

Review

Nutrigenetic modulators of COMT and MAO pathways: relevance of green tea and curcumin in psycho-oncology

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Abstract: Cancer is associated with significant psychological burdens, including anxiety, depressive symptoms, and a decline in quality of life. Psycho-oncological outcomes are shaped by the complex interplay of biological, psychological, and social factors, necessitating personalized and multidisciplinary approaches. In recent years, there has been growing interest in whether genetic variations in enzymes such as catechol-O-methyltransferase (COMT) and monoamine oxidase (MAO) may modulate psychological responses in cancer patients. Correspondingly, due to epigenetic effects in cancer patients, nutraceutical compounds such as epigallocatechin-3-gallate (EGCG) and curcumin are noteworthy for their antioxidant and anti-inflammatory effects and are being investigated for their potential interactions with the COMT/MAO pathways. Preclinical and mechanistic studies suggest that these compounds may influence neurotransmitter metabolism, gene expression, and stress responses. However, direct clinical studies assessing their effects on psychological outcomes by COMT/MAO genotype are quite limited.

The aim of this review is to evaluate the relationships among COMT and MAO polymorphisms, nutraceutical interventions, and antidepressants (particularly SSRIs and MAO inhibitors) commonly used in psycho-oncological patients. This review positions COMT and MAO based nutrigenetic approaches alongside psychotropic drug use as a promising but still developing field; it emphasizes the need for more robust and direct clinical evidence to safely and effectively translate these approaches into clinical practice. In this context, personalized strategies that go beyond standard approaches, considering genetic variations, individual biochemical responses, and nutraceutical interactions together, may be more rational and effective; therefore, the need for a personalized approach in this field is increasingly recognized.

Keywords: nutrigenetics, epigenetic nutrition, COMT, MAO, green tea, curcumin, psycho-oncology

1. Introduction

Cancer is often associated not only with a physical symptom burden but also with significant psychological morbidities, including anxiety, depressive symptoms, sleep disturbances, fatigue, and a decline in quality of life. In psycho-oncology, these outcomes must be carefully distinguished: transient emotional responses to diagnosis or treatment are not equivalent to clinically significant psychological distress or psychiatric illness. This distinction is important because psycho-oncological care is based on multidimensional assessments that include not only biological markers but also symptom severity, coping capacity, social support, illness perception, and patient-reported outcomes. Therefore, recent literature emphasizes the need for more personalized and multidisciplinary psycho-oncology models that integrate

psychosocial, clinical, and, when necessary, biological determinants [1-2].

Within this broad framework, there has been growing interest in whether individual differences in neurotransmitter metabolism can partially explain variability in psychological outcomes among cancer patients. In this context, the enzymes catechol-O-methyltransferase (COMT) and monoamine oxidase (MAO) are particularly noteworthy; both enzymes play roles in the metabolism of catecholamines and other neuroactive compounds. Specifically, functional variants such as the Val158Met polymorphism in the COMT gene have been associated with enzyme activity, cortical dopamine levels, stress response, and various psychological phenotypes. Studies in psycho-oncology suggest that these genetic differences may be related to symptoms such as anxiety, fatigue, and depression and could help explain inter-individual differences. Furthermore, mechanisms involving COMT and MAO are thought to influence psychological symptoms and treatment responses through neurotransmitter metabolism [3-5].

In parallel with this, growing interest in complementary and integrative oncology has heightened attention to the potential effects of nutraceuticals and bioactive food components on these pathways. Among these compounds, particularly epigallocatechin-3-gallate (EGCG), the main catechin of green tea, and curcumin, the primary polyphenolic compound of turmeric, stand out. These compounds are of interest for their antioxidant, anti-inflammatory, and epigenetic regulatory properties, and preclinical studies suggest they can modulate multiple pathways implicated in carcinogenesis, neuroinflammation, and cellular stress responses [6-8]. Additionally, experimental data suggest that EGCG and curcumin may directly or indirectly influence mechanisms associated with COMT and MAO [9-10].

Additionally, although EGCG and curcumin are generally regarded as 'harmless supplement products', their potential interactions with pharmacological treatments in oncology practice should be considered. The effects of these compounds on cytochrome P450 enzymes and phase II metabolic pathways may lead to pharmacokinetic and pharmacodynamic changes when used alongside chemotherapeutics and psychiatric medications [11-13]. In the context of psycho-oncology, it has been reported that the efficacy and side effect profiles of antidepressants such as SSRIs and other supportive therapies vary among individuals; this variability is associated with genetic factors involved in drug metabolism. There is also evidence suggesting that genetic variations in enzymes such as MAO and COMT, which are involved in monoamine metabolism, can influence antidepressant response. However, the integration of pharmacogenetic data into clinical practice remains limited, and further clinical evidence is needed for the routine use of personalised treatment approaches [14-16]. Particularly within the oncology population, more research is required to understand the effects of curcumin and EGCG on MAO and COMT enzymes at dietary physiological doses, their epigenetic regulatory roles, and their reflection in clinical outcomes. Therefore, the aim of this review is not to present the effects of EGCG and curcumin on psycho-oncological outcomes as definitive clinical benefits; rather, it is to evaluate the relationship between these compounds and the COMT and MAO pathways, epigenetic mechanisms, and psychological symptoms based on current evidence. Additionally, this review aims to discuss the clinical significance of these mechanisms within the context of multidisciplinary psycho-oncological care, the importance of patient-reported outcomes, and potential herbal-drug interactions. This approach seeks to clarify the limitations of existing evidence and guide future clinical studies.

2. Psycho-Oncological Concepts and Clinical Framework

Personalised psycho-oncology is a holistic approach that addresses the psychological and general health needs of cancer patients in accordance with individual differences. This model aims to move away from the 'one-size-fits-all' treatment concept by considering genetic, clinical, and environmental characteristics, and to provide appropriate interventions to the right patient at the right time [1,17]. Supported by a patient-centered approach, this framework seeks to enhance adherence and quality of life by integrating the individual's values and preferences into the treatment process. In oncology, taking into account the tumor's genetic makeup and microenvironment allows for the development of more effective and less side-effect-prone treatment strategies [18]. However, there are still some challenges in integrating this holistic approach into clinical practice, particularly regarding communication and interdisciplinary coordination [19-21].

From a psycho-oncology perspective, it is known that psychiatric and psychological issues are quite common in cancer patients and directly influence the course of the disease. Approximately half of the patients experience such problems, with adjustment disorders and depression being the most prominent [22-24]. These comorbid conditions are associated with increased mortality, and delays in diagnosis or limited access to treatment can further heighten this risk [25]. Additionally, increased cortisol levels caused by chronic stress and changes in the immune system may negatively affect the prognosis of the disease. The reduction in treatment adherence due to mental health status is also a significant factor that markedly worsens clinical outcomes [26-27]. Therefore, early diagnosis, personalised interventions, and an interdisciplinary approach play a critical role in improving both psychological well-being and oncological outcomes.

The diagnosis and treatment process of cancer is not only physically demanding but also imposes a significant psychological burden. Among the most commonly assessed conditions in psycho-oncology are distress, anxiety, depression, sleep disturbances, fatigue, and a decline in quality of life [2,28]. However, it is important to accurately differentiate these symptoms clinically; transient emotional reactions that occur at the time of diagnosis must be distinguished from clinically significant psychiatric conditions.

Psychological outcomes are not limited solely to biological mechanisms; they are shaped by the interaction of numerous factors such as the stage of the disease, treatments applied and their side effects, the level of social support, coping strategies, and the individual's psychiatric history. This indicates that psycho-oncological risk has a multidimensional and dynamic nature [1]. Therefore, an effective psycho-oncological approach requires an integrated and personalised model that addresses biological, psychological, and social components together.

3. COMT and MAO Biology and Genetic Variations

Catechol O-methyltransferase (COMT) is an important enzyme involved in the second phase of metabolism, responsible for inactivating endogenous or exogenous catechols such as catecholamines and catechol estrogens, as well as the metabolism of certain drugs. Catechol O-methyltransferase transfers a methyl group from S-adenosylmethionine (SAM) to a substrate molecule containing catechol. A genetic variant in the COMT gene, Val158Met, results in a several-fold reduction in enzymatic activity, potentially leading to the accumulation of carcinogenic endogenous catechol estrogens and reactive intermediates, thereby increasing the

risk of carcinogenesis. EGCG can act as a potent inhibitor of the COMT enzyme and may potentially delay the detoxification of endogenous catechol estrogens that could be carcinogenic [10]. The best-known functional polymorphism in the COMT gene is the Val158Met (rs4680) variant. This variant causes significant differences in enzyme activity; the Val/Val genotype is associated with high enzyme activity, while the Met/Met genotype is linked to low activity and consequently increased synaptic dopamine levels [3]. It is suggested that these biochemical differences may influence psychological processes such as stress response, emotional regulation, and anxiety [5].

Monoamine oxidase (MAO) enzymes are mitochondrial structures located on the X chromosome that facilitate the breakdown of neurotransmitters such as dopamine, norepinephrine, and serotonin. Polymorphisms in these genes can alter enzyme activity, thereby affecting neurotransmitter levels and related behavioural processes. In particular, low MAO activity has been associated with slower degradation of monoamines and increased dopamine levels, which has been linked to positive symptoms of schizophrenia. In this context, the MAO rs6323 T allele has been found to be associated with reduced enzyme activity and elevated dopamine levels [29-32].

4. The Biochemical and Epigenetic Effects of EGCG and Curcumin

Epigallocatechin-3-gallate (EGCG) and curcumin are plant-derived polyphenolic compounds that are notable for their antioxidant, anti-inflammatory, and epigenetic regulatory properties. These compounds can modulate epigenetic mechanisms by affecting DNA methylation, histone modifications, and gene expression [6-7].

Epigallocatechin gallate (EGCG), a polyphenolic catechin predominantly found in green tea; has been shown to increase apoptosis and suppress cell proliferation through epigenetic mechanisms. In this context, it is reported that regular consumption may be particularly associated with a reduction in the risk of breast and prostate cancers. Curcumin, on the other hand, is a phenolic compound obtained from the rhizomes of the *Curcuma longa* (turmeric) plant, and has traditionally been used for medicinal purposes in China and India. The literature indicates that curcumin possesses anti-cancer properties; especially through its effects on epigenetic regulation, it is considered a potential non-toxic hypomethylating agent in the context of breast cancer [6, 33-35].

DNA methylation is a fundamental mechanism that regulates gene expression through DNMT enzymes, and an increase in DNMT enzymes in breast cancer is associated with the silencing of tumour suppressor genes. Epigallocatechin gallate (EGCG) exerts a regulatory effect on DNA methylation by inhibiting the activity of DNMT enzymes. This inhibition can particularly support gene re-expression by reducing hypermethylation in the promoter regions of tumour suppressor genes. Thus, EGCG stands out as an agent capable of modulating abnormal methylation patterns at the epigenetic level, thereby potentially suppressing cancer development. Curcumin, on the other hand, modulates disrupted DNA methylation patterns by suppressing DNMT activity; it supports the re-expression of tumour suppressor genes while balancing oncogenic processes. Additionally, by indirectly contributing to DNA methylation processes alongside components that affect one-carbon metabolism, it exhibits chemopreventive effects at the epigenetic level [9, 36].

5. Nutrigenetic Interactions and Psycho-oncological Outcomes

The nutrigenetic approach examines how individuals' genetic makeup influences their response to nutrient components. The COMT enzyme plays a role in the

metabolism of catechol compounds and therefore has the potential to interact directly with polyphenols such as EGCG. Methylation of EGCG by COMT may reduce its biological activity; conversely, inhibition of COMT activity could enhance the effects of this compound [10].

It is theoretically suggested that in individuals with low COMT activity (Met/Met genotype), increased catecholamine levels due to reduced enzyme activity may modulate the stress response, and this could influence individual treatment responses; however, these relationships are largely based on mechanistic and experimental data [37].

Curcumin and green tea catechins (especially EGCG) are natural compounds that can inhibit enzymes such as Monoamine Oxidase (MAO) and Catechol-O-methyltransferase (COMT), which play a role in monoamine metabolism. It has been reported that curcumin particularly exhibits inhibition on MAO-A and MAO-B. Curcumin's MAO inhibitory effects are largely limited to in vitro studies and animal models [38-40]. This effect may increase synaptic levels by reducing the breakdown of serotonin and other monoamines. Since curcumin shows an inhibitory effect on the Monoamine Oxidase (MAO) enzyme responsible for the metabolism of monoamine neurotransmitters, it is considered a potential natural source for MAO inhibitors used in Parkinson's disease and other neurological disorders [39]. Additionally, studies conducted on animal models indicate that curcumin reduces depressive-like behaviours through MAO inhibition, and this effect may be related to central monoaminergic systems [40] (**Table 1**).

Green tea catechins, particularly EGCG, are metabolised by O-methylation via Catechol-O-methyltransferase (COMT) and can also inhibit this enzyme. COMT is responsible for the inactivation of catecholamines such as dopamine and Levodopa (L-DOPA) through methylation. Therefore, inhibition of COMT can increase the bioavailability and dopaminergic effects of L-DOPA by reducing its methylation; it is also thought that green tea catechins may exert effects through a similar mechanism [41]. EGCG and green tea extract may have neuroprotective effects by reducing dopaminergic neuron loss in Parkinson's disease models. Considering the roles of Monoamine Oxidase (MAO) and COMT in catecholamine metabolism, COMT inhibition is used as a therapeutic approach in Levodopa treatment [42] (**Table 1**).

The clinical significance of concurrent use with antidepressants primarily arises with Selective Serotonin Reuptake Inhibitors (SSRIs) and Monoamine Oxidase Inhibitors (MAOIs). SSRIs increase synaptic serotonin levels by inhibiting presynaptic serotonin reuptake, while MAOIs inhibit the monoamine oxidase enzyme, reducing the breakdown of monoamines and thus increasing neurotransmitter levels [43-44]. If curcumin's MAO-inhibitory effect is added to this, the serotonergic load theoretically increases, raising the risk of Serotonin Syndrome. Therefore, agents such as phenelzine and tranylcypromine should not be used together with MAO inhibitors. One study indicated that a combination of multiple serotonergic agents + curcumin (turmeric supplement) + opioid (fentanyl) could excessively increase serotonin levels, leading to the development of Serotonin Syndrome. Curcumin's inhibitory effect on Monoamine Oxidase (MAO), when used alongside serotonergic drugs like fluoxetine, may reduce serotonin breakdown and increase its accumulation. Additionally, the administration of fentanyl can trigger serotonergic effects, potentially causing symptoms such as acute loss of consciousness and myoclonus, thereby precipitating Serotonin Syndrome. In brief, the combined use of curcumin

+ serotonergic antidepressant + opioid may have a cumulative effect that triggers serotonin toxicity, so caution is advised [45]. Another study observed that adding a low dose of curcumin (40 mg/day) to sertraline treatment did not provide additional improvement over placebo in major depression symptoms, but the group receiving curcumin had lower anxiety levels and no serious side effects. This suggests that the combination of curcumin + sertraline does not significantly increase depression but may have a mild positive effect on anxiety and appears safe [46]. (**Figure 1**)

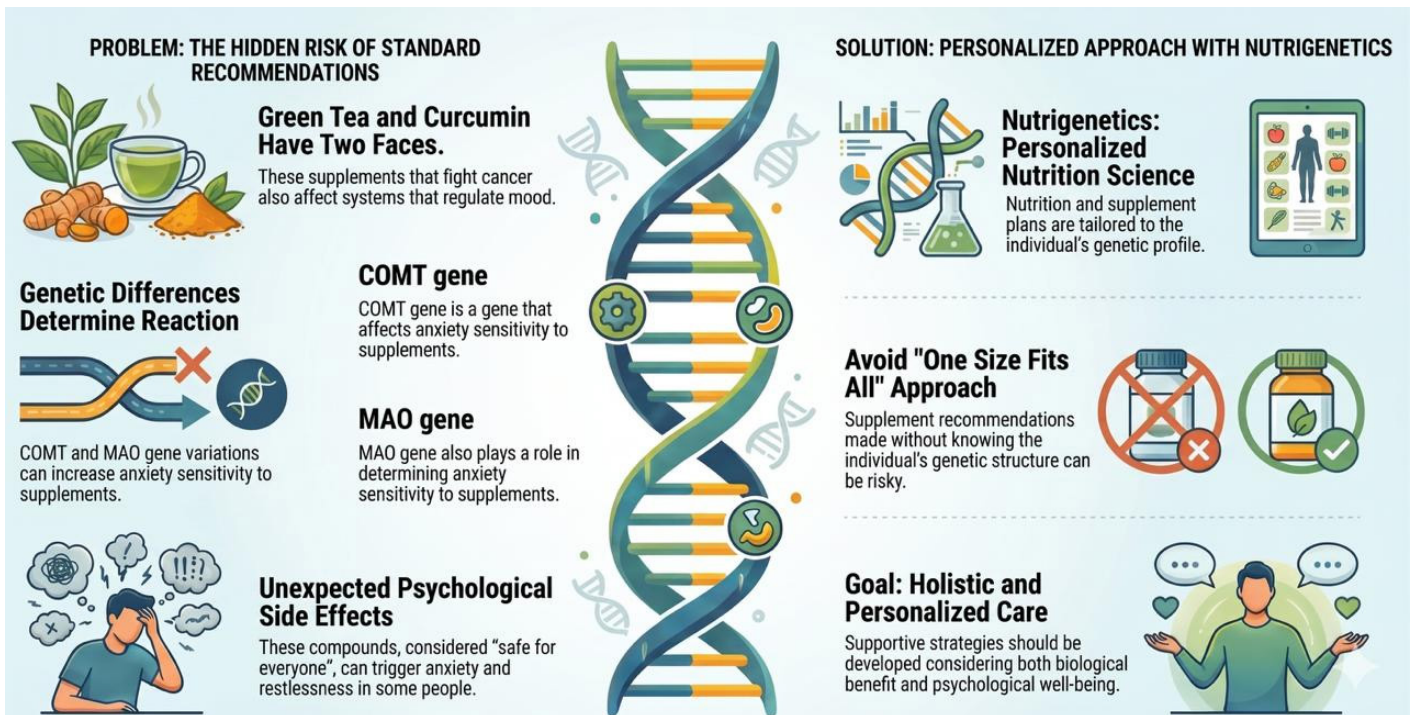


Figure 1: COMT/MAO Pathway Modulation by Green Tea Polyphenols and Curcumin: Implications for Precision Psycho-Oncology

While curcumin and green tea catechins may exert neuroprotective and potential therapeutic benefits by affecting monoamine metabolism through MAO and COMT enzymes, they may also increase the risk of serotonin syndrome when used in combination with serotonergic antidepressants and MAO inhibitors by cumulatively elevating serotonin levels. Therefore, in clinical use, individual differences based on dose, combination, and genetic variations should be considered.

Table 1: The effects of curcumin and EGCG on the monoaminergic system in the context of nutrigenetics

Dietary compound	Mechanism of interaction (with SSRI/MAOI)	Clinical significance	Recommended approach	References
Curcumin	Inhibition of MAO-A/B reduces monoamine degradation; may increase serotonergic load when combined with SSRIs	Risk of serotonin syndrome	Use cautiously with serotonergic medications	[38,40].
Curcumin	May increase monoamine accumulation when combined with MAO inhibitors	High risk	Not recommended in patients using MAOIs	[43].

Continuation Table:

Dietary compound	Mechanism of interaction (with SSRI/MAOI)	Clinical significance	Recommended approach	References
EGCG (Green tea catechins)	Metabolized by COMT via O-methylation and may inhibit COMT; can affect catecholamine metabolism	Variable individual effects due to altered neurotransmitter levels	Use caution with high-dose extracts	[41].
EGCG (Green tea catechins)	May reduce L-DOPA metabolism via COMT inhibition; combined MAO/COMT effects may increase catecholamine levels	Moderate risk	Avoid high doses in patients using MAOIs	[41,42].

6. Limitations and Future Perspectives

The current literature is insufficient to clearly define the effects of nutraceutical interventions on psycho-oncological outcomes in relation to COMT/MAO genetic variations. Future research should prioritize well-designed clinical studies in cancer populations, incorporating genotype-based stratification, validated psychological outcome measures, and prospective, controlled study designs.

Furthermore, the integration of nutrigenetic approaches into psycho-oncology should be evaluated not only in terms of biological mechanisms but also within established multidisciplinary care models. In this regard, genetic data should be considered a supportive component of clinical decision-making, rather than a sole determinant.

7. Conclusion

This review has holistically evaluated the potential effects of nutraceutical compounds such as curcumin and EGCG on psycho-oncological processes, in relation to genetic variations in Catechol-O-methyltransferase (COMT) and Monoamine Oxidase (MAO). The findings indicate that these compounds may exert potential biological effects through monoamine metabolism, epigenetic regulation, and antioxidant mechanisms. However, the evidence demonstrating that these effects are directly and consistently reflected in clinical outcomes remains limited.

From a nutrigenetic perspective, particularly the variability in COMT activity among individuals is an important factor that can influence the metabolism and bioavailability of catechol compounds such as EGCG. In individuals with low COMT activity, catecholamine levels and stress responses may differ, which can also lead to variability in responses to nutraceutical interventions. However, these interactions are largely theoretical and mechanistic in nature, and how they translate to clinical outcomes in psycho-oncology remains unclear.

However, considering the multifactorial nature of psycho-oncological outcomes; genetic variations and nutraceutical compounds are not solely determinative. The stage of the disease, treatment-related side effects, psychosocial factors, and concurrent pharmacological treatments significantly influence this process. In particular, the potential pharmacodynamic interactions of curcumin and green tea catechins with antidepressants and other medications (such as increased serotonergic activity) should be carefully evaluated from a clinical perspective.

In conclusion, COMT/MAO-based nutrigenetic approaches offer a promising

framework for personalised supportive treatment in the field of psycho-oncology; however, current evidence is insufficient for the routine clinical integration of these approaches. Therefore, future support through genotype-based, prospective, and controlled clinical studies is necessary

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