems can develop into chronic conditions with negative consequences. These problems lead to significant expenses for both personal and public health. According to a meta-analysis of research done between 2007 and 2018, the prevalence of chronic psychiatric diseases in Iran was 31.03%, which is comparable to the rise observed in Western nations. According to research, these issues are more common in women than in men (14.9% vs. 29.8%). A rise in mental problems has been connected to changes in lifestyle, including sedentary lives, chronic stress, less social support, sleep patterns, sun exposure, and food choices.

Although there is scant research on the relationship between nutrition and mental health, the two have a clear link (Lopresti *et al*., 2013;). Research on the link between nutrition and mental health has primarily examined dietary quality and nutrient intake. It has been demonstrated that dietary intakes of certain minerals (11), such as iron (Zhang *et al*., 2013), vitamin B6, omega-3 fatty acids, zinc, vitamin B12, folate, and selenium, are linked to better anxiety and sadness. Additionally, following a Western-style or poor diet, which includes a high intake of red and processed meat, saturated fatty acids (SFA), and simple sugar, is linked to negative outcomes, while adhering to healthy and high-quality diets, like the Mediterranean-style diet, is linked to a lower incidence of mental disorders (Weng et al., 2012; Jacka et al., 2010). Various nutritional tools and approaches have been used to study the link between diet quality and illness risk. The Index of Nutritional Quality (INQ) is widely used to assess various disorders and outcomes. The INQ provides an overall assessment of diet quality and can change total energy consumption (Vahid *et al*., 2018). The INQ compares micronutrient consumption to recommended amounts, adjusted for total energy intake (Vahid *et al*., 2018). The INQ could be used to achieve three different outcomes. In the first scenario, the individual drinks the same amount of the nutrient as recommended. In other ways, individuals may consume more or less micronutrients than the suggested levels (Vahid *et al*., 2018). This allows us to assess a person's diet quality and its impact on health outcomes/diseases. Several research have employed this strategy and found interesting results, but few have focused on mental problems.

Examining eating patterns and quality can offer a full understanding of dietary habits. Predicting illness risk with these factors is more accurate and powerful than relying solely on food or nutrients (Sadeghi *et al*., 2019)). A full examination of diet and nutrient patterns is important due to their complex composition to understand disease etiology and potential dietary impacts on the prevention, treatment, and control of chronic diseases (Jafari *et al*., 2015). This review analyzes the relationship between food quality (evaluated by INQ), nutritional patterns, and mental health in adolescent girls. We hypothesized that a high-quality, nutrient-rich diet reduces the risk of acquiring mental problems and may help alleviate them.

1. **Methods**

Methodology background: The review focused on nutritional advice related to psychoprophylaxis and dietary management of psychiatric diseases. Despite numerous studies, there is still a lack of current knowledge on this topic. To address this, the authors comprehensively reviewed relevant sources and compiled them in one place.

**Evaluation of anthropometric factors and blood pressure**:

Anthropometric measures were made without shoes and while wearing light clothing. Using a portable stadiometer (Seca, Germany) and a standardized scale (Seca, Germany), weight and height were recorded to the closest 0.1 cm and 0.1 kg, respectively. Body Mass Index (BMI) is computed by dividing weight in kilograms by height in square meters. BMI was recorded as a standardized z-score for girls aged 5 to 19. BMI categories were defined using cut-off criteria established by the World Health Organisation. Participants were classified as severely thin (BMI z-score < -3), thin (BMI z-score < -2), normal weight (− 2 < BMI obesity (BMI z-score > +2), overweight (BMI z-score > +1), or z-score < 0 and 0 < BMI z-score < +1). Using a non-stretchable tape measure with a precision of 0.1 cm, the Waist Circumstance (WC) was measured at the narrowest part of the torso. Systolic and diastolic blood pressure (SBP and DBP) were measured with a sphygmomanometer to the closest 5 mmHg.

**Dietary fat, omega-3 fatty acids**

Omega-3 fatty acids have been the subject of the majority of studies on dietary fat and men's health. ADHD, depression, and bipolar illness have all been related to omega-3 fatty acids, specifically eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA). Epidemiological studies suggest a link between higher fish consumption and lower prevalence of bipolar and mood disorders (Parker *et al*., 2006; Appleton *et al*., 2007; Appleton *et al*., 2007; Sanchez *et al*., 2007; Kamphuis *et al*.,2006), as oily fish contain long-chain omega-3 fatty acids (Parker *et al*., 2006). Not all research, though, supports this. Studies show that the amount of DHA in brain tissue and the amount in erythrocyte membranes are positively correlated (Makrides et al., 1994). The connection between essential fatty acids and a number of illnesses, such as ADHD, bipolar disorder, depression, prenatal depression, and postpartum depression, is discussed in this section.

**Bipolar disorder**

There is interest in using omega-3 fats to treat bipolar disorder because low plasma omega-3 and high omega-6 levels are familial trait indicators for the condition (Sobczak *et al*., 2004). The findings of current investigations are varied. An open-label pilot trial found that taking 2000mg EPA/day reduced Hamilton Depression Rating Scale scores by 50% or more for 7 out of 10 bipolar disorder patients after one month. However, the ideal dose and duration of EPA treatment remain unknown (Osher *et al*., 2005). However, in patients with bipolar depression or rapid cycling bipolar disease, 6000 mg/day of ethyl-EPA had little benefit when compared to a liquid paraffin placebo (Keck et al., 2006). The study asked about the proper dosage and whether DHA should be used in treatments. Participants in the double-blind, randomized controlled trial (RCT) were not permitted to change the dosage of their mood stabilizer medications during the experiment, and the attrition rate was high (54%). Regardless of medication use, consuming 6200 mg EPA and 3400 mg DHA daily led to a much longer remission in bipolar illness than a placebo of olive oil esters, according to an earlier RCT (Stoll et al., 1999).

the connection between manic symptoms and necessary fatty acids. Even after adjusting for depression, there was a negative association between severe symptoms and free plasma arachidonic acid (an omega-6 fatty acid) and EPA in a small sample of patients with acute manic episodes. This study found a positive correlation between arachidonic acid: EPA ratios and the severity of depressive symptoms (Sublette *et al*., 2007). The study had a small sample size and difficulty recruiting patients with acute mania, leading to the possibility that the most severe symptoms were not included. As an adjuvant treatment for bipolar disease, 1000 and 2000 mg/day of ethyl-EPA significantly decreased depressed symptoms when compared to a paraffin oil placebo, according to a 12-week RCT. On the other hand, manic symptoms remained unchanged. The two ethyl-EPA dosages and the placebo group did not differ in terms of the degree of improvement or the severity of symptoms after 12 weeks. Twelve of the twenty-six placebo participants in this study needed to change their medication because of side effects or mental health concerns. (Sublette *et al*., 2007). Only 7 out of 24 participants on 1000 mg ethyl-EPA and 7 out of 25 on 2000 mg ethyl-EPA required medication adjustments, indicating that ethyl-EPA provided a better response than placebo. Both the treatment group (440 mg EPA and 240 mg DHA/day in addition to valproate) and the placebo group (olive oil and valproate) experienced a significant improvement in manic symptoms compared to baseline, according to a double-blind, placebo-controlled study involving 14 bipolar disorder patients (Chiu et al., 2005). There was no change in the severity of symptoms between the treatment and placebo groups over the four-week trial. Nevertheless, the omega-3 dosages were low in comparison to earlier research.

The relationship between omega-3 fatty acids and childhood bipolar illness is less well studied. New research has been published, though. After eight weeks, consuming 1290–4300 mg of EPA and DHA daily dramatically reduced symptoms in 16 pediatric patients in an open-label pilot study (Wozniak et al., 2007). However, the dosage of omega-3 used varied according on clinical judgment, and following the trial, both depressive and manic symptoms remained (Gracious *et al*., 2006). When compared to an olive oil placebo, flax oil, which includes omega-3 alpha-linolenic acid, did not improve mood in a large RCT of forty-four children with bipolar illness. Results were mixed due to the trial's duration (2–16 weeks), the usage of psychiatric drugs by certain youngsters, and the different dosages of flax oil (550–6000 mg alpha-linolenic acid/day).

**Depression**   
Depressive symptoms are associated with lower omega-3 status, especially in cases of severe or recurrent depression as opposed to milder or isolated episodes. When compared to controls, those with major depressive disorder showed significantly lower post-mortem DHA levels in their orbital frontal cortex (Astorg et al., 2008). The study had a tiny sample size and no information (Mcnamara *et al*., 2007) on participants' diet or omega-3 intake before death. The sample represented all patients with serious depressive disorders. Additionally, the intensity of the symptoms was unknown. There was no association between depression scores and plasma omega-6:omega-3 ratios, arachidonic acid:EPA, arachidonic acid:DHA, total omega-3, alpha-linolenic acid, EPA, or DHA levels, according to a study conducted on 192 community members (Appleton et al., 2008). This group suffered from mild to moderate depression despite having little omega-3 fatty acid intake. When it comes to severe depression or clinical settings, omega-3 status might be more important (Appleton *et al*., 2008). An older study indicated that post-myocardial infarction (MI) patients with depression had significantly higher plasma arachidonic acid:EPA ratios than those without depression (Schin *et al*., 2007). In a 2004 study, people with MI and unstable angina who also had significant depression had lower levels of total omega-3 and DHA in their plasma and higher ratios of omega-6:omega-3 fatty acids in their plasma (Frasure et al., 2004). Dietary intake was not evaluated in either study.

The association between depression and omega-3 fatty acid levels may be impacted by regional differences in diet and lifestyle. Despite the high consumption of fish and shellfish in Japan, there is no discernible relationship between dietary omega-3 intake and the prevalence of depression among those who live in the country. Adipose levels of omega-3 fatty acids do not correlate with depression in rural Greece, where depressive symptoms are uncommon (Murakami et al., 2008). The cuisine and way of life, however, are not comparable to those in the US or northern Europe (Miyake et al., 2006). Essential fatty acid metabolism may be disturbed by depression. Dietary omega-3 and erythrocyte omega-6 content did not significantly differ between 10 patients with major depressive disorder and 14 healthy controls (Mamalakis *et al*., 2008). However, patients had significantly lower erythrocyte membrane omega-3 levels (Edwards *et al*., 1998).

According to the study, more severe symptoms were linked to higher linoleic acid levels and lower DHA levels in the entire group. Dietary omega-3 was also associated with the severity of depression in the patient group. To precisely measure omega-3 intake, this study used a 7-day weighed food record (Appleton et al., 2006). Analyzing the correlation between erythrocyte omega-3 levels and clinical state while adjusting for dietary intake and determining whether the relationship changes based on whether major depressive disorder is present or not would be intriguing. Research design has made it difficult to interpret the contradictory findings of intervention trials on omega-3 as a therapy for depression. Appleton et al. (2007) state that there is limited empirical data to support the efficacy of omega-3 on depression. A meta-analysis of 12 trials on omega-3 fatty acids for treating depression found that most were small, short-term, employed different combinations and doses, and had heterogeneous samples. According to research, major depression may be more affected by omega-3 fatty acids than other forms of depression. A recent review by Lin and Su examined various EPA dosages, however it omitted any more recent trials than those by Appleton et al. When compared to a placebo, EPA levels of 4000 mg/day or above had significant antidepressant benefits, although doses of 2000 mg/day or less did not significantly differ (Grenyer *et al*., 2007).

Results have been mixed when omega-3 is used as a complement to antidepressant medication for major depressive disorder. Adults who took 600 mg of EPA and 2200 mg of DHA daily in a 16-week double-blind RCT did not see any improvement over those who took a placebo of olive oil. A twelve week of research combining DHA and EPA with olive oil found that treatment and placebo groups experienced increased mood, although there was no significant difference (Silvers *et al*., 2005). The severity of depression is unclear, as participants had been taking antidepressants for at least 2 months before the trial. A study found that a greater dose of EPA (2200mg DHA and 4400 mg EPA/day) significantly improved depression symptoms compared to an olive oil esters placebo across an 8-week double-blind RCT (Lin *et al*., 2007).

As a monotherapy, the advantages of omega-3 supplementation are still unknown. In comparison to ethyl-EPA and fluoxetine alone, a recent study showed significant improvement in major depressive disorder patients following 4 weeks of treatment with 1100 mg of ethyl-EPA and 20 mg of fluoxetine daily. All three groups had made significant progress from their baselines by the second week. There was no difference between the two monotherapies, however the improvement continued into weeks six and eight. A placebo group was not included due to ethical concerns (Jazayeri et al., 2008). This study, however, is the first to examine EPA monotherapy. A 20% dropout rate, a small sample size of 16 individuals per group, and the omission of standard deviations or standard errors for depression are among the issues baseline assessment. People with major depressive illness had twice as much erythrocyte DHA as the control group in a 36-person double-blind RCT of DHA monotherapy. The two groups' response rates to depression symptoms, however, were identical. The researchers postulated that either EPA was more beneficial, the 6-week treatment time was insufficient, the DHA amount was insufficient, or both EPA and DHA were required (Marangell *et al*., 2003).

Due to potential confounding factors, EPA and DHA combined as an omega-3 monotherapy have not demonstrated any discernible benefits. Both the treatment group (630 mg EPA and 850 mg DHA/day) and the control group (olive oil) experienced a substantial improvement in mood after 12 weeks as compared to baseline. The therapy group did not, however, significantly outperform the controls. Although there was a large sample size (n = 218) in this study (Rogers et al., 2008), the omega-3 dosage was less than in other investigations. Men with angina who were advised to eat more fish or take fish oil supplements participated in a randomized controlled experiment that was published in 2003 showed no change in mood compared to those who did not (Ness *et al*., 2003). This was even though the trial was substantially longer (6 months) and had a much bigger sample size (n = 377). The baseline fish intake was 'low' (less than twice a week), but there was no measurement of the omega-3 levels in the plasma or erythrocytes. Whether the baseline fish intake was sufficient for mood impacts is unknown. No particular amount or kind of fish was provided, although participants were encouraged to eat more fish or take fish oil supplements. Therefore, it's possible that the increased intake of fish oil was insufficient to have a therapeutic effect.

Omega-3 fatty acid dosage may not have a linear impact on symptoms of depression. Higher doses of omega-3 may lessen the effects of antidepressants, according to a double-blind dose-finding study conducted in patients with severe depressive disorder. After 12 weeks, 1000mg/day DHA was more effective than 2000 or 4000 mg/day, although the difference was insignificant (Mischoulon *et al*., 2008). However, there was no placebo arm in this trial. Therefore, all three trial groups may have spontaneous remission. An earlier and larger double-blind trial found evidence of a therapeutic window. A liquid paraffin placebo or 1000, 2000, or 4000 mg/day of ethyl-EPA were randomly allocated as supplemental treatments for adults with depression who were still receiving standard antidepressant therapy (Peet and Horrobin, 2005). Those taking 1000 mg/day of ethyl-EPA at 12 weeks showed a significant improvement in their baseline symptoms. But when compared to the placebo group, there was only marginal evidence that the 4000 mg/day group had improved more (Peet and Horrobin, 2005).

As with bipolar disorder, nothing is known about the connection between omega-3 fatty acids and childhood depression. Compared to placebos of olive oil and safflower oil, children with major depressive disorder showed significant improvement after 16 weeks when given 380 mg of EPA and 180 mg of DHA daily, according to a small double-blind RCT.

**Table 1. Review of selected studies on the psych-protective effects of substances contained in food.**

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| --- | --- | --- | --- |
| **Source** | **Sample** | **Preparation (Bacterial strain** | **Results** |
| Healthy persons | | | |
| Diop *et al*., 2008 | Healthy adults, Blinded, randomized study  Duration- 12 weeks | *Lactobacillus acidophilud* Rosella-52, *Bifidobacterium longum* Rosell-175 (3\*109 CFU/day) | For the first time, probiotic treatment has been demonstrated to lessen gastrointestinal issues in stressed individuals:   • gastrointestinal problems were significantly reduced in comparison to the placebo group;   • A notable decrease in the intensity of nausea and stomach pain brought on by stress. |
| Messaoudi *et al*.,2011 | Healthy adults. Double-blind, randomized study. N = 55 duration - 30 days | *Lactobacillus helveticus* R0052, *Bifidobacterium longum* R0175 (3 \* 109 CFU / day) | The first study to demonstrate that taking a psychobiotic reduces mental health problems brought on by stress:   • A decrease in anxiety symptoms as measured by the HSCL-90;   notable decrease in symptoms of depression and anxiety;   verified decrease in urine levels of the stress hormone cortisol;  • In the group of people with lower cortisol levels (less stressed), improvements in depression and anxiety scores on the PSS, HADS, and HSCL-90 scales. |
| **Depression** | | | |
| Wallace *et al*., 2020 | Depressed patients who were not taking antidepressants. Blinded, randomized study. N = 108 duration - 16 weeks | **Lactobacillus helveticus** R0052, *Bifidobacterium longum* R0175 (6 \* 109 CFU / day) | Scores on the evaluation scales decreased following four weeks of using the psychobiotic:    • Low mood as measured by the Montgomery-Asberg Depression Scale (MADRS) and the Quick List of Depressive Symptoms (QUIDS-SR16);    • Stress level as measured by the Sleep Quality Questionnaire (PSQI);   SHAPS, or the Scale of Perceived Pleasure, measures anhedonia.   Anxiety level as measured by the State and Trait Anxiety Inventory (STII) and the Generalized Anxiety Questionnaire (GAD-7). |
| Kazemi *et al*. 2019 | Depressed patients who were taking antidepressants (sertaline, escitalopram, fluixetine, or amitriptyline). RCT study.  N = 81 duration - 8 weeks | *Lactobacillus helveticus, Bifidobacterium longum* | • Lower Beck Depression Scale scores (in comparison to the prebiotic galactooligosaccharide or placebo groups).  • Reduce the ratio of kynurenine to tryptophan by increasing the generation of serotonin from tryptophan. |
| Rudzki *et al*. 2019 | Patients with depression. Double-blind RCT study. N = 60 duration - 8 weeks | SSRI+ *Lactobacillus plantarum* 299v (10 \* 109 CFU / day) | Adding probiotic microorganisms to SSRI treatment In MDD patients, Lactobacillus Plantarum 299v decreased KYN levels and enhanced cognitive function. When compared to the placebo group, the LP299v group's cognitive performance may have improved due in part to lower KYN levels. |
| Wallace *et al*. 2020 | Patients with depression. Double-blind RCT study. N = 10 duration - 8 weeks | *Lactobacillus helveticus Rosell*-52, *Bifidobacterium Longum Rosell*-175 13 \* 109 CFU) | Depression symptoms can be lessened with the use of probiotics. |
| Heirazadeh Rad *et al*.2020 | Patients with depression. RCT post hoc analysis. N = 78 duration - 8 weeks | *Lactobacillus helveticus* *Rosell*-52, *Bifidobacterium Longum Rosell*-175 (10 x 10 CFU) | Eight-week supplementation in depressed patients improved depressive symptoms, likely by increasing BDNF levels |

**Attention deficit hyperactivity disorder and behavioural problems**

There is limited research on the relationship between nutrition and conduct or learning difficulties, with most studies focussing on ADHD. A longitudinal study indicated that low DHA levels at birth were linked to childhood 'internal' problem behaviours such anxiety, depression, somatic complaints, and withdrawal at age. However, DHA status did not correlate with 'external' issue behaviors like violence or rule-breaking. Levels of long-chain polyunsaturated fatty acids at age 7 (Karabbendam *et al*., 2007) were not linked to problematic behaviours. This longitudinal study had drawbacks, such as relying on parental reports instead of an objective measure of behaviour and potential confounding by contextual factors including parenting styles and psychopathology (Karabbendam *et al*., 2007). Nonetheless, the study exemplifies "early programming" for subsequent disease.

People with ADHD might need more omega-3 fatty acids. ADHD and behavioral problems are associated with low essential fatty acid plasma levels (Rojas and Chan 2005). Despite having equal dietary levels of alpha-linolenic acid, DHA, and EPA as well as omega-3:omega-6 ratios, adolescents with ADHD had lower levels of total omega-3 fatty acid, DHA, and omega-3:omega-6 ratios in their erythrocyte membranes than controls, according to a small cross-sectional study (Colter et al., 2008). Hyperactivity, restlessness, oppositional behavior, and inattention are all negatively correlated with erythrocyte DHA content. People with ADHD ate more energy and macronutrients, as well as more trans, saturated, and total fat. Oppositional, hyperactive, and troublesome behaviors were associated with higher amounts of total and saturated fat (Colter *et al*., 2008). Individuals with ADHD may demand more energy, as evidenced by reduced body fat mass and percentage body fat mass compared to controls.

Vital fatty acid metabolism may be delayed in people with ADHD. Children with ADHD may have trouble converting 18-carbon chain fatty acids (linoleic and alpha-linolenic acids) into longer-chain polyunsaturated fatty acids (Chen et al., 2004). Children with and without ADHD did not differ in their dietary intakes of linoleic, alpha-linolenic, oleic (omega-9), or saturated fatty acids, nor in their intakes of energy, protein, total fat, or carbohydrates, according to the study. However, children with ADHD had significantly lower alpha-linolenic acid, arachidonic acid, DHA, total omega-3, and omega-3:omega-6 ratios in their erythrocyte membranes. ADHD individuals had lower erythrocyte alpha-linolenic acid levels compared to controls, despite equal food intakes. This suggests that children with ADHD may have difficulty absorbing, transferring, or storing this crucial fatty acid.

ADHD symptoms have been shown to improve regularly in open-label trials. For eight weeks, giving fish oil supplements to children with ADHD (2500 mg/10 kg body weight/day) significantly improved their attention and hyperactivity levels and decreased the ratios of arachidonic acid to EPA in their erythrocyte membranes (Germano et al., 2007). According to Sorgi et al. (2007), a small 8-week pilot study revealed notable improvements in conduct disorder, oppositional/defiant behavior, hyperactivity, inattention, and plasma phospholipids EPA and DHA. After three months, 30 children who were not receiving stimulant medication had significantly fewer symptoms of ADHD when 400 mg of flaxseed and 50 mg of vitamin C per day were taken, according to another open-label trial (Joshi et al., 2006). Flax oil increased EPA and DHA and significantly reduced arachidonic acid in the erythrocyte membrane. These studies did not analyze or control for dietary intake, making it unclear how diet affected plasma phospholipid profiles. The open-label design of these trials makes them susceptible to the placebo effect.

In comparison to a placebo of palm oil, administering fish and evening primrose oils, which contain the omega-6 fatty acid gamma-linolenic acid, to Australian children over a 15-week period resulted in a significant improvement in Conners ADHD and Global Index scores as well as hyperactivity, impulsiveness, oppositional behavior, and restlessness. A group that received fish and evening primrose oils, as well as vitamins and minerals, was part of a large RCT with 104 participants (Sinn et al., 2007). All three groups consumed fish, evening primrose oils, multivitamins, and minerals during the 15-week trial. While the group taking fish and evening primrose oils continued to improve on Conner's ADHD and Global Indices, restlessness, impulsiveness, and hyperactivity, the placebo group showed a significant improvement by week 15 (Sinn et al., 2007). Supplementing with micronutrients did not yield any more advantages than necessary fatty acids. There were no baseline tests to check for micronutrient or essential fatty acid deficiencies in the subjects. Serious symptoms that required medication or decreased adherence to the research procedure were among the reasons why trial participants left. Unlike medications, which only had benefits for four weeks, fish and evening primrose oils shown ongoing improvement after 15 weeks of treatment (Sinn *et al*., 2007).

Results from smaller, older RCTs were not always consistent. A combination of long-chain omega-3 and omega-6 fatty acids (480 mg DHA, 80 mg EPA, 40 mg arachidonic acid, and 96 mg gamma-linolenic acid) did not alleviate symptoms in a 4-month controlled pilot study with 50 participants (Stevens et al., 2003). Despite elevated EPA and DHA levels in the erythrocyte membrane, children with ADHD exhibit signs of essential fatty acid deficiency. Although they were not included in the analysis, the study assessed 3-day diet records at the start and finish of the intervention (Richardson and Puri 2002). According to a 12-week pilot study, giving kids omega-6 and omega-3 fatty acids (186 mg EPA, 480 mg DHA, 96 mg gamma-linolenic acid, 864 mg linoleic acid, and 42 mg arachidonic acid) significantly reduced ADHD symptoms. The study had limitations, including that none of the 41 children had been diagnosed with ADHD, and the fatty acid profiles of plasma and erythrocyte membranes were not evaluated. An RCT of 54 ADHD youngsters found that supplementing with 345 mg DHA/day for 4 months significantly boosted plasma DHA while decreasing EPA and arachidonic acid levels (Vogit *et al.,* 2001). At baseline and 4 months, the treatment and control groups had similar mean Child Behaviour Checklist and Conners' Rating Scale scores. The absence of benefit of DHA supplementation may indicate that youngsters require a longer term or a greater dose. Additionally, as the children continued their medicine throughout the research, DHA may not be useful as an additional treatment. The last two trials did not account for background omega-3 consumption.

Research on ADHD and omega-3 fatty acids has been focused on children and found some benefits. However, few research have been conducted on adults, making it premature to conclude. According to one study, DHA levels in adult erythrocyte membranes or blood did not correlate with the severity of ADHD symptoms (Young et al., 2004). It was unclear, though, if eating had an impact on the fatty acid profile, and the study did not take alcohol or tobacco usage into consideration. Despite not fasting when blood samples were taken, ADHD individuals exhibited lower levels of erythrocyte omega-3 and DHA as well as serum phospholipid levels of DHA, docosapentaenoic acid (omega-3), and omega-6 fatty acids (Young et al., 2004). Blood phospholipid profiles were analyzed in an RCT comparing fish oil, flax oil, and olive oil in individuals with ADHD; however, the study did not investigate whether the treatments had an impact on ADHD symptoms. An earlier study found that after 13 weeks, fish oil had no effect on the levels of hostility in young, healthy people. On the other hand, antagonism was considerably lower in individuals who received soybean oil as a placebo (Hamazaki et al., 1998). Both groups' levels of hostility stayed the same. Although dietary information was not included in this study, which was carried out in Japan, individuals there frequently consume large amounts of fish. Whether the detected levels of aggression were clinically significant or not was not evident.

Because of the small sample numbers and possible placebo effects on mood, studies using omega-3 formulations have been difficult to evaluate. Since certain oils might not be physiologically inactive, selecting an appropriate placebo is essential in RCTs (Parker et al., 2006). Linoleic acid, an omega-6 fatty acid and precursor to arachidonic acid, is found in olive oil, a common placebo. (Chiu et al., 2005; Gracious et al., 2006; Grenyer et al., 2007; Silver et al., 2005; Rogers et al., 2008; Stevens et al., 2003) Olive oil includes oleic acid, which is transformed into oleamide, a hallucinogenic substance. According to research, rapeseed and soybean oils, which are high in alpha-linolenic acid and linoleic (omega-6) fatty acids, have been employed as placebos. Sunola oil and corn oil also contain linoleic acid. The bioactive potential of olive oil esters, which were utilized as placebos in several experiments, remains unclear. Some studies do not specify which placebo was utilised. Choosing the right carrier is crucial. A study found that using food as a vehicle for oils was not optimal due to poor blinding. It's possible that the active component in seafood isn't DHA/EPA. Baseline dietary intakes are not routinely evaluated, or are not corrected for in analyses. Diets consumed during trials are frequently poorly controlled.

A British pilot investigation found that a diet with lower fat content than advised was linked to higher levels of anger, aggression, and sadness in healthy persons (Wells *et al*., 1998). The authors did not investigate the potential negative impact of a low fat diet, which accounts for only 25% of total calories, on mood.

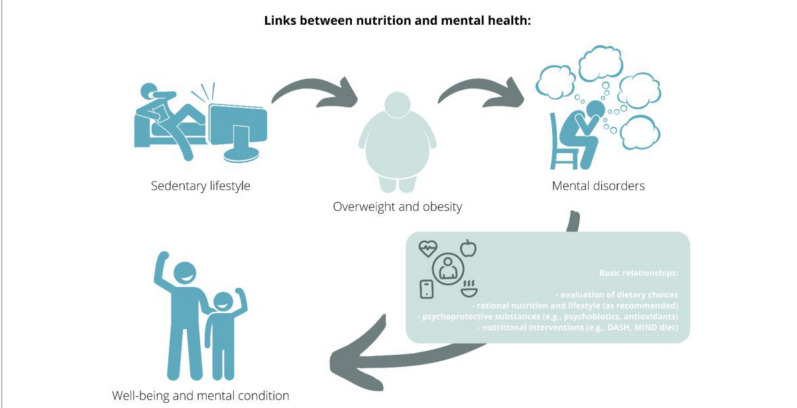


Fig 1. Link between mental health and nutrition

**Dietary protein and carbohydrate**

Compared to omega-3 fatty acids, the majority of the study on the effects of protein and carbs on mood and behavior was conducted in the 1970s and 1980s. By altering the brain's absorption of tryptophan, a precursor to serotonin, dietary protein and carbohydrate levels may have an effect on mood. According to the Wurtman theory, carbohydrate-rich meals release insulin, leading to peripheral tissues absorbing amino acids other than tryptophan (Bellisle *et al*., 1998).

The brain receives more tryptophan when the ratio of tryptophan to large neutral amino acids is higher (Benton *et al*., 1999). The Wurtman effect can be avoided by consuming only 5% of calories as protein (Young, 1991). Even high-carbohydrate foods include enough protein to counteract this effect. It's unclear whether plasma amino acid ratios correlate with human brain ratios (Markus *et al*., 1998). Few research have employed the Wurtman Hypothesis to change mood. High carbohydrate, low protein diets can considerably increase plasma tryptophan to neutral amino acid ratios. Diets strong in protein and low in carbohydrates have been shown to exacerbate depression in people who are highly sensitive to stress. Diets high in carbohydrates and low in protein have been shown to cause depression in people who are less stressed. The study did not evaluate baseline plasma amino acid ratios, so whether the test meals affected fasting amino acid profiles is unclear. The diets were only administered on the test day, therefore the results only show acute effects. A decade ago, a study on weight loss diets found that low tryptophan to large neutral amino acid plasma ratios were linked to poorer mood. However, the study did not control for protein and carbohydrate intake because participants were advised to follow a 'mixed' or 'vegetarian' diet. Mood may be influenced by variables other than the makeup of the diet and its metabolic effects. Dietary palatability is one instance. The general population is unlikely to ingest the abnormal food regimens needed to produce these effects. Other studies have used direct techniques, including supplementation to modify amino acid ratios, to raise plasma tryptophan levels. Research has yielded inconsistent findings, and young, healthy individuals may be more resistant to dietary modifications and supplementation. (Talbot *et al*., 2006).

Another significant component of carbs is refined sugar. Parents firmly believe that youngsters who consume sugar become hyperactive (Rojas and Chan 2005). Relatively recent empirical research has examined the effects of refined sugar on behavior and mood. The 1970s, studies found that removing and reintroducing sucrose to a child's diet led to greater hyperactivity. However, these studies lacked a control group and used double-blind methods. Removing sugar significantly alters a diet's overall makeup (Bellisle, 2004). In young people, consuming 100 g of sucrose did not affect mood, save for reduced anxiety after 4 hours. However, saccharin and water did cause mood changes Serum glucose levels were not assessed, although they were compared to fasting. In certain tests, an inappropriate placebo was used. Orange juice, which includes simple sugars, was used as a control in a study assessing sugar solutions. Hospitalized children with severe behavior disorders, attention deficit disorder, and anxiety showed no change in behavior.

According to controlled studies, even in kids with attention deficit disorder, sucrose does not make them hyperactive. After ingesting glucose or sucrose, some studies demonstrate a decrease in activity levels. Expectations from parents regarding how refined sugar affects kids' behavior are also important. Active youngsters require more energy than those who are sedentary, and increased sugar intake can contribute to these needs. Benton's review found that high sugar intake can have negative impacts on youngsters, but adults with low blood sugar may act aggressively (Benton, 2007). Benton postulated that low blood sugar in adults would not directly cause aggressive behavior, but rather be a sign of a serotonin deficiency. It is economical to remove added or refined sugar from children's diets, and there may be nutritional advantages as well, such as a decrease in obesity and dental cavities.

**Food additives**

The Feingold diet, which excludes salicylates, artificial food colourings, and artificial flavourings, is not effective in treating ADHD (Rojas and Chan 2005; Bellisle, 2004; Stevenson, 2006).In 2004, Schab and Trinh conducted a meta-analysis of 15 trials on using artificial food colours and flavours in hyperactivity. The study found heterogeneity in terms of dosage, colour types, ADHD diagnosis, and washout periods used in cross-over studies. The meta-analysis did not confirm whether children with formal hyperactivity diagnoses were more vulnerable to the effects of colours and tastes than those with informal or no diagnosis. However, Schab and Trinh found a robust connection between hues and tastes and hyperactivity. Earlier studies had methodological issues. Children who improved on a limited foods diet were evaluated and included in a double-blind cross-over randomized controlled trial (RCT) for children aged 3 to 12 with DSM III attention deficit disorder with hyperactivity. Although it is uncertain whether the extent of the change is clinically important, the psychologists' assessment of the children's behavior showed a significant change. The limited foods diet used to assess children was less strict than an exclusion diet because it included potentially triggering substances. For instance, the diet included bananas, which are strong in amines, another potentially irritating class of chemicals, and unidentified vegetables that might have contained salicylates. Chocolate (amines), pineapple juice (both amines and salicylates), and apricot juice (salicylates) were among the meals that were used to mask the experimental foods during the double-blind phase (Swain *et al*., 2004).

Although recent studies have improved in design, there are still issues. Artificial food coloring and the preservative sodium benzoate increased hyperactivity levels in 3- and 8- to 9-year-old children in the community, according to a recent double-blind RCT by McCann et al. The supplied doses were equivalent to the amount of additives found in 112-224 g of confectionery daily. The compounds studied were a mixture, so it's unclear whether one(s) caused hyperactivity or if they worked together. The study did not control children's meals or quantify their baseline additive intakes, which led to a great deal of variation in the findings. Equal levels of artificial coloring and preservatives were used in a previous double-blind controlled research, while a background diet devoid of these additives was not used. There was no difference between the treatment and placebo, according to objective assessment. During the placebo and treatment phases, there was a noticeable rise in hyperactivity, according to parental evaluations. The study group had a higher rate of hyperactivity than the overall population, which raised concerns about the research. Furthermore, the ADHD diagnosis was just provisional because the trial was done on 3-year-olds (McCann *et al*., 2007; Eigenmann *et al*., 2007).

According to McCann's trial, more nutritious foods may eventually be replaced by candy in children's meals. Children's diets may not need artificial food coloring and flavoring, but sodium benzoate is an essential preservative for food safety. Eliminating artificial coloring, flavoring, and preservatives from food intended for children should improve public health. However, a diet free of these ingredients may be too restrictive for hyperactive youngsters. Dietary treatment for hyperactivity may not be effective for all children due to unknown toxicities, pharmacological effects, or allergies. Before implementing a diet free of artificial food additives, it is important to establish tools to determine susceptibility to these substances. Additionally, there is little reason to assume that all additives in the Western diet are harmful (Benton, 2007).

**Mental health**

The NP with high intakes of calcium, β-carotene, vitamins A, D, K, and C, as well as dietary fiber, had a protective impact and an inverse relationship with psychological stress, depression, and anxiety after controlling for confounding variables. Additionally, there was a statistically significant negative correlation between depression, anxiety, and psychological stress and an NP that was high in essential amino acids, zinc, vitamin B-complex, phosphorus, selenium, and cholesterol. Vitamins A, D, K, B6, B12, and folate were also found to have protective benefits against anxiety in the INQ analysis. To show the intricate relationship between NPs and psychological problems and uncover its potential processes, more research using prospective designs and a bigger sample size is required.

**Micronutrients**

Many nutrients are believed to affect behavior and mood. The connection between micronutrients and mood, however, has received little empirical investigation. Benton's review did not address which micronutrients were crucial for behavior; instead, it focused on nutrition and violent and aggressive behavior in both adults and children, rather than mood.

Common symptoms and clinical indicators can result from vitamin deficiencies. Low mood has been associated with thiamine deficiency, and supplementation may improve mood in people with normal thiamine levels. It's unclear, though, if this is because of a pharmacological or physiological effect. Pellagra, a niacin deficiency, can lead to mental health issues. Depressive symptoms may be exacerbated by low folate and vitamin B6 levels. Bipolar disorder and depression have been associated with oxidative stress. Major depression may be brought on by insufficient antioxidant defenses, and depression itself may be a chronic inflammatory disease (Jacka and Berk, 2007; Pasco et al., 2008; Kaplan et al., 2007). Although no intervention studies have been done, low vitamin E levels may be associated with depression because of its antioxidant qualities.

Long-term deficiencies or psychological stress can impact brain growth and nutrition absorption, which can lead to mood disorders. Supplementing healthy young adults with a multivitamin enhanced self-reported mental health in women but not in men, according to a year-long double-blind RCT (Benton et al., 1995). Women who had higher serum levels of vitamin B6 and riboflavin reported feeling happier. There was no baseline vitamin status comparison between men and women, however the gender difference could not be explained (Ballin et al., 1992). Men with depressed symptoms consumed less folate than women, according to recent studies conducted on Japanese people. Compared to men in the first quartile of vitamin B12 intake, men in the second quartile experienced significantly lower rates of depressive symptoms.

Iron deficiency without anemia has been linked to depression in people using oral contraceptives. In a double-blind RCT, adolescent girls who received iron supplementation reported considerably higher levels of improvement in lassitude, concentration, and mood compared to the placebo group. Most of those who improved had hypoferremia at the start of the experiment and were normoferremic after the supplementation period. Selenium deficiency can negatively affect mood and anxiety. The time needed to dramatically change selenium levels in the central nervous system, the possible toxicity of excess selenium, and the difficulty in measuring dietary selenium intake have all been highlighted in investigations on selenium supplementation and depletion (Benton et al., 1991; Hawkes et al., 1996). Depression may also be caused by low calcium, magnesium, and zinc levels.

The connection between behavioral problems and micronutrients has been the subject of numerous empirical studies over the past five years. According to a case-control study, teens' ADHD, restlessness, oppositional, hyperactivity, inattention, cognitive problems, and problematic behaviors were all positively correlated with iron intake. The iron, thiamine, and riboflavin intakes of people with ADHD were significantly greater. According to a prior study, children with ADHD had higher serum iron, vitamin C, and iron intakes than children without ADHD. Iron deficiency did not affect either group. Higher levels in both studies (Chen et al., 2004; Germano et al., 2007), especially in Colter's group, suggest that people with ADHD may consume more calories, protein, and carbohydrates. Giving flax oil to children with ADHD raised lipid peroxidation in intervention studies, despite the addition of vitamin C as an antioxidant. However, ADHD symptoms significantly improved in this open-label research. Increased antioxidant levels may be necessary to minimise peroxidation. Adding a multivitamin and minerals did neither improve or decrease the efficacy of omega-3 fatty acids in treating ADHD in children, according to the previously published large RCT. Zinc sulfate, when added to methylphenidate, significantly decreased ADHD symptoms in children after 6 weeks when compared to methylphenidate alone, according to an earlier small double-blind RCT. Baseline micronutrient deficiencies were not taken into consideration in any of the three investigations (Joshi et al., 2006; Sinn and Bryan 2007; Akhondzadeh et al., 2004). Higher intakes than those provided by the intervention are probably needed by a clinical population. Treating blood vitamin deficiencies in young offenders has been shown to improve antisocial behavior, including aggression, according to an intervention trial conducted ten years ago.

According to Kaplan *et al*.,2007 there are insufficient controlled studies on micronutrients for a meta-analysis. Measuring micronutrient status and interpreting its impact on the brain poses practical challenges. According to research, brain micronutrient levels may not be effectively reflected by cerebrospinal fluid levels, plasma or serum nutrient levels, or trustworthy serum indicators of long-term micronutrient status. Since depression symptoms can lead to inadequate micronutrient consumption, which in turn encourages poor nutritional intake and deficiencies in other health behaviors, a relationship does not necessarily suggest cause and effect. This also holds true for research on omega-3 fatty acid intake and fish. Furthermore, especially in cases of disease, 'enough' nutritional intake can not translate into enough quantities of physiological tissue.

**Conclusion**

The complex relationship between diet and mental health in adolescent girls shows a critical and promising field. Pervasive throughout the evaluation is the importance of consuming correct, healthy foods that effectively reduce mental health dangers, including melancholy, nervousness, and behavioral issues. Micronutrients such as omega-3, zinc, iron, vitamins B6, B12, D, and folate have precise relations with psychological performance and might serve as both a protective step and treatment. Nutrient density, typical for the Mediterranean diet, reveals the model as favorable for mental health, while the Western diet has patients with unfavorable outcomes.

This evidence highlights the need to establish how not just single nutrients but macronutrient distribution and food patterns impact mental health. The INQ incorporates nutrients density to energy and allows for dietary assessment, facilitating intervention to improve diet. While current studies indicate great potential in natural treatments, the research science also points to some shortcomings: omega-3 fatty acids, for one, were the subject of varying results, and micronutrient effects are still not well-studied. It also holds true from cultural, regional and individual perspectives making it difficult for a one-size-fits-all strategy.

Mental illnesses are best tackled by incorporating nutrition into overall health interventions with backing from enlarged, duration, and control experiments. Efficient intervention requires multidisciplinary approaches that consider the health, education, and policy system to support and offer what the adolescent girls require and need to know for the change to come in diet. As this generation faces increasing mental illnesses, using the health-promoting role of diet provides a preventive approach in improving such peoples’ quality of life and well-being.

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