

Review Article

NUTRITIONAL GENOMICS: THE USE OF PERSONALIZED DIETS FOR MANAGING DEPRESSION.

ABSTRACT

Millions worldwide suffer from major depression, and recent studies have highlighted the effect of diet on mental health. However, research using nutritional genomics to create personalized diets for managing depression is still limited. This review explores the role of nutritional genomics in developing personalized dietary strategies for depression, focusing on key genetic markers. A comprehensive review of current literature, with a focus on studies that analyze the interplay between specific genes, nutrient intake, and depression was carried out. Databases like PubMed, Google Scholar, and Web of Science were searched using keywords like “nutritional genomics,” “personalized diets,” and “depression.” Relevant articles were also selected based on their contribution to how genetic variations affect dietary components linked to mental health. We identified several genetic markers that may influence how individuals with depression respond to specific nutrients, such as vitamin D, folate, and omega-3 fatty acids. These markers include variations in genes related to neurotransmitter pathways, inflammation, and neuroplasticity. This review highlights the potential of nutritional genomics as a tool for creating more individualized dietary plans for managing depression. With the use of genetic insights into dietary recommendations, healthcare providers could offer more targeted interventions.

Keywords: [Nutritional Genomics, Personalized diets, Depression, Mental Health, Genetic Markers].

1. INTRODUCTION

The success of the human genome project has influenced the application of genetic studies describing single-nucleotide polymorphism that have highlighted that varying genetic information to virtually every aspect of medicine, and nutrition science is not left out [1]. Nutritional genomics is a field of nutritional science that involves understanding an individual's genetic makeup and how they interact with the bioactive compounds of dietary sources to elicit phenotypic characteristics including treatment responses. It encompasses the understanding of the effects of dietary constituents on gene expression (nutrigenomics), impact of genetic variability on dietary responses (nutrigenetics), and nutritional epigenomics which describes how dietary sources influence epigenetic modifications [2,3]. Global genomic diversity and heterogeneity evidenced in genome-wide genes influence varying dietary responses among the human population. It has laid bare the defect in general dietary recommendations as nutritional benefits differ among individuals. This thus emphasizes the need for a more individualized focused approach to dietary recommendations and not the traditional one-size-fits-all approach. This approach called personalized, or precision nutrition/diet describes the consideration of individuals' genetic makeup in designing meal plans and allows for a more beneficial diet (4, 5, 6). Personalized diet or nutrition explores the interplay between genetic variants and metabolism considering how they influence health and dietary responses [7].

Mental health conditions are the most frequent causes of hospital visits; and depression is a common mental health disorder ranked as the 4th leading cause of disability worldwide with about 3.8% of the world population affected [8,9]. Studies have implicated

diet as a risk factor for mental health, some highlighting the neurobiological mechanisms modified by diet to predispose to mental disorders such as depression [10, 11]. Another study also points to the genetic risk of depression associated with low dietary intake of tryptophan corroborating the diet as casual evidence of depression [12]. It is only implicative that if diet can influence mental health disorders, then it can also be used to alleviate mental health burdens. The treatment of mental health disorders with psychotherapy and pharmacological therapies has been characterized by unresponsiveness among subgroups of patients. These approaches are also not readily available in low- and middle-income countries, necessitating the advent of new treatment strategies such as lifestyle modifications, particularly diet [13, 14]. Pieces of evidence point to the potential of managing mental health disorders particularly depression through dietary interventions [15, 16] demonstrated an effective management of depressive symptoms with a personalized diet approach among older adults in an observational cohort study with a concomitant improvement in gut microbiota diversity [17]. In a similar vein, an interventional study involving the use of molecular profiles of participants to recommend a personalized diet resulted in a significant improvement in clinical outcomes for people with severe depression [18]. Calabrese and colleagues also used a personalized ketogenic diet to alleviate the burden of depression in a retrospective study [19]. While these pieces of evidence point to the potential of personalized diets in the management of depression, sufficient data is needed to validate the interplay between genetic profiles and depression management with diets. Therefore, this review seeks to highlight the genetic modifications influenced by personalized diets in improving depressive symptoms through gene-diet interactions.

2. NUTRITIONAL GENOMICS AND DEPRESSION

The development of several neuropsychiatric conditions like depression is reported to be greatly affected by the relationship between genes and diet. Studies have reported that what we eat influences our gene's response in our mental health, and this association may further be exploited to provide solutions for optimal mental health [20]. For the fact that several findings have demonstrated the association between nutritional abnormalities and increased psychological distress, the discipline of nutritional genomics helps in better knowledge of psychological disorders like depression [21]. According to Paans *et al.* [22] our mental health is affected by what we eat. Consequently, an individual's psychological state may influence their food preferences that may further affect their mental state [22]. A study by Gezahegn *et al.* [23] observed that people going through depression are prone to uncontrollable weight loss and nutritional deficiencies.

Polymorphism in some genes including brain-derived neurotrophic factor (BDNF), oxytocin receptor (OXTR), nuclear receptor subfamily 3 group C member 1 (NRC31), sodium dependent serotonin transporter (SLC6A4), FK506 binding protein 5 gene (FKBP5), spindle and kinetochore-associated complex subunit 2 (SKA2) are believed to contribute to development of depression [24]. According to Park *et al.* [25] individual phenotypes are influenced by genetic polymorphisms like single nucleotide polymorphisms (SNPs) that may predispose to variability of the development of certain diseases.

The *SLC 6A4* gene encodes the serotonin transporter protein (5HTT), which mediates serotonergic signaling as well as serotonin concentration [26]. The *HTR2A* gene on the other hand encodes the G-protein-coupled receptor (5-HT2A) that is involved in postsynaptic serotonin signaling and is targeted by antidepressants [27]. Some evidence suggests that nutritional supplementation with Tryptophan (TRP) influences an individual's emotional state through interacting with the central nervous system [28]. As a result, tryptophan rich diet may help in alleviating depression. Depression is also associated with low concentration of BDNF [29]. Single nucleotide polymorphisms (SNPs) in the *BDNF* gene may be a major depression causing factor. The most prevalent SNP of *BDNF* gene is Val66Met (rs6265) in which there is a replacement of valine/methionine which affects protein sorting and availability in the synaptic gap [30].

3. KEY NUTRIENTS INFLUENCING DEPRESSION

The effect of a mother's diet on a child's mental health has prompted the assumption that depression has a link to diet [31]. A study observed that the quality of what we eat as well as dietary patterns in different regions may contribute to depression [32]. For instance, a study showed that those who consume more grains and vegetables tend to have less severe depression as these foods may contain several essential vitamins and mineral elements [33].

As a cofactor vitamin B6 plays a role in depression by affecting neurotransmission in the brain [34, 35]. Levels of vitamin B6 among people going through depression have been observed to be lower compared to individuals in good mental health. For example, the active vitamin B6 form Pyridoxal phosphate, was found to be less in depressed individuals. Similarly, another study observed an increase in depression with a corresponding lowered vitamin B6 concentration [36].

Numerous studies have shown that lack of vitamin B12 in diet may be a contributor to depression [37]. Vitamin B12 role in depression may be because of its association with several neurotransmission systems. These connections may cause atrophy, neuronal death, and reduced neurotransmitter communication, which could result in depression. [38, 39]. A 6-year study in the elderly, reported no correlation between vitamin B12 levels and depression [40, 41]. Furthermore, there is a reported link between depression and vitamin D given its crucial role in serotonin formation [42]. The tryptophan hydroxylase 2 gene, which is involved in serotonin synthesis and is therefore thought to manage depression by maintaining serotonin levels, is stimulated by vitamin D, which regulates serotonin production in the brain. [43]. This link between depression and vitamin D deficiency may be exacerbated by increased free radicals and calcium ions in neuronal cells. Vitamin D supplementation has also been shown to lower depression and raise serotonin levels in the blood in those suffering from mild to severe depression. [44].

Omega-3 fatty acids are essential for proper brain function because of their impact on neuroinflammation, neurogenesis, and neurotransmission [45, 46]. Depression is directly impacted by several omega-3 fatty acids, including eicosapentaenoic (EPA) and docosahexaenoic (DAE) acids. They are further usually advised for the treatment of depressive disorders due to their function in depression. [47, 48]. Low levels of omega-3 fatty acids have been shown to exacerbate depression symptoms [49]. A study [49] suggests that consuming 1g of omega-3 fatty acids daily with an EPA concentration of 60.0% or more will probably help with depression.

Additionally, Due to its involvement in the production of several neurotransmitters, such as dopamine, adrenaline, and serotonin [50, 51], folic acid helps prevent depression by making antidepressant medications more effective [52]. When folic acid levels are low, antidepressant medication effectiveness is decreased. A folic acid deficit is linked to aberrant levels of the neurotransmitters mentioned above, which leads to a decline in neurochemistry and the development of depression [53, 54].

4. GENETIC VARIABILITY AND NUTRIENT METABOLISMS IN DEPRESSION

Current advances in nutrition research are focused on exploring the differences in the way different people react to certain nutrients or diet which may be due to a genetic variant modulating on a specific phenotype [55]. For instance, genetic makeup may determine which group of people will be more sensitive to certain nutrients that may pose a danger of developing mental conditions like depression [56]. Numerous genome-wide association studies have demonstrated that an individual's genotype influences serum metabolite levels and more accurately predicts cognitive function. For example, in certain people, variations in the transcobalamin (*TCN2*) gene may result in decreased quantities of vitamin B12 intermediates [57]. As a result of the foregoing, transmembrane proteins

such as IF, transcobalamin II, cubilin, and haptocorrin are considered for the relationship between brain health and genotype [58].

Recent studies [59, 60] have further shown evidence that suggests the link between Methylene tetrahydrofolate reductase (MTHFR) C677T variants and depression. A positive correlation between depression and TT/CT genotypes among individuals was reported [60], which involved 402 depressed subjects and 600 controls. Another study found depression to be lowered in individuals with MTHFR TT genotypes and predicted greater treatment response with 50 mg citalopram. Additionally, two other studies using large cohorts found no association of the MTHFR C677T polymorphism to disease incidence [61, 62]. However, these studies did not use indices of severity or progression to predict the interplay between MTHFR alleles and mood [62].

Abnormal dietary polyunsaturated fatty acid (PUFA) levels also are associated with depression. For example, blood levels of PUFA including docosahexaenoic acid (DHA), eicosapentaenoic (EPA) and arachidonic acid (ARA) have been observed in people suffering from depression [63]. Genetic variation in fatty acid desaturase 1 and 2 gene (*FADS1* and *FADS2*) is also known to be associated with dietary differences in the fatty acids, pointing to the involvement of single nucleotide polymorphisms in depression [64].

5. GENE-DIET INTERACTIONS IN DEPRESSION

Gene-Diet Interactions refer to the ways in which a person's unique genetic makeup can influence how they respond to different dietary components; this can affect their mental health especially about depression [65]. Consequently, nutritional genomics has increasingly embraced studying these interactions as a way of developing personalized diets. Up-to-date research has demonstrated some genetic variations may act as risk factors for depression. For instance, a study linked polymorphisms in genes and risk of depression like 5-HTTLPR (serotonin transporter gene), and these specific versions of genes will likely respond differently to nutrients that alter neurotransmitter action [66].

Specific variants significantly associated with depression include Apolipoprotein E (APOE), piccolo presynaptic cytomatrix protein (PCLO), methylenetetrahydrofolate reductase (MTHFR), translocase of outer mitochondrial membrane 40 homolog (TOMM40), guanine nucleotide binding protein (G protein) beta polypeptide 3 (GNB3) [67], and solute carrier family 6 (neurotransmitter transporter) member 4 (SLC6A4) are in fact significantly associated with MDD [68, 69].

- **Serotonin Transporter Gene (SLC6A4) and Folate**

The SLC6A4 gene encodes the serotonin transporter. This gene is crucial for the reuptake of serotonin, a neurotransmitter linked to mood regulation. There are variations in this gene particularly 5-HTTLPR polymorphisms that have been associated with varying responses to dietary folate (vitamin B9) [70]. Serotonin synthesis depends on folate which is one type of neurotransmitter. Depressed individuals have also been reported to often have low levels of serum folate and those who have types of SLC6A4 gene can therefore be more prone to benefit from taking folic acid supplements [71, 72].

- **BDNF Gene and Omega-3 Fatty Acids**

The gene Brain-Derived Neurotrophic (BDNF) Factor is responsible for neuroplasticity, which is the capacity of the brain to adapt and self-adjust. The Val66Met variant in this gene can impact how individuals respond to omega-3 fatty acids, which are popular for their anti-inflammatory actions on the brain [73]. Among uses, Omega3 fatty acids may support BDNF expression, hence boosting mood and cognitive function. Thus, some BDNF genotypes may be associated with a better response to omega 3 supplementation in treating depression [74, 75].

- **MTHFR Gene and Homocysteine Metabolism**

The Methylene tetrahydrofolate (MTHFR) Reductase gene is critical for the metabolism of homocysteine, an amino acid linked to cardiovascular and neurological health. The C677T variation in the MTHFR gene can cause elevated homocysteine levels that are linked to depression [76]. The MTHFR enzyme synthesized donates a methyl group using 5- methyltetrahydrofolate as a cofactor to convert homocysteine to methionine

thus contributing to 1-carbon metabolism. According to more recent studies, people with the TT genotype may need more folate, and this genetic variation may affect how likely they are to develop vascular and neoplastic disorders as well as neural tube abnormalities (NTD) [77]. Folate and vitamin B12 are important in reducing homocysteine levels. Individuals with the C677T variant may benefit from increased intake of these vitamins, potentially reducing the risk or severity of depression [78, 79].

- **COMT Gene and Diet-Induced Dopamine Regulation**

The COMT gene, Catechol-O-Methyltransferase, that degrades dopamine, a neurotransmitter, has been found to be essential for mood and motivation. One such polymorphism, Val158Met affects the action of this enzyme resulting in different levels of dopamine in the prefrontal cortex [80]. Diets high in phenylalanine and tyrosine which are amino acids forerunners of dopamine may cross interact with COMT variants affecting mood regulation. They may also respond differently to these nutrients depending on whether they carry Met allele (which indicates reduced COMT activity leading to increased dopamine levels) or Val allele [81].

- **APOE Gene and Dietary Fats**

The primary lipid-carrying protein produced in the central nervous system (CNS), APOE is involved in lipid metabolism in the brain and throughout the body [82]. Apolipoprotein E (APOE) gene, a well-studied gene in lipometabolism, is also associated with Alzheimer's disease. Seneff (2009) pointed out that $\epsilon 4$ allele increases the risk of depression largely in a high fat diet context. Humans have three isoforms of APOE, namely $\epsilon 2$, $\epsilon 3$, and $\epsilon 4$, that are distinguished by just one or two single nucleotide polymorphisms [83]. APOE $\epsilon 4$ allele may worsen depressive symptoms if one consumes diets with too much saturated fats. However, it is opined that omega-3 fatty acids enriched diets including polyunsaturated fats lowers this risk [84].

5.1 Nutritional Influences on Gene Expression

Dietary factors can alter gene expression, which affects neuroplasticity, oxidative stress, and inflammatory pathways—all of which are connected to depression. Several nutrients, including zinc, fatty acids, certain sterols, and the metabolites of vitamins A and D, have a direct impact on transcription [85]. Dietary fiber components may indirectly influence gene expression through changes in mechanical stimulation, hormonal signaling, and metabolites produced by the gut microbiota [86]. Examples include substances that have been demonstrated to affect the expression of genes involved in mood regulation, such as vitamin D, folate, and omega-3 fatty acids. It has also been demonstrated that a few other dietary variables and lifestyle choices, including fasting, obesity, and diabetes mellitus (DM), significantly impact the baseline activity of oxidative enzymes in living things [87]. The metabolizing enzymes exhibit fluid pleomorphism, allowing them to adapt to any genetic modifications resulting from interactions between our genes and nutrition. All of this points to the possibility of customizing dietary therapies for depression management based on a person's genetic composition [88]. Emerging evidence further suggests that personalized nutrition, guided by genetic information, can lead to more effective management of depression. Furthermore, it is generally recognized that depression and malnutrition are associated; yet there is still a lack of information regarding accepted methods for assessing a patient's nutritional state, making it more challenging to draw conclusions regarding deficits [89]. Observational studies of depression patients who have undergone nutritional assessment are rare, even though clinical practice now recommends supplementation intake to supply certain common deficiencies, primarily omega-3 polyunsaturated fatty acid (ω -3 PUFA) and vitamin D [90].

Combining the effects of various genotypes on the metabolism of certain nutrients with a few components of human diets, most notably the Modern Western Diet (MWD), can lead to harmful gene-diet interactions [91]. The amount or activity of enzymes that synthesize or catabolize nutrients can be impacted by these interactions because they alter the expression of genes linked to metabolism [92]. In the end, this could affect clinical phenotypes, such as human disease as well as molecular phenotypes and further even depression. Other notable challenges remain: the complexity of gene-diet

interactions, the need for more extensive and diverse population studies, and the ethical considerations surrounding genetic testing for personalized nutrition [93].

6. METHODOLOGY

Review Criteria: A literature search on nutritional genomics was done using several databases, including Web of Science, BioMed Central, PubMed, Science Direct, Springer, and National Center for Biotechnology Information (NCBI). The Use of Personalized Diets in Managing Depression” to determine the current state of nutrigenomics research in managing depression worldwide. The following search phrases were utilized in the study: Key Genes, diet-gene interaction, nutrigenetics, nutrigenomics Africa, nutrigenomics worldwide, evaluation of personalized diet approaches, nutritional interventions, tailoring diets to genetic profiles and gene-environment interaction. To find more possible citations, we also looked through each study's bibliography. While there were plenty of publications on diet-gene interaction in these databases, there weren't many on nutrigenomics research that focused on using personalized diet for managing mental health, particularly depression. Other examined areas include the fields of dietary guidance, deficient illnesses, diet supplementation, and food-gene interaction.

Data Collection: Search terms that were combined to locate publications released between January 2000 and August 2024 were "key genes and diet in depression disorder" OR "major depression" AND "gene candidate," or "major depressive disorder" OR "major depression" AND "polymorphism." We looked at full texts, abstracts, and titles in the order that they qualified; we verified the information twice to ensure accuracy.

Analysis Techniques: The key findings of each study included, as well as the extracted data were used to create a descriptive and tabular synthesis (Fig. 1).

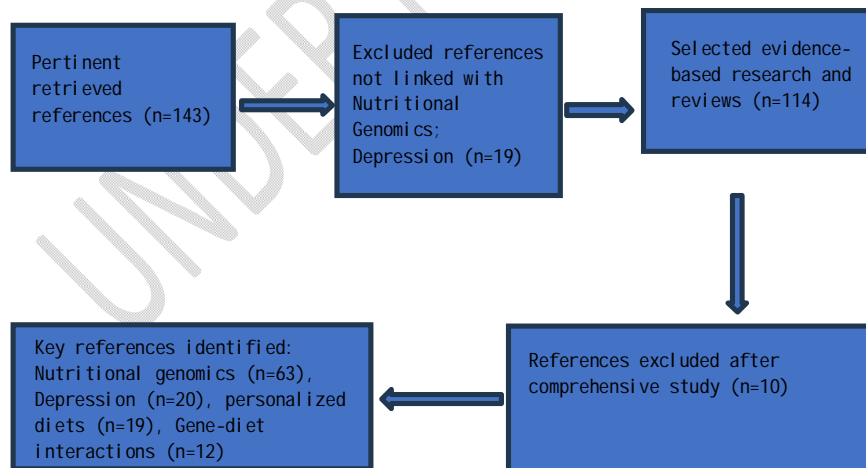


Figure 1: Selection of the research articles based on the review topic.

7. EVALUATION OF PERSONALIZED DIET APPROACHES

Personalized diet approaches, informed by an individual's genetic makeup, are a growing necessity of study aimed at optimizing mental health interventions and could even be modified to specifically manage depression [94]. Evaluating these strategies involves determining how effective, feasible and sustainable it is to tailor nutrition interventions to individuals' genetic profiles. Consequently, these personalized dietary interventions have been shown to be more successful than generalized approaches, particularly in relation to handling depression [95]. For instance, it has been discovered that people with specific genetic variations linked to the metabolism of omega-3 fatty acids benefit more from diets high in these nutrients than people without such variants (Figure 2). This is in line with research showing that neuroinflammation and neurotransmitter function are both significantly influenced by omega-3 fatty acids, contributing to the pathophysiology of depression [96]. The recommended personalized diet plan (Table 1) is designed to help manage depression by considering specific individual genetic profiles, while (Figure 2) illustrates the effectiveness of dietary interventions based on key gene variants related to depression; these interventions are tailored to optimize mental health outcomes.

Table 1: Identification of key genes linked to certain dietary recommendation

Key Gene	Dietary Recommendation
SLC6A4 (Serotonin Transporter)	Folate supplementation
BDNF (Brain-Derived Neurotrophic Factor)	Omega-3 supplementation
MTHFR (Methylenetetrahydrofolate Reductase)	Folate and Vitamin B12
COMT (Catechol-O-Methyltransferase)	Diet rich in phenylalanine and tyrosine
APOE (Apolipoprotein E)	Diets low in saturated fats, high in unsaturated fats

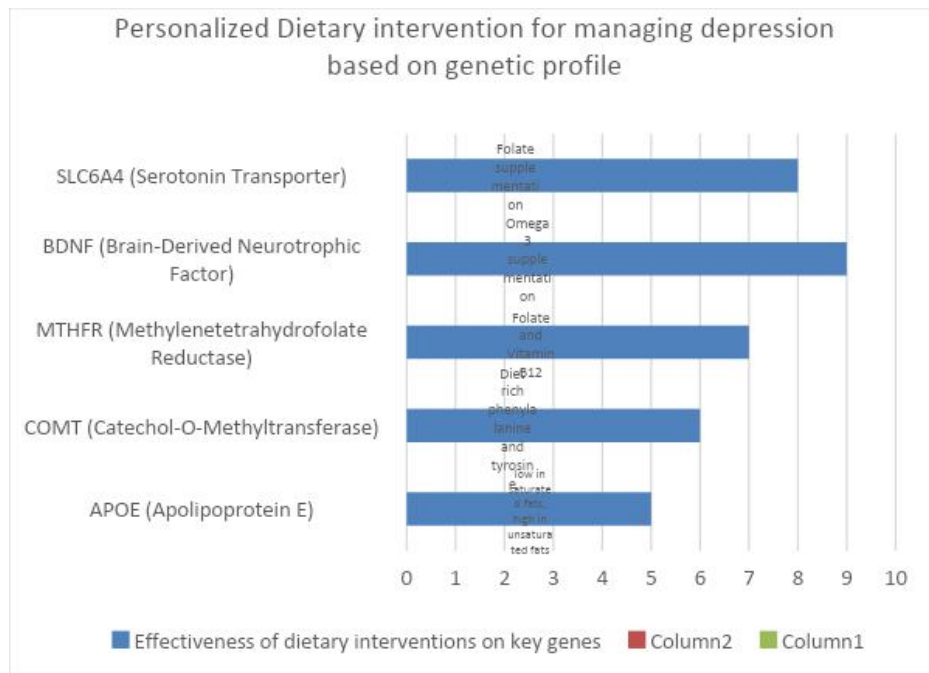


Figure 2: Impact of Personalized Diet Plans on Depression Management Based on Genetic Profiles of Key Genes (SLC6A4, BDNF, MTHFR, COMT, APOE)

Practicality and Implementation

Although there are many potential advantages to personalized diets, there are practical obstacles that should be addressed before these approaches can be widely applied. One primary challenge is the cost and availability of genetic testing, which is still a barrier, as well as the need for specialized knowledge not yet common in clinical settings for data interpretation [97]. Nevertheless, technological advancements and the declining cost of genetic testing provide hope that personalized diets may soon become a more viable option soon [98].

Long-Term Benefits and Risks

The long-term benefits of personalized diets are promising, with potential for even more sustainable and effective management of depression. A person's genetic profile could be used to customize dietary suggestions for even better mental health outcomes. This could also help address the underlying biological pathways that contribute to depression. However, there are also potential risks, such as the over-reliance on genetic data that does not take into consideration the complex interplay between environment, lifestyle and other variables which affects mental health [99]. Future research addressing the existing gaps in our knowledge between genes and diets will be critical to the management of depression through tailored diet methods. As more data becomes available, it will be possible to create more precise and individualized nutritional recommendations that can be incorporated into clinical practice and provide hope to individuals with depression [100].

8. DISCUSSION

This review article highlights the important role that nutritional genomics plays in managing depression. Depression is a complex disorder, which has been treated through drug therapy and counseling [101]. However, these approaches don't always include the impact that nutrition might have on mental health, or the role that genetic variability can have in determining how rapidly patients with depression respond to

specific nutrients. Thus, we noted that some nutrients—like polyphenols, omega-3 fatty acids, B vitamins, and folate—can help reduce depression symptoms when they are related to a person's genetic profile [102]. This approach does not only provide a more exact way towards dealing with depression but also stresses the necessity of incorporating nutrition into mental health care services. These kinds of nutrients are highly reliant on genetic polymorphisms i.e. certain genes responsible for activities such as neurotransmitter metabolism (serotonin, dopamine) or inflammatory pathways [103]. Persons with different MTHFR gene variations could benefit from higher doses of folic acid or using supplements to improve neurotransmitter synthesis thereby potentially reducing depressive symptoms [104]. Similarly, the COMT gene, which affects dopamine metabolism, could influence the effectiveness of certain dietary interventions for boosting mood. By understanding these genetic nuances, healthcare providers can tailor dietary recommendations to enhance treatment efficacy and reduce risk of the adverse effects, thereby moving closer to the ideal of precision medicine in mental health care [105].

Practical Applications

The possibilities of these findings extend to clinical practice, where nutritional genomics could significantly impact the approach to managing depression. Mental health practitioners can begin including dietary evaluations in their routine checks, evaluations, which will improve the general and better understanding of how nutrition affects mental health [106]. By identifying genes, nutrition experts can recommend personalized food plans that can supplement some traditional treatment options like pharmacotherapy and psychotherapy [107]. For example, those with depression who have a hereditary tendency to low levels of omega-3 fatty acids may be recommended to take supplements or increase their diet of foods high in omega-3 fatty acids, such as oily fish. Moreover, including customized diets for therapeutic purposes could potentially function as an affordable and easily accessible supplement to conventional remedies [108]. These nutritional practices are also a good option for patients who don't respond well to medication or have severe side effects because they typically do not have side effects that significantly affect users and are easy to implement into any healthcare delivery system [109]. It could also serve as a much more progressive way by empowering the patient and including them in the healing process, which may lead to better outcomes, through personalized diet.

Other practical applications involve educating patients about the connection between nutrition and mental health. By encouraging the understanding of how their genetic makeup can influence their response to certain foods, patients may become even more motivated to modify their diets in ways that promote their mental well-being [110]. This educational aspect is essential to promote long-term adherence to personalized dietary recommendations and ensuring that patients can make knowledgeable decisions regarding their health. This application could also help the healthcare system by providing alternative therapies and lowering the dependency on pharmaceutical treatments [111].

Future Directions

Nutritional genomics offers several promising areas for future research, and there is a need for larger clinical trials to establish the effect of personalized diets in treating depression across even diverse populations. It is crucial to recognize the challenges and areas requiring further exploration. One key area for future research is the need to examine the long-term effects of personalized dietary interventions on depression. Such studies should include diverse populations to determine the generalizability of findings across different genetic backgrounds and environmental contexts [112]. Furthermore, understanding these intricate relationships will be vital for developing more comprehensive and successful personalized treatment plans. Another important direction for future research involves integrating the use of nutritional genomics into existing frameworks for mental health care. This would entail creating standardized guidelines for genetic testing in clinical settings and training mental health professionals on the fundamentals of nutritional genomics while considering patient privacy ethics [113]. The creation of easily navigable, user-friendly tools and resources for patients and clinicians is another necessary step in integrating these research findings. For instance, this will help provide personalized meal plans and supplement suggestions based on individual

genetic profiles using digital platforms that can integrate genetic data with dietary recommendations. These tools and technologies have the potential to be even more valuable in remote areas where there is limited access to specialized treatment [114].

9. CONCLUSION

Nutritional genomics offers a promising new approach to managing depression through providing more personalized and effective treatment options. Reducing symptoms of depression and improving general mental health can be greatly aided by dietary therapies that are specifically tailored to an individual's genetic composition. As research progresses, integrating these insights into clinical practice could change how we approach mental health care, particularly depression, making personalized nutrition a more accessible and valuable tool. Through these, potentially paving the way for a future where personalized diets become a standard part of mental health treatment, offering new hope for individuals struggling with depression.

REFERENCES

1. Corrêa TA, Quintanilha BJ, Norde MM, Pinhel MA, Nonino CB, Rogero MM. Nutritional genomics, inflammation and obesity. *Archives of Endocrinology and Metabolism*. 2020; 64(3): 205-222. <https://doi.org/10.20945/2359-3997000000255>
2. Guasch-Ferré M, Dashti HS, Merino, J. Nutritional Genomics and Direct-to-Consumer Genetic Testing: An Overview. *Advances in Nutrition*. 2018; 9(2): 128–135. <https://doi.org/10.1093/advances/nmy001>
3. Osada, J. Nutrition Genomics. *International Journal of Molecular Sciences*. 2023; 24(7): 6490. <https://doi.org/10.3390/ijms24076490>
4. de Toro-Martín J, Arsenault BJ, Després JP, Vohl MC. Precision Nutrition: A Review of Personalized Nutritional Approaches for the Prevention and Management of Metabolic Syndrome. *Nutrients*. 2017; 9(8): 913. <https://doi.org/10.3390/nu9080913>
5. Mullins VA, Bresette W, Johnstone L, Hallmark, B, Chilton FH. Genomics in Personalized Nutrition: Can You “Eat for Your Genes”? *Nutrients*. 2020; 12(10): 3118. <https://doi.org/10.3390/nu12103118>
6. Voruganti VS. Precision Nutrition: Recent Advances in Obesity. *Physiology*. 2023; 38(1): 42–50. <https://doi.org/10.1152/physiol.00014.2022>
7. Franzago M, Alessandrelli E, Notarangelo S, Stuppia L, Vitacolonna E. Chrono-Nutrition: Circadian Rhythm and Personalized Nutrition. *International Journal of Molecular Sciences*. 2023; 24(3): 2571. <https://doi.org/10.3390/ijms24032571>
8. Kessler RC, Bromet EJ. (2013). The Epidemiology of Depression Across Cultures. *Annual Review of Public Health*. 2023; 34(1): 119–138. <https://doi.org/10.1146/annurev-publhealth-031912-114409>
9. Manger S. (2019). Lifestyle interventions for mental health. *Australian Journal of General Practice*. 2023; 48(10): 670–673. <https://doi.org/10.31128/AJGP-06-19-4964>
10. Marx W, Moseley G, Berk M, Jacka F. Nutritional psychiatry: The present state of the evidence. *Proceedings of the Nutrition Society*. 2017; 76(4): 427–436. <https://doi.org/10.1017/S0029665117002026>
11. Sousa-Santos N, Fialho M, Madeira T, Clara C, Veiga, S., Martins R *et al*. Nutritional counselling in adults promoting adherence to the Mediterranean diet as adjuvant in the treatment of major depressive disorder (INDEPT): A randomized open controlled trial study protocol. *BMC Psychiatry*. 2023; 23(1): 227. <https://doi.org/10.1186/s12888-023-04705-z>

12. Bruncsics B, Hullam G, Bolgar B, Petschner P, Millinghoffer A, Gecse K *et al.* Genetic risk of depression is different in subgroups of dietary ratio of tryptophan to large neutral amino acids. *Scientific Reports*, 2023; 13(1): 4976. <https://doi.org/10.1038/s41598-023-31495-x>
13. Depressive disorder (depression). (n.d.). Retrieved August 13, 2024, from <https://www.who.int/news-room/fact-sheets/detail/depression>
14. Firth J, Solmi M, Wootton RE, Vancampfort D, Schuch FB, Hoare E *et al.* A meta-review of “lifestyle psychiatry”: The role of exercise, smoking, diet and sleep in the prevention and treatment of mental disorders. *World Psychiatry*. 2020; 19(3): 360–380. <https://doi.org/10.1002/wps.20773>
15. Hepsomali P, Groeger JA. Diet, Sleep, and Mental Health: Insights from the UK Biobank Study. *Nutrients*. 2021; 13(8): 2573. <https://doi.org/10.3390/nu13082573>
16. Magzal F, Turróni S, Fabbri M, Barone M, Vitman Schorr A, Ofran A *et al.* A personalized diet intervention improves depression symptoms and changes microbiota and metabolite profiles among community-dwelling older adults. *Frontiers in Nutrition*. 2023; 10. <https://doi.org/10.3389/fnut.2023.1234549>
17. Ceolin G, Breda V, Koning E, Meyyappan AC, Gomes FA, Moreira JD *et al.* A possible antidepressive effect of dietary interventions: emergent findings and research challenges. *Current Treatment Options in Psychiatry*. 2022; 9(3): 151-162.
18. Connell J, Toma R, Ho CHC, Shen N, Moura P, Le T, *et al.* Data-driven precision nutrition improves clinical outcomes and risk scores for IBS, depression, anxiety, and T2D. *American Journal of Lifestyle Medicine*. 2023: 15598276231216393.
19. Calabrese L, Frase R, Ghaloo M. Complete remission of depression and anxiety using a ketogenic diet: Case series. *Frontiers in Nutrition*. 2024; 11. <https://doi.org/10.3389/fnut.2024.1396685>
20. Cheema MAR, Mahmood K, Basharat S, Haleem DJ, Khan RA. Need of Nutrigenomic Studies for the Prevention and Treatment of Mood and Neurodegenerative Disorders. *Food Nutr OA*. 2018; 1(1): 106.
21. Kring AM, Johnson SL. Abnormal psychology: The science and treatment of psychological disorders. *John Wiley & Sons*. 2022
22. Paans NP, Bot M, van Strien T, Brouwer IA, Visser M, Penninx BW. Eating styles in major depressive disorder: Results from a large-scale study. *Journal of psychiatric research*. 2018; 97:38-46.
23. Gezahegn E, Edris M, Dachew BA. Prevalence and factors associated with undernutrition among adults with major depressive disorder in Northwest Ethiopia. *Psychiatry Journal*. 2016; 2016(1):7034582.
24. Ortega MA, Fraile-Martínez Ó, García-Montero C, Alvarez-Mon MA, Lahera G, Monserrat J, Llaveró-Valero M, Mora F, Rodríguez-Jiménez R, Fernández-Rojo S, Quintero J. Nutrition, epigenetics, and major depressive disorder: understanding the connection. *Frontiers in Nutrition*. 2022; 9:867150.
25. Park SH, Choi HK, Park JH, Hwang JT. Current insights into genome-based personalized nutrition technology: a patent review. *Frontiers in Nutrition*. 2024; 11:1346144.
26. dependent associations between serotonin transporter gene (SLC6A4) DNA methylation and late-life depression. *BMC psychiatry*. 2018; 18:1-0.
27. Birla M, Choudhary C, Singh G, Gupta S, Vavilala P. The Advent of Nutrigenomics: A Narrative Review with an Emphasis on Psychological Disorders. *Preventive Nutrition and Food Science*. 2022; 27(2):150.
28. Gibson EL. Tryptophan supplementation and serotonin function: genetic variations in behavioural effects. *Proceedings of the Nutrition Society*. 2018; 77(2):174-88.
29. Yang T, Nie Z, Shu H, Kuang Y, Chen X, Cheng J, Yu S, Liu H. The role of BDNF on neural plasticity in depression. *Frontiers in cellular neuroscience*. 2020; 14:82.

30. Egan MF, Kojima M, Callicott JH, Goldberg TE, Kolachana BS, Bertolino A, Zaitsev E, Gold B, Goldman D, Dean M, Lu B. The BDNF val66met polymorphism affects activity-dependent secretion of BDNF and human memory and hippocampal function. *Cell*. 2003; 112(2):257-69.
31. Marx W, Lane M, Hockey M, Aslam H, Berk M, Walder K *et al*. Diet and depression: exploring the biological mechanisms of action. *Molecular psychiatry*. 2021; 26(1):134-50.
32. Ljungberg T, Bondza E, Lethin C. Evidence of the importance of dietary habits regarding depressive symptoms and depression. *International journal of environmental research and public health*. 2020; 17(5):1616.
33. Gibson-Smith D, Bot M, Brouwer IA, Visser M, Giltay EJ, Penninx BW. Association of food groups with depression and anxiety disorders. *European journal of nutrition*. 2020; 59:767-78.
34. Mesripour A, Hajhashemi V, Kuchak A. Effect of concomitant administration of three different antidepressants with vitamin B6 on depression and obsessive compulsive disorder in mice models. *Research in Pharmaceutical Sciences*. 2017; 12(1):46-52.
35. Baradia G, Kumar P, Seth AK, Toora BD, Sharma P, Trehan SK. Levels of Serum Vitamin B6 and GABA In Clinically Depressed Patients: A Study. *Annals of International Medical and Dental Research*. 2018; 4(5):1.
36. Kafeshani M, Feizi A, Esmailzadeh A, Keshteli AH, Afshar H, Roohafza H, Adibi P. Higher vitamin B6 intake is associated with lower depression and anxiety risk in women but not in men: A large cross-sectional study. *International Journal for Vitamin and Nutrition Research*. 2019.
37. Ekinci GN, Sanlier N. The relationship between nutrition and depression in the life process: A mini-review. *Experimental gerontology*. 2023; 172:112072.
38. Sangle P, Sandhu O, Aftab Z, Anthony AT, Khan S. Vitamin B12 supplementation: preventing onset and improving prognosis of depression. *Cureus*. 2020; 12(10).
39. Huang, T. L., *et al*. The effects of omega-3 fatty acids in depression: a review of the literature. *Journal of Affective Disorders*. 2005; 65(1): 19-28.
40. Elstgeest LE, Brouwer IA, Penninx BW, Van Schoor NM, Visser M. Vitamin B12, homocysteine and depressive symptoms: a longitudinal study among older adults. *European journal of clinical nutrition*. 2017; 71(4):468-75.
41. Markun S, Gravestock I, Jäger L, Rosemann T, Pichierrri G, Burgstaller JM. Effects of vitamin B12 supplementation on cognitive function, depressive symptoms, and fatigue: a systematic review, meta-analysis, and meta-regression. *Nutrients*. 2021; 13(3):923.
42. Berridge MJ. Vitamin D and depression: cellular and regulatory mechanisms. *Pharmacological reviews*. 2017; 69(2):80-92.
43. Ceolin G, Mano GP, Hames NS, Antunes LD, Brietzke E, Rieger DK, Moreira JD. Vitamin D, depressive symptoms, and Covid-19 pandemic. *Frontiers in neuroscience*. 2021; 15:670879.
44. Alghamdi S, Alsulami N, Khoja S, Alsufiani H, Tayeb HO, Tarazi FI. Vitamin D supplementation ameliorates severity of major depressive disorder. *Journal of Molecular Neuroscience*. 2020; 70:230-5.
45. Lange KW. Omega-3 fatty acids and mental health. *Global Health Journal*. 2020; 4(1):18-30.
46. Nasir M, Bloch MH. Trim the fat: the role of omega-3 fatty acids in psychopharmacology. *Therapeutic advances in psychopharmacology*. 2019; 9:2045125319869791.
47. Thesing CS, Bot M, Milaneschi Y, Giltay EJ, Penninx BW. The association of omega-3 fatty acid levels with personality and cognitive reactivity. *Journal of psychosomatic research*. 2018; 108:93-101.
48. Sánchez-Villegas A, Álvarez-Pérez J, Toledo E, Salas-Salvadó J, Ortega-Azorín C, Zomeño MD *et al*. Seafood consumption, omega-3 fatty acids intake, and life-time prevalence of depression in the PREDIMED-plus trial. *Nutrients*. 2018; 10(12):2000.

49. Milaneschi Y, Peyrot WJ, Nivard MG, Mbarek H, Boomsma DI, WJH Penninx B. A role for vitamin D and omega-3 fatty acids in major depression? An exploration using genomics. *Translational psychiatry*. 2019; 9(1):219.
50. Bender A, Hagan KE, Kingston N. The association of folate and depression: A meta-analysis. *Journal of psychiatric research*. 2017; 95:9-18.
51. Zheng W, Li W, Qi H, Xiao L, Sim K, Ungvari GS *et al*. Adjunctive folate for major mental disorders: A systematic review. *Journal of affective disorders*. 2020; 267:123-30.
52. Abdelmaksoud A, Vojvodic A, Ayhan E, Dönmezdil S, Jovicevic TV, Vojvodic P *et al*. Depression, isotretinoin, and folic acid: a practical review. *Dermatologic therapy*. 2019; 32(6):e13104.
53. Halaris A, Sohl E, Whitham EA. Treatment-resistant depression revisited: a glimmer of hope. *Journal of personalized medicine*. 2021; 11(2):155.
54. Mikkelsen K, Stojanovska L, Prakash M, Apostolopoulos V. The effects of vitamin B on the immune/cytokine network and their involvement in depression. *Maturitas*. 2017; 96:58-71.
55. Eagappan K, Sasikumar S. Nutrient–gene interactions in pathological conditions. *Int J Pharm Bio Sci*. 2013; 3(4):193-7.
56. Mitchell ES, Conus N, Kaput J. B vitamin polymorphisms and behavior: evidence of associations with neurodevelopment, depression, schizophrenia, bipolar disorder and cognitive decline. *Neuroscience & Biobehavioral Reviews*. 2014; 47:307-20.
57. Matteini AM, Walston JD, Bandeen-Roche K, Arking DE, Allen RH, Fried LP *et al*. Transcobalamin-II variants, decreased vitamin B12 availability and increased risk of frailty. *The Journal of nutrition, health and aging*. 2010; 14(1):73-7.
58. Kurnat-Thoma EL, Pangilinan F, Matteini AM, Wong B, Pepper GA, Stabler SP *et al*. Association of transcobalamin II (TCN2) and transcobalamin II-receptor (TCbIR) genetic variations with cobalamin deficiency parameters in elderly women. *Biological Research for Nursing*. 2015; 17(4):444-54.
59. Wu YL, Ding XX, Sun YH, Yang HY, Chen J, Zhao X *et al*. Association between MTHFR C677T polymorphism and depression: an updated meta-analysis of 26 studies. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*. 2013; 46:78-85.
60. Hartig J, Nemeş B. BDNF-related mutations in major depressive disorder: a systematic review. *Acta Neuropsychiatrica*. 2023; 35(1):5-26.
61. Gaysina D, Cohen S, Craddock N, Farmer A, Hoda F, Korszun A *et al*. No association with the 5, 10-methylenetetrahydrofolate reductase gene and major depressive disorder: Results of the depression case control (DeCC) study and a meta-analysis. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics*. 2008 Sep 5;147(6):699-706.
62. Lizer MH, Bogdan RL, Kidd RS. Comparison of the frequency of the methylenetetrahydrofolate reductase (MTHFR) C677T polymorphism in depressed versus nondepressed patients. *Journal of Psychiatric Practice*. 2011; 17(6):404-9.
63. Currenti W, Godos J, Alanazi AM, Lanza G, Ferri R, Caraci F *et al*. Dietary fats and depressive symptoms in Italian adults. *Nutrients*. 2023; 15(3):675.
64. Xie L, Innis SM. Association of fatty acid desaturase gene polymorphisms with blood lipid essential fatty acids and perinatal depression among Canadian women: a pilot study. *Lifestyle Genomics*, 2010; 2(4-5), 243-250.
65. Nedaeinia R, Jafarpour S, Safabakhsh S, Ranjbar M, Poursafa P, Perez P *et al*. Lifestyle Genomic interactions in Health and Disease. *Healthy Lifestyle: From Pediatrics to Geriatrics*. 2022:25-74.
66. Raiston CL, Miller AH. The evolutionary significance of depression in Pathogen Host Defense (PATHOS-D). *Molecular psychiatry*. 2013; 18(1):15-37.
67. Lopizzo N, BocchioChiavetto L, Cattane N, Plazzotta G, Tarazi FI, Pariante CM *et al*. Gene–environment interaction in major depression: focus on experience-dependent biological systems. *Frontiers in psychiatry*. 2015; 6:68.

68. Baba H, Nakano Y, Maeshima H, Satomura E, Kita Y, Suzuki T, et al. Metabolism of amyloid-beta protein may be affected in depression. *J Clin Psychiatry*. 2012; 73:115–20. doi:10.4088/JCP.10m06766 PubMed Abstract
69. Namekawa Y, Baba H, Maeshima H, Nakano Y, Satomura E, Takebayashi N, et al. Heterogeneity of elderly depression: increased risk of Alzheimer's disease and Abeta protein metabolism. *Prog Neuropsychopharmacol Biol Psychiatry*. 2013; 43:203–8. doi:10.1016/j.pnpbp.2012.12.016
70. Postmortem GW. Current status of the human evidence for the genetics of depression. *Behavioral Genetics of the Mouse: Volume 2, Genetic Mouse Models of Neurobehavioral Disorders*. 2014:254.
71. Lewis, S. J., et al. Association between the 5-HTTLPR polymorphism and unipolar depression: A meta-analysis. *Molecular Psychiatry*. 2006; 11(8): 734-738.
72. Papakostas, G. I., et al. Folate and S-adenosylmethionine for major depressive disorder: A review of the literature. *Journal of Clinical Psychiatry*. 2012; 73(5): 593-602.
73. Marzola P, Melzer T, Pavesi E, Gil-Mohapel J, Brocardo PS. Exploring the role of neuroplasticity in development, aging, and neurodegeneration. *Brain Sciences*. 2023; 13(12):1610.
74. Ryu, H., et al. The role of BDNF in the pathophysiology of major depression: A combination of genetic, epigenetic, and environmental factors. *Journal of Psychiatric Research*. 2013; 47(1): 1-12.
75. Mitchell, J. A., et al. The association between brain-derived neurotrophic factor Val66Met and depression: A meta-analysis. *Psychological Medicine*. 2012; 42(4): 729-739.
76. Wan L, Li Y, Zhang Z, Sun Z, He Y, Li R. Methylenetetrahydrofolate reductase and psychiatric diseases. *Translational psychiatry*. 2018; 8(1):1-2.
77. Dashputre NL, Sable RR, Khairnar SJ, Patil SB, Kadam JD. Fundamentals of Nutrigenetics and Nutrigenomics. *In Nutrigenomics and Nutraceuticals*. 2024: 1-24. Apple Academic Press.
78. Coppen A, Bolander-Gouaille C. Treatment of depression: time to consider folic acid and vitamin B12. *Journal of psychopharmacology*. 2005; 19(1):59-65.
79. Gilbody, S., et al. The effect of vitamin B12, folic acid and B6 on cognitive and psychological function in older people: A randomized controlled trial. *Journal of Affective Disorders*. 2007; 103(1-3): 291-299.
80. Tassone G, Carradori S, Maramai S, D'Agostino I. Catechol-O-methyltransferase (COMT). In *Metalloenzymes*. 2024: 63-81. Academic Press.
81. Gadow, K. D., et al. Association of the COMT gene with symptom severity in adults with attention-deficit hyperactivity disorder. *Journal of Psychiatric Research*. 2009; 43(4): 403-410.
82. Qi G, Mi Y, Shi X, Gu H, Brinton RD, Yin F. ApoE4 impairs neuron-astrocyte coupling of fatty acid metabolism. *Cell reports*. 2021; 34(1).
83. Xiyang M, Wenbo W, Wangyi F, Qinghuai L. Association of apolipoprotein E polymorphisms with age-related macular degeneration subtypes: an updated systematic review and meta-analysis. *Archives of medical research*. 2017; 48(4):370-7.
84. Panza F, et al. Apolipoprotein E and depression in healthy elderly subjects: A systematic review. *Journal of Psychiatric Research*. 2010; 44(12): 855-865.
85. Juszczak G, Mikulska J, Kasperek K, Pietrzak D, Mrozek W, Herbet M. Chronic stress and oxidative stress as common factors of the pathogenesis of depression and Alzheimer's disease: The role of antioxidants in prevention and treatment. *Antioxidants*. 2021; 10(9):1439.
86. Murga-Garrido SM, Hong Q, Cross TW, Hutchison ER, Han J, Thomas SP et al. Gut microbiome variation modulates the effects of dietary fiber on host metabolism. *Microbiome*. 2021; 9(1):117.
87. Enkhmaa B, Surampudi P, Anuurad E, Berglund L. Lifestyle changes: effect of diet, exercise, functional food, and obesity treatment on lipids and lipoproteins. *Endotext [Internet]*. 2018.

88. Kiecolt-Glaser JK, Belury MA, Andridge R, Malarkey WB, Glaser R. Omega-3 supplementation lowers inflammation and anxiety in medical students: a randomized controlled trial. *Brain, behavior, and immunity*. 2011; 25(8):1725-34.
89. Serón-Arbeloa C, Labarta-Monzón L, Puzo-Foncillas J, Mallor-Bonet T, Lafita-López A, Bueno-Vidales N, Montoro-Huguet M. Malnutrition screening and assessment. *Nutrients*, 2022;14(12), 2392.
90. Teusen C, Bühner M, Hapfelmeier A, von Schrottenberg V, Linde K, Gensichen J, Schneider A. Development and psychometric evaluation of a questionnaire for the assessment of depression in primary care: a cross-sectional study. *BMJ open*. 2024; 14(7):e084102.
91. Chilton FH, Dutta R, Reynolds LM, Sergeant S, Mathias RA, Seeds MC. Precision nutrition and omega-3 polyunsaturated fatty acids: A case for personalized supplementation approaches for the prevention and management of human diseases. *Nutrients*. 2017; 9(11):1165.
92. Kohlmeier M. Nutrient metabolism: structures, functions, and genes. Academic Press; 2015.
93. Mullins VA, Bresette W, Johnstone L, Hallmark B, Chilton FH. Genomics in personalized nutrition: can you “eat for your genes”? *Nutrients*. 2020; 12(10):3118.
94. Maj M, Stein DJ, Parker G, Zimmerman M, Fava GA, De Hert M *et al*. The clinical characterization of the adult patient with depression aimed at personalization of management. *World Psychiatry*. 2020; 19(3):269-93.
95. Singar S, Nagpal R, Arjmandi BH, Akhavan NS. Personalized Nutrition: Tailoring Dietary Recommendations through Genetic Insights. *Nutrients*. 2024; 16(16):2673.
96. Rainey-Smith SR, Gu Y, Gardener SL, Doecke JD, Villemagne VL, Brown BM *et al*. Mediterranean diet adherence and rate of cerebral A β -amyloid accumulation: Data from the Australian Imaging, Biomarkers and Lifestyle Study of Ageing. *Translational psychiatry*. 2018; 8(1):238..
97. Alarcón Garavito GA, Moniz T, Deom N, Redin F, Pichini A, Vindrola-Padros C. The implementation of large-scale genomic screening or diagnostic programmes: A rapid evidence review. *European Journal of Human Genetics*. 2023; 31(3):282-95.
98. Nielsen DE, El-Sohemy A. A randomized trial of genetic information for personalized nutrition. *Genes & nutrition*. 2012; 7(4):559-66.
99. Ordovás JM, Smith CE. Epigenetics and cardiovascular disease. *Nature Reviews Cardiology*. 2010; 7(9):510-9.
100. Corella D, Coltell O. Precision Nutrition and Cardiovascular Disease Prevention: From Specific Dietary Components to Personalized Nutrition. *Advances in Nutrition*. 2018; 9(4): 313-325.
101. Maj M, Stein DJ, Parker G, Zimmerman M, Fava GA, De Hert M *et al*. The clinical characterization of the adult patient with depression aimed at personalization of management. *World Psychiatry*. 2020; 19(3):269-93.
102. Piao J, Wang Y, Zhang T, Zhao J, Lv Q, Ruan M *et al*. Antidepressant-like effects of representative types of food and their possible mechanisms. *Molecules*. 2023; 28(19):6992.
103. Kiani AK, Bonetti G, Donato K, Kaftalli J, Herbst KL, Stuppia L *et al*. Polymorphisms, diet and nutrigenomics. *Journal of preventive medicine and hygiene*. 2022; 63(2 Suppl 3):E125.
104. Levin BL, Varga E. MTHFR: addressing genetic counseling dilemmas using evidence-based literature. *Journal of genetic counseling*. 2016; 25:901-11.
105. Alhajji L, Nemeroff CB. Personalized medicine and mood disorders. *Psychiatr Clin North Am*. 2015; 38(3):395-403.
106. Loughman A, Staudacher HM, Rocks T, Ruusunen A, Marx W, O'neil A, Jacka FN. Diet and mental health. *Microbes and the Mind*, 2021;32, 100-112.
107. Menke A. Precision pharmacotherapy: psychiatry's future direction in preventing, diagnosing, and treating mental disorders. *Pharmacogenomics and personalized medicine*. 2018: 211-22.

108. Derossi A, Husain A, Caporizzi R, Severini C. Manufacturing personalized food for people uniqueness. An overview from traditional to emerging technologies. *Critical reviews in food science and nutrition*. 2020; 60(7):1141-59.
109. Gautam S, Jain A, Gautam M, Vahia VN, Grover S. Clinical practice guidelines for the management of depression. *Indian journal of psychiatry*. 2017; 59(1): 34-50.
110. Pelletier LG, Dion SC, Slovinec-D'Angelo M, Reid R. Why do you regulate what you eat? Relationships between forms of regulation, eating behaviors, sustained dietary behavior change, and psychological adjustment. *Motivation and emotion*. 2004; 28(3):245-77.
111. Middleton KR, Anton SD, Perri MG. Long-term adherence to health behavior change. *American journal of lifestyle medicine*. 2013; 7(6):395-404.
112. Opie RS, O'Neil A, Itsiopoulos C, Jacka FN. The impact of whole-of-diet interventions on depression and anxiety: a systematic review of randomised controlled trials. *Public health nutrition*. 2015; 18(11):2074-93.
113. Kohlmeier M, De Caterina R, Ferguson LR, Görman U, Allayee H, Prasad C *et al*. Guide and position of the International Society of Nutrigenetics/Nutrigenomics on personalized nutrition: part 2-ethics, challenges and endeavors of precision nutrition. *Lifestyle Genomics*. 2016; 9(1):28-46.
114. Rosenfeld RM, Shiffman RN, Robertson P. Clinical practice guideline development manual: a quality-driven approach for translating evidence into action. *Otolaryngology—Head and Neck Surgery*. 2013;148(1): 1-55.