Research Article ISSN 2641-4317

International Journal of Psychiatry Research

Mental Health Therapy: Multivitamins

Ashok Kumar Dudi[1,2,3*]

[1]*National Career Service Centre for Differently Abled, Ranchi, India.*

[2]*Rehabilitation Council of India, New Delhi. India.*

[3]*Indian Association of Clinical Psychologists, Bhiwadi, Rajasthan, India.*

***Correspondence:**
Ashok Kumar Dudi, National Career Service Centre for Differently Abled, I.T.I. campus, Po- Hehal, Ranchi, Jharkhand, India, Tel: +91 7480880888.

Received: 08 May 2023; **Accepted:** 16 Jun 2023; **Published:** 21 Jun 2023

Citation: Ashok Kumar Dudi. Mental Health Therapy: Multivitamins. Int J Psychiatr Res. 2023; 6(3): 1-6.

ABSTRACT

Aims and Objectives: *This study promotes mental health multivitamin supplementation as an alternate treatment.*

Methodology: *Complementary therapy of multivitamin supplementation is designed using introspection and Google Scholar.*

Research and Results: *Nutritional deficiencies can affect the brain and neurological system. Oxidative stress damage DNA, proteins, dendrites, and mitochondria. Mental health is linked to gut-environment impacts via the brain-gut axis. Serotonin, GABA, epinephrine, norepinephrine, and dopaminergic pathways can be affected by psychiatric dietary therapy. N-acetylcystine (NAC), coenzyme Q10, and NAD+ may treat neurological and neuroprogressive illnesses. Retinoic acid induce embryonic stem cell neural lineage differentiation, which can damage adult hippocampal neurogenesis (AHN) and mental health. Multivitamins support brain and body function.*

Conclusion: *Psychiatric problems can be healed with multivitamins.*

Keywords

Mental Health, Multivitamins, Human intelligence.

Introduction

God made human beings according to his design, with perfect health and intelligence that surpasses all other species. Human intelligence has advanced technology and inventiveness, yet his competitive desire has made him angry and stressed. New psychotic illnesses have resulted. Simultaneous advances in medicine and technology have given us great new diagnostic and therapeutic capabilities. Although it calms an anxious mind, psychiatric patients actively resist pharmaceutical medicine therapy because it generates greater patient suffering as well as extrapyramidal side effects and fail to improve their well-being.

Ancient nutritional therapy is forgotten. Nutritional supplementation has shown the ability to heal mental diseases. Multivitamins boost mental wellness safely and effectively. In this mini narrative review, we'll first examine why mental disorders occur to determine how multivitamins benefit mental health. To determine how multivitamins affect brain physiology and neurotransmitters, one must know the cause of the psychopathology. Vitamins taken orally must reach the brain. The next section illustrates the brain-gut axis. Then vitamins' significance in brain energy production is described. Next, we define and classify mental health vitamins. Vitamin B along with many other vitamins are studied individually, along with their deficiency syndromes, benefits, sources, and dosages. Finally, its therapeutic potential and significance are examined. Lastly, this therapy's drawbacks are evaluated. First, we'll examine the author's approach.

Methods and tools

The author is a psychologist with twenty years of introspection experience. After recovering from a psychiatric issue (schizophrenia), the author self-analyzed through introspection methodology and used Google Scholar tools to help patients and mental health professionals design a novel supplementary therapy:

multivitamin supplementation. Review quality is impartial and transparent. Only high-quality systematic reviews that address the research issue are included.

Fundamental Mental Illnesses Cause

To treat psychopathology, we must identify its causes. Nutrition is one of many lifestyle factors, including social, psychological, and biological ones, that can contribute to psychological disorders. Nutrient deficiencies affect the central nervous system (CNS). Stress, neurotransmitter release, reuptake, metabolism, and energy sources may all influence psychiatric conditions. Over the past few decades, it has become clear that genotype affects chemical response through differences in cellular components like receptors, enzymes, and transporters [1].

Stress weakens brain immunity. Via the vagus nerve, the brain stem and supraschalemic nucleus (SCN) generate circadian rhythms. The hypothalamus controls nasal airflow rhythm and brain activity. Psychiatric disturbance begins with right nostril blockage, interrupting SCN output and lateralizing ultradian rhythms. This may create hypoxia to neuronal cells. Hypoxia may promote mitochondrial malfunction and neurodegeneration in the brain neuronal cells. Oxidative stress (OxS) from hypoxia reduces respiratory complex activity and damages the mitochondrial respiratory chain complex. Reactive oxygen species (ROS) and reactive nitrogen species (RNS) damage mitochondria. Mitochondrial malfunction causes neurodegeneration and mental disease [2].

Neuropsychiatric disorders cause neuroprogression (brain neural circuit loss) due to mitochondrial dysfunction, nitro-oxidative stress, nitrosative stress, and decreased antioxidant defenses. In these diseases, downregulation of Sirtuin 1 (SIRT) deactilase signaling may harm cellular antioxidant defenses and energy production due to its central role in adenosine triphosphate (ATP) production [3]. The hippocampus controls memory and emotion. Only two brain regions—the hippocampus being one—continue neurogenesis throughout adulthood. Adult hippocampus neurogenesis (AHN) creates new neurons. Hormones, neurotransmitters, cytokines, and growth factors control AHN [4]. Neurological diseases may worsen AHN [5]. Oxidative stress causes neurodegeneration, DNA, protein, and dendritic malfunction [6].

Multivitamin Supplements and Mental Health: Brain Physiology

Nutritional treatments typically alter specific biological reactions. Psychiatric disorders involve serotonin, GABA, epinephrine, norepinephrine, and dopaminergic pathways. Mitochondria, nicotinamide adenine dinucleotide phosphate (NADPH) oxidase, and substrate oxidase synthase (NOS) generate and release H_2O_2, which controls brain redox-sensitive signaling pathways and mechanisms. H_2O_2 dismuted from superoxide radicals produced by the mitochondrial electron transport chain regulates subsecond dynamic brain activity and neuromodulation, including dopamine, glutamate, GABA, and serotonin levels. [3]. GABA controls emotions, while serotonin, epinephrine, norepinephrine, and dopamine affect learning, memory, and motivation. [7-9]. Multiple vitamins may synergize mental health.

The Brain-Gut Axis: Multivitamins and Mental Wellness

Dietary changes improve mental health. Fortunately, gut microbiome research in mental health is growing rapidly, and "psychobiotics" are needed to treat mental health disorders. The brain and gut function together. The "gut-brain axis" connects the central nervous system and the gastrointestinal system. Mental illness is linked to gut-environment effects on complex human physiological genetic traits (like emotions). Environmental stressors alter nervous system activity. Neff (2020) [10] advocated that the central nervous system's (CNS) enteric nervous system sends gastric digestion and nutrient composition information to the brainstem's solitary nucleus. It mediates antibody-targeted tissues. One's gut flora affects nutrient and mineral absorption, enzyme, vitamin, and amino acid production, metabolism, immunity, and the neuroendocrine system. This could improve human psychology [10]. The gut microbiome breaks down vitamins and other nutrients eaten by mouth before transporting them to different brain and body regions. Vitamin mobility from the bloodstream benefits brain cells, neurons, leukocytes, and erythrocytes, among others [11].

Psychiatric patients should receive regular healthcare and make lifestyle changes like eating a healthy, fiber-rich diet, establishing regular routines, and exercising more. Multivitamins and other micronutrients may help treat schizophrenia and depression [12]. Nutritional and health product therapies for attention deficit hyperactivity disorder (ADHD), bipolar disorder (BD), cognitive impairment (CI), and mood dysregulation (MD) have shown promise [13].

Therapeutic Energy Generation

In contrast to chemical and structural similarities, cellular co-enzymes are what organize vitamins. As co-enzymes, they catabolize and anabolate cells. B vitamins help the brain produce energy, synthesis, and restoration of DNA and RNA, genomic and non-genomic methylation, signaling molecules, and neurochemicals. Vitamin B and action mechanisms play major roles in cellular processes. Vitamin B aids cell metabolism as a co-enzyme. Vitamin B, as a coenzyme, binds with apoenzyme protein to form halo-enzyme, which enhances diverse catabolic metabolic reactions that generate energy. Vitamins produce bioactive molecules through anabolic reactions [14].

Producing energy catabolically: Vitamin B's catabolic process in eukaryotic mitochondria generates energy. Vitamin B deficiency reduces catabolic processes and cell energy production. Vitamin B coenzymes (riboflavin, thiamine, pantothenic acid, and niacin) help the citric acid cycle by producing ATP, the cell's energy currency, through mitochondrial aerobic respiration. Thiamin, biotin, and vitamin B_{12} are substrates for the citric acid cycle in mitochondrial metabolism. Thiamin produces glucose, and biotin, or vitamin B_{12}, produces amino acids and fatty acids [15]. The citric acid cycle produces acetyl-CoA from substrate and

pantothenic acid. Eight enzymatic reactions turn probiotins, fats, and carbohydrates into acetyl-CoA, which generates reduced nicotinamide adenine dinucleotide - NAD (NADH) and reduced flavin adenine dinucleotide – FAD ($FADH_2$) energy. NADH and $FADH_2$ undergo enzymatic reactions with co-enzymes because FAD (B_2) and NAD (B_3) in the citric acid cycle pass through the electron transport chain and synthesize ATP [16,17].

Clinical Anabolism Procedure: Two interconnected enzymatic cycles—folate and methionine—generate ATP via the electron transport chain in the citric acid cycle. Oral folate intake from food enters the folate cycle and helps synthesize Deoxyribonucleic acid (DNA) and ribonucleic acid (RNA) [18]. The foliate cycle produces methionine cycle methyl groups. The methionine cycle uses homocystine as a starting point to produce S-adenosyl methionine (SAMe)[19]. Folate is useful, according to Wesson et al., and Alport et al. [20,21].

Essential Multivitamins for Mental Wellness

Multivitamins are organic compounds that the gut cannot produce and must be taken in small amounts through food. They are essential for normal physiological function. Stress increases adrenaline and other free-radical-producing compounds. Free radicals cause aging, immunity, and normal functioning. Vital nutrients fight free radicals.

Oral vitamins are needed. Normal physiological and neurological function requires thirteen vitamins. Four fat-soluble vitamins—A, D, E, and K—and nine water-soluble vitamins exist. Riboflavin (B_2), thiamine (B_1), niacin (B_3), pantothenic acid (B_5), pyridoxine (B_6), floatate (B_9), and cololamin (B_{12}) are water-soluble vitamins. Although plants produce the first eight vitamins, animal-derived food will indirectly contain vitamin B_{12} [22]. In the adaptive process, creatures effectively take vitamins directly from the environment, and the body ceases to have the potentiality for a clade-specific palette unless synthesis of vitamins to reduce cellular energy expenditure from within is needed for metabolism and oxidative stress.

Vitamins improve mood. Vitamins A, B_1, B_2, B_6, B_{12}, C, E, floate, biotin, and nicotinamide are taken daily. Riboflavin and pyridoxine boost mood. Thiamine improves mood and attention. Vitamins B_2 and B_6 calm anxiety. Vitamins B1 and B6 boost regulation. Vitamins B_1, B_2, B_6, E, and biotin help with mental health and sleep. These vitamins cause changes with 12 months of supplementation [23].

Vitamin B's

Vitamin B's metabolism and neurochemical synthesis affect brain function. Vitamin B's cross the bold brain barrier and choroid plexus using dedicated transport chains. Several homeostasis processes regulate brain functioning after this vitamin crosses particular cellular pathways [24].

Thiamine (Vitamin B_1)

Vitamin B1 activates neurotransmitters, fatty acids, aromatic amino acids, steroids, and nucleic acids and is a brain pentose phosphate system cofactor [25]. Thiamine helps acetycholine neurochemically structure cells, metabolism, and brain stimulation [26]. Vitamin B1 deficiency causes memory loss, irritability, sleeplessness, confusion, and emotional difficulties [25]. Severe deficiency causes ataxia, amnesia, confabulation, abnormal eye and motor movement, apathy, and neurodegeneration [27]. Whole grains, brown rice, potatoes, green vegetables, eggs, liver, pork, and pasta contain it. Add 1.2 mg daily (RDA).

Riboflavin (Vitamin B_2)

Riboflavin supplies flavoprotein coenzymes that limit cell enzymatic reactions. Folate, niacin, and vitamin B6 are created, transformed, and recycled; all heme proteins, including hemoglobin, P450 enzymes, nitric oxidase synthesis, and proteins; electron transport and oxygen storage. Flavoproteins assist brain lipids in using EFA [15]. Flavoproteins regulate thyroid hormones and iron absorption [15,28]. It increases endogenous glutathione redox cycle antioxidants. Riboflavin shortages hinder the process.

Brain dysfunction, personality change, tiredness, anemia, weakness, rashes, and riboflavin insufficiency can develop [15]. Legumes, leafy greens, kidneys, liver, mushrooms, yeast, and dairy contain much. Deficiency needs 1.3 mg per day (RDA).

Niacin (Vitamin B_3)

Nicotinamide adenine dinucleotide (NAD) and NAD phosphate (NADP) enzymes and pathways control peripheral and brain cell activity. Oxidation processes, deoxyribonucleic acid (DNA) metabolism and repair, intracellular calcium signaling energy production, antioxidant protection, and tetrahydrofolate variation through folate conversion [29]. Naicin deficiency can cause psychosis [30]. Psychotic symptoms, paranoia, anxiety, despair, memory loss, pellagrous insanity, and vertigo progression might result from naicin insufficiency. It can be supplemented with fish, meat, cereals, whole grains, nuts, legumes, and mushrooms; 16 mg of daily supplementation is advised (RDA).

Pantothenic Acid (Vitamin B_5)

Pantothenic acid synthesizes Coenzyme A (CoA). Pantothenic acid synthesizes cholesterol, phospholipids, fatty acids, and amino acids; maintains oxidative metabolism; and synthesizes numerous neurotransmitters and steroid hormones [16]. Pantothenic acid insufficiency can cause behavior changes, encephalopathy, and demyelination [16]. Whole-grain grains, beef, and broccoli are abundant in it; 5 mg daily is advised (RDA).

Pyridoxine, pyridoxal, or pyridoxamine (Vitamin B_6)

Vitamin B_6 synthesizes neurotransmitters such as dopamine, serotonin, GABA, noradrenaline, and melatonin through amino acid metabolism as a cofactor in the folate cycle. Brain glucose regulates genetic expression, transcription, and immunity [31,32]. Pyridoxal-5'-phosphate, which is involved in the one-carbon cycle and tryptophan metabolism, is down-regulated in severe inflammation [33]. Sakakeeny et al. [33] suggest that inflammation may promote cognitive impairment and dementia. Vitamin B_6 insufficiency causes cognitive decline, depression, dementia,

autonomic dysfunction, and brain abnormalities. Deficiency can cause melancholy, dementia, convulsions, irritability, cognitive decline, poor alertness, autonomic dysfunction, anemia, and anemia [32]. Potatoes, beans, bananas, fish, meat, and nuts are rich sources. Daily supplementation recommended is 1.5 mg (RDA).

Biotin (Vitamin B_7)

Biotin regulates glucose metabolism, gluconeogemesis lipogenesis, pancreatic beta-cell activity, hepatic glucose absorption, and insulin receptor transcription because the brain is sensitive to it [34]. Deficits may cause glucocorticoid dysfunction like Type II diabetes [34]. Deficits can produce hallucinations, sadness, lethargy, and seizures. Eggs, pork, liver, and leafy greens are sources; 30 mg per day RDA is required.

Folate (Vitamin B_9) and Cobolamin (Vitamin B_{12})

Depression patients had 25% lower blood folate levels, which may contribute to poor antidepressant treatment outcomes. Folate and folic acid are often used interchangeably; however, folate is a broad term for Vitamin B_9 in its many forms, whereas folic acid is made of THF, DHF, 5-MTHF, and 5,10-methylenetetrahydrofolate (5,10-MTHF). Folate is easier to absorb than folic acid for mental wellness. Folate is vitamin B_9, while folic acid is a nutritional supplement.

Folate is best since folic acid is useless. Methylfolate is three times higher than serum levels in the CNS and can penetrate the blood-brain barrier, unlike folate [35]. Genetic defects can reduce the body's ability to make methylfolate. The body makes methylfolate from folic acid. Folate deficiency is widespread in psychiatric illnesses and negatively linked with homocystine. Increase foliate to reduce red blood cells (RBC) plasma homocystine. Folate should be taken alongside vitamin B_{12} but at distinct times to avoid pancreatic enzymes interfering with folate absorption. Folate supplementation can help treat schizophrenia and depression.

Folate is assisted by Vitamin B_{12}. Vitamin B_{12} insufficiency traps folate in methyltetrahydro folate, causing a folate shortage [15]. Folate deficiency lowers genes, causing demyelination, shrinkage, and faulty action potential propagation [29]. Folate deficiency can lead to neuropsychiatric issues and anemia.

Tetrahydrobiopterin in the folate cycle converts amino acids into monoamine neurotransmitters (dopamine, serotonin, adrenaline, noradrenaline, melatonin, etc.) and nitric oxide [36]. Milk, eggs, chicken, yogurt, fish, and meat are high in vitamin B_{12}. Folate and vitamin B_{12} RDAs are 500 and 2.4 g, respectively.

Other Vitamins

Vitamin C

Vitamin C regulates brain neurotransmitter production and release. Vitamin C cofactors dopamine beta-hydroxylase to convert dopamine to noradrenaline. Vitamin C affects glutamatergic, dopaminergic, and synaptic acetylcholine and catecholamine release. Vitamin C (3 grams per day) synthesizes dopamine and norepinephrine. Vitamin C prevents glutamate excitotoxicity and brain antioxidation. Plasma and leucocyte levels show vitamin C storage and consumption to protect against vanadium damage [37].

Vitamin E (tocopherols and tocotrienols)

Vitamin E deficiency is linked to hypertensive vascular illnesses, peripheral neuropathy, cerebellar dysfunction, and cognitive deterioration. It causes oxidative stress in the CNS. Vitamin E (not selenium) affects iron-induced lipid peroxidation [38]. Vitamin E has a daily RDA of 1000 mg.

Vitamin D

Vitamin D deficiency can cause autism, depression, and dementia. Vitamin D supplementation's effects on depression are inconsistent [39]. However, mental illness patients on daily SSRIs had a higher risk of clinical fragility fracture due to falls and lower spine bone mineral density [40].

Mental Healthcare Possibilities

Healthy eating is great psychotherapy. Due to the brain's sensitivity to biochemical and physiologic changes, stomach blood oxygenation or diet quality can affect the brain. Lifestyle and nutrition can control psychiatric conditions by affecting psychological stress, neurotransmitter release, reuptake, and metabolism, or by providing material for neurotransmitter synthesis [1].

Under oxidative stress, hypoxia, inflammation, or mitochondrial insufficiency, cellular function and rebuilding may require increased nutrient intake. Vitamins, minerals, and other naturally occurring metabolites improve health and neurological symptoms.

Oral N-acetylcystine (NAC)

NAC manipulates cystine and generates hydrogen sulfide (H_2S). NAC protects and restores nerve cells from H_2S neurotoxicity. H_2S, generated by an elevated level of NAC in the cystol, can localize into mitochondria, activate ATP production by delivering electrons to mitochondrial electron transport chain (ETC), and lower reactive oxygen species (ROS) levels via its efficacious scavenging activity to improve ATP and ROS production by dysfunctional mitochondria [3].

Coenzyme Q_{10}

Mitoquinoine, a form of coenzyme Q_{10} (CoQ), can cross the blood-brain barrier and concentrate in mitochondria, improving mitochondrial function and reducing brain nitrooxidative stress. This optimizes mitochondrial activity even in bioenergetic malfunction [3].

NMN and NR

Nicotinamide adenine dinucleotide NAD^+ may help treat neurological and neuroprogressive disorders. Oral nicotinamide mononucleotide (NMN) nicotinamide riboside (NR) (NAD^+ precursor molecules) quickly raise NAD^+ levels in the body and mind. NAD^+-boosting supplements include nicotinamide. The hippocampus and hypothalamus benefit from increased brain NAD^+. NMN increases NAD^+, SIRT 1, and mitochondrial

respiration, which boosts brain and peripheral ATP production [23].

Pyridoxine, a coenzyme, makes serotonin, norepinephrine, and aminobutric acid. Dopamine and norepinephrine require ascorbic acid. Folate and vitamin B_{12} synthesize and degrade monoamine neurotransmitters. Riboflavin, nicotinamide, and thiamine start the brain's glucose metabolism.

Vitamin and mineral deficiencies can harm AHN and mental health, causing depression and spatial learning issues. Returning retinoic acid (RA) induces embryonic stem cell neuronal lineage differentiation in culture. Known metabolic targets for retinoid-induced genes [41]. The diet also controls AHN. Calorie intake, meal frequency, texture, and content affect AHN to modulate cognitive performance and mood. Returning RA to normal levels can improve AHN and mental health by reducing depressive behavior and spatial learning caused by vitamin imbalance [41].

Thus, blood-absorbed multivitamins can affect brain physiology. These findings reduce mitochondrial oxidative stress in neural circuits that process cognitive and emotional information [13]. Restoring brain cell mitochondrial function may reverse neurodegeneration.

Negative Outcomes of Multivitamins

Vitamins and minerals—hermetic, essential nutrients—keep the body healthy. Vitamin supplementation should be considered for hormesis. Hormesis occurs when minimal exposure to a toxic chemical reverses the desired outcome. Too much of a vitamin or mineral can cause hypervitaminosis, tissue mineralization, and electrolyte imbalance, but the body needs low daily doses. Homeostasis disruption causes a hormetic response [42]. Biochemical and physiological control mechanisms maintain internal stability and adapt to external stressors in living things.

Last but not least, multivitamin supplementation is merely a complementary therapy for mental health, and pharmaceutical medicine should not be abruptly stopped. A mental health professional must be in charge of multivitamin therapy.

Limitations

Introspective approaches are unrepeatable and biased, even with precautions. In large doses, all microminerals (trace elements) are toxic, and many are linked to cancer. Dietary advice for mood disorders is lacking in high-quality research. Supplemental therapy has slow effects, making it hard to measure.

Conclusion

The multifaceted effect that vitamins have on improving brain psychophysiology makes them a potentially remarkable healing and therapeutic tool for a wide range of mental health problems.

References

1. Harrison PJ. The current and potential impact of genetics and genomics on neuropsychopharmacology. European Neuropsychopharmacology. 2015; 25: 671-681.
2. Dudi AK. Genesis of Schizophrenia: An Introspective Review. Open Journal of Medical Psychology. 2023; 12: 71-81.
3. Morris G, Walder KR, Berk M, et al. The interplay between oxidative stress and bioenergetic failure in neuropsychiatric illnesses: can we explain it and can we treat it? Molecular biology reports. 2020; 47: 5587-5620.
4. Zhao C, Deng W, Gage FH. Mechanisms and functional implications of adult neurogenesis. Cell. 2008; 132: 645-660.
5. Mu Y, Gage FH. Adult hippocampal neurogenesis and its role in Alzheimer's disease. Molecular neurodegeneration. 2011; 6: 1-9.
6. Dean O, Giorlando F, Berk M. N-acetylcysteine in psychiatry: current therapeutic evidence and potential mechanisms of action. Journal of Psychiatry and Neuroscience. 2011; 36: 78-86.
7. Jenkins TA, Nguyen JCD, Polglaze KE, et al. Influence of tryptophan and serotonin on mood and cognition with a possible role of the gut-brain axis. Nutrients. 2016; 8: 56.
8. Moret C, Briley M. The importance of norepinephrine in depression. Neuropsychiatric disease and treatment. 2011; 7: 9-13.
9. Chiapponi C, Piras F, Fabrizio Piras, et al. GABA system in schizophrenia and mood disorders: a mini review on third-generation imaging studies. Frontiers in psychiatry. 2016; 7: 61.
10. Neff AS. Technical and theoretic limitations of the experimental evidence supporting a gut bacterial etiology in mental illness. Clinical Therapeutics. 2020; 42: e74-e81.
11. Wallimann T, Riek U, Möddel M. Intradialytic creatine supplementation: A scientific rationale for improving the health and quality of life of dialysis patients. Medical hypotheses. 2017; 99: 1-14.
12. Arroll MA, Wilder L, Neil J. Nutritional interventions for the adjunctive treatment of schizophrenia: a brief review. Nutrition journal. 2014; 13: 1-9.
13. Du J, Zhu M, Bao H, et al. The role of nutrients in protecting mitochondrial function and neurotransmitter signaling: implications for the treatment of depression, PTSD, and suicidal behaviors. Critical reviews in food science and nutrition. 2016; 56: 2560-2578.
14. McCORMICK DB. Bioorganic mechanisms important to coenzyme functions. Handbook of Vitamins, 4th ed.; Zempleni J, Rucker RB, McCormick DB, Suttie JW, Eds, 2007.
15. Rivlin RS. Keeping the young-elderly healthy: is it too late to improve our health through nutrition? The American journal of clinical nutrition. 2007; 86: 1572S-1576S.
16. Harris CB, Chowanadisai W, Mishchuk DO, et al. Dietary pyrroloquinoline quinone (PQQ) alters indicators of inflammation and mitochondrial-related metabolism in human subjects. The Journal of nutritional biochemistry. 2013; 24: 2076-2084.
17. Depeint F, Bruce WR, Shangari N, et al. Mitochondrial function and toxicity: role of the B vitamin family on mitochondrial energy metabolism. Chemico-biological interactions. 2006; 163: 94-112.
18. Crabtree MJ, Amy Tatham, Ashley B Hale, et al. Critical role for tetrahydrobiopterin recycling by dihydrofolate reductase in regulation of endothelial nitric-oxide synthase coupling:

relative importance of the de novo biopterin synthesis versus salvage pathways. Journal of Biological Chemistry. 2009; 284: 28128-28136.
19. García-Minguillán CJ, Fernandez JD, Santiago Ceruelo, et al. Riboflavin status modifies the effects of methylenetetrahydrofolate reductase (MTHFR) and methionine synthase reductase (MTRR) polymorphisms on homocysteine. Genes & nutrition. 2014; 9: 1-11.
20. Wesson VA, Levitt AJ, Joffe RT. Change in folate status with antidepressant treatment. Psychiatry research. 1994; 53: 313-322.
21. Govender K, Mocke G, Alport M. Video-imaged surf zone wave and roller structures and flow fields. Journal of Geophysical Research: Oceans. 2002; 107: 9-21.
22. Smith AG, Croft MT, Moulin M, et al. Plants need their vitamins too. Current opinion in plant biology. 2007; 10: 266-275.
23. Benton D, Fordy J, Haller J, The impact of long-term vitamin supplementation on cognitive functioning. Psychopharmacology. 1995; 117: 298-305.
24. Spector R, Johanson CE. The nexus of vitamin homeostasis and DNA synthesis and modification in mammalian brain. Molecular brain. 2014; 7: 1-9.
25. Kerns JC, Arundel C, Chawla LS. Thiamin deficiency in people with obesity. Advances in nutrition, 2015; 6: 147-153.
26. Hirsch JA, Parrott J. New considerations on the neuromodulatory role of thiamine. Pharmacology. 2012; 89: 111-116.
27. Bates CJ, Prentice A, Birch MC, et al. Dependence of blood indices of selenium and mercury on estimated fish intake in a national survey of British adults. Public health nutrition. 2007; 10: 508-517.
28. Powers HJ, Corfe B, Nakano E. Riboflavin in development and cell fate. Water Soluble Vitamins: Clinical Research and Future Application. 2012: 229-245.
29. Bailey LB. The rise and fall of blood folate in the United States emphasizes the need to identify all sources of folic acid. Oxford University Press. 2007; 528-530.
30. Miller CL, Dulay JR. The high-affinity niacin receptor HM74A is decreased in the anterior cingulate cortex of individuals with schizophrenia. Brain Research Bulletin. 2008; 77: 33-41.
31. Anitha M, Abraham PM, Paulose C. Striatal dopamine receptors modulate the expression of insulin receptor, IGF-1 and GLUT-3 in diabetic rats: effect of pyridoxine treatment. European journal of pharmacology. 2012; 696: 54-61.
32. Dakshinamurti S, Dakshinamurti K. Antihypertensive and neuroprotective actions of pyridoxine and its derivatives. Canadian Journal of Physiology and Pharmacology. 2015; 93: 1083-1090.
33. Sakakeeny L, Roubenoff R, Obin M, et al. Plasma pyridoxal-5-phosphate is inversely associated with systemic markers of inflammation in a population of US adults. The Journal of Nutrition. 2012; 142: 1280-1285.
34. Via M. The malnutrition of obesity: micronutrient deficiencies that promote diabetes. International Scholarly Research Notices. 2012.
35. Kelly RL. The three sides of a biface. American antiquity. 1988; 53: 717-734.
36. Sthal S, Genética y psiquiátrica. Stahl SM. Psicofarmacología Esencial de Sthal. Bases Neurocientíficas y Aplicaciones Prácticas. 2008.
37. Naylor GJ, Smith AH, Vanadium: a possible aetiological factor in manic depressive illness. Psychological Medicine. 1981; 11: 249-256.
38. Meydani M. Vitamin E requirement in relation to dietary fish oil and oxidative stress in elderly. Free radicals and aging. 1992: 411-418.
39. León C, F Alvarez-Lerma, S Ruiz-Santana, et al. Fungal colonization and/or infection in non-neutropenic critically ill patients: results of the EPCAN observational study. European Journal of Clinical Microbiology & Infectious Diseases. 2009; 28: 233-242.
40. Richards JB, Papaioannou A, Adachi JD, et al. Effect of selective serotonin reuptake inhibitors on the risk of fracture. Archives of Internal Medicine. 2007; 167: 188-194.
41. Crandall J, Sakai Y, Zhang J, et al. 13-cis-retinoic acid suppresses hippocampal cell division and hippocampal-dependent learning in mice. Proceedings of the National Academy of Sciences. 2004; 101: 5111-5116.
42. Hayes D. Nutritional hormesis. European Journal of Clinical Nutrition. 2007; 61: 147-159.