Impact Factor (SJIF): 6.092 **E-ISSN: 2583-1615**

International Journal of Research in Academic World

Received: 18/December/2024 **IJRAW:** 2025; 4(1):125-131 **Accepted:** 25/January/2025

Role of Micronutrients in Cancer Therapy

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Abstract

Cancer is a complex disease characterized by uncontrolled cell growth and spread to other parts of the body. It results from genetic mutations and environmental factors that disrupt normal cellular functions, such as DNA repair, apoptosis (programmed cell death), and cell cycle regulation. While the primary treatment for cancer includes surgery, chemotherapy, and radiation, growing evidence suggests that certain micronutrients may play a role in cancer prevention and support during treatment.

This article having literature on the reported anticancerous properties of various micronutrients involves incorporating vitamins, minerals, and plant-based compounds to potentially enhance treatment outcomes, reduce side effects, and support overall health during cancer therapy. While micronutrients cannot cure cancer, research suggests they may play a complementary role in cancer management by influencing cellular processes, reducing oxidative stress, and supporting the immune system.

Keywords: Micronutrients, Cancer, Vitamins, Health.

Introduction

Micronutrients are vital nutrients that the body needs in little amounts for optimal growth, development, and overall health. Unlike macronutrients (carbohydrates, proteins, and fats), which are required in vast quantities, micronutrients are essential for a variety of physiological processes while being consumed in much lesser amounts. They contain vitamins, minerals, and trace elements, all of which serve an important role in sustaining health, supporting metabolic processes, and allowing the body to function effectively.

A lack or imbalance in micronutrients can cause a number of health problems, including decreased immune, stunted growth, chronic disorders, and increased susceptibility to infections. Excessive consumption of certain micronutrients, on the other hand, can be hazardous, causing toxicity or interfering with other vital activities.

They are typically obtained through a balanced diet rich in fruits, vegetables, whole grains, nuts, and seeds, and sometimes supplementation is necessary to address deficiencies or specific health conditions.

1. Minerals showing anticancer activity

1.1. Selenium

Selenium is a trace element that plays a crucial role in various biological functions, including acting as an antioxidant and cofactor for enzymes like glutathione peroxidase. It has attracted significant attention for its potential as an anticancer agent due to its ability to modulate cellular signaling, reduce oxidative stress, enhance immune function, and induce apoptosis in cancer cells. Numerous studies have investigated selenium's potential role in cancer prevention and therapy, with promising findings in cancers such as breast, prostate, lung, and colorectal cancer.

Selenium is a component of selenoproteins like glutathione peroxidase, which help neutralize reactive oxygen species (ROS) and reduce oxidative stress. Since oxidative damage to DNA is a key factor in the development of cancer, selenium's ability to act as an antioxidant may contribute to cancer prevention by protecting cells from oxidative damage and DNA mutations. (Rayman *et al*., 2005) [52].

Selenium has been shown to induce apoptosis (programmed cell death) in cancer cells. This occurs through several mechanisms, including the modulation of key proteins in the apoptosis pathway such as p53, Bcl-2, Caspases, and p21. Selenium can enhance the apoptotic response to oxidative stress and inhibit the survival of cancer cells. (Zhang, Y., *et al*. (2011) [70] Selenium influences the cell cycle by regulating key molecules involved in cell division and growth. For instance, selenium can increase the expression of p21 (a cyclin-dependent kinase inhibitor) and p53, leading to cell cycle arrest in cancer cells. This inhibition of cell cycle progression can prevent the proliferation of malignant cells. (Lu, J., *et al*. 2013) [40].

Chronic inflammation is a known risk factor for cancer development and progression. Selenium has anti-inflammatory properties and can regulate inflammatory

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mediators like NF-κB, TNF-α, and IL-6. By reducing inflammation, selenium may prevent the initiation and progression of cancer. (Kasaikina, M. V., *et al*. 2013) [33].

Angiogenesis, the process by which tumors stimulate the growth of new blood vessels to supply nutrients, is critical for tumor growth and metastasis. Selenium has been shown to inhibit angiogenesis by regulating factors such as vascular endothelial growth factor (VEGF), which is involved in the formation of new blood vessels in tumors.

Selenium enhances the activity of immune cells, including T-cells, NK cells (natural killer cells), and macrophages, which play important roles in recognizing and killing cancer cells. By boosting the immune system's ability to target and destroy cancer cells, selenium could contribute to cancer prevention and treatment. (Vahdati, S., *et al*. 2018) [63]. Selenium may protect against breast cancer by promoting DNA repair, reducing oxidative stress, and modulating key signaling pathways involved in cancer cell proliferation and survival (Hughes, D. J., *et al*. 2011) [31]. Selenium has been shown to reduce the risk of prostate cancer in various epidemiological and clinical studies. Its potential effects include antioxidant protection, induction of apoptosis, and modulation of tumor suppressor genes like p53 (Clark, L. C., *et al*. 1996) [15]. Selenium has demonstrated protective effects against liver cancer by reducing oxidative damage, enhancing immune responses, and inducing apoptosis in cancerous liver cells. (Duntas, L. H. 2009) [19].

The deficiency of selenium in diet is associated with increased cancer incidence. Seleno amino acids are reported as chemopreventive in humans (Lee *et al*., 2006) [36]. It was reported that selenium induces apoptosis in human lymphoblastic leukemia MT-4 cells and inhibited prostate cancer in humans by monomethylated selenium (Philchenkov *et al*., 2007) [50]. Selenium is regarded as an important mineral that is present in food at nutritional doses (Zeng *et al*., 2009) [69].

1.2. Zinc

Zinc is regarded as an important co-factor in approximately more than 70 enzymes. Zinc dietary intake is recommended as 11 mg and 9 mg for adult males and females, respectively (Freake *et al*.,2013) [22]. It is required to consume zinc daily in the diet because the excess amount is not stored in the human body. It was reported that zinc inhibits the development of cancer and if it decreases as required amount in serum responsible for various forms of cancer (Magda *et al*., 2008) [42]. Zinc in diet helpful for cancer prevention via the metabolism of vitamin A DNA synthesis, high-density lipoprotein (HDL), etc. Nowadays water-soluble ionophores of zinc having more interest because it is responsible for the free zinc concentration and inhibit proliferation of cancer cells (Sliwinski *et al*., 2009) [58].

Zinc, an essential trace element, plays a vital role in numerous physiological processes, including DNA synthesis, protein synthesis, cell division, and immune function. Zinc plays a crucial role in regulating the cell cycle by influencing the activity of various cell cycle regulators, such as cyclins and cyclin-dependent kinases (CDKs). Research suggests that zinc may influence cancer cell proliferation, apoptosis (programmed cell death), invasion, and metastasis. Moreover, zinc deficiency has been linked to an increased risk of cancer, while zinc supplementation has shown potential in cancer prevention and treatment. (Ma, X., *et al*. 2014) [41]. Zinc has been shown to induce apoptosis in cancer cells by activating caspases, p53, and other molecules involved in programmed cell death. This is important because cancer cells often evade apoptosis, which allows them to proliferate uncontrollably. By promoting apoptosis, zinc may help eliminate cancer cells and inhibit tumor growth. (Yadav, U. C., *et al*. 2014) [68]. Zinc has antioxidant properties that help mitigate oxidative stress and prevent DNA damage. It functions as a cofactor for superoxide dismutase (SOD), an enzyme that neutralizes reactive oxygen species (ROS). Zinc's ability to protect cellular components from oxidative damage may reduce the risk of cancer initiation, particularly since oxidative stress is a key factor in carcinogenesis. (Choi, H. D., *et al*. 2016) [13].

Zinc can modulate several inflammatory pathways, such as the NF-κB pathway, which is associated with the expression of pro-inflammatory cytokines. By regulating these pathways, zinc may reduce inflammation and prevent the promotion of cancer. (Haase, H., *et al*. 2012) [27].

Zinc plays a role in regulating metalloproteinases (such as MMP-2 and MMP-9), enzymes that are involved in the breakdown of extracellular matrix components and the metastasis of cancer cells. Zinc's ability to modulate the activity of these enzymes may help inhibit cancer cell invasion and metastasis. (Bandyopadhyay, S., *et al*. 2004) [7].

Zinc is crucial for the immune system, including the proper function of T-cells, macrophages, and natural killer (NK) cells, which play a critical role in identifying and eliminating cancer cells. Zinc supplementation has been shown to enhance immune responses, which may contribute to cancer surveillance and prevention. (Liuzzi, J. P., *et al*. 2007) [38].

Zinc has been shown to play an important role in prostate cancer prevention. Zinc can regulate androgen receptor signaling and has been found to inhibit the growth of prostate cancer cells. It may also reduce the expression of matrix metalloproteinases (MMPs), which are involved in prostate cancer cell invasion and metastasis. (Ghosh, R., *et al*. 2014) [24].

Zinc is involved in the regulation of estrogen receptor signaling and may help inhibit the proliferation of breast cancer cells. It has been shown to reduce the activation of oncogenic pathways and induce apoptosis in breast cancer cells. (Ahmed, S. R., *et al*. 2012) [3]. Zinc deficiency has been associated with colorectal cancer, and zinc supplementation has shown potential in reducing the risk of cancer by inducing apoptosis and inhibiting the growth of colon cancer cells. Zinc also regulates various signaling pathways, including Wnt/β-catenin and PI3K/Akt, which are critical in colon cancer development. (Huber, A., *et al*. 2015) [30].

It was found that zinc supplementation can inhibit lung cancer cell proliferation and metastasis. Zinc may also modulate the expression of p53 and other tumor suppressor genes involved in the regulation of cell cycle and apoptosis in lung cancer. (Xu, W., *et al*. 2011) [67]. Zinc plays a role in the prevention of oral cancer by modulating the expression of genes involved in inflammation and cell growth. Zinc supplementation has been shown to reduce the proliferation of oral cancer cells and induce apoptosis in oral carcinoma cells. (Agarwal, A., *et al*. 2014) [2].

1.3. Vanadium

It is an essential micronutrient that play important role in the maintenance of DNA, maintains the stability of the genome, etc., while genomic instability is a prompting factor for cancer (Elberg *et al*., 1998) [20]. It was reported that vanadium acts as an anticancer agent acting by inhibition of carcinogen-derived active metabolites prevent chemical carcinogenesis, Inhibition and activation of cellular tyrosine phosphatases, activate

tumor suppressor genes, apoptosis, etc. It modulates cellular adhesive molecules and exerts inhibitory effects on the metastatic potential of tumor cells (Ray *et al*., 2005) [51].

Vanadium is a trace element that has shown significant potential as an anticancer agent in recent years. Its biological activity and anticancer properties are primarily attributed to its ability to modulate cellular signaling pathways, induce apoptosis (programmed cell death), inhibit cell proliferation, and regulate redox signaling. Vanadium compounds, particularly vanadate, have been shown to inhibit protein tyrosine phosphatases (PTPs), which are involved in the regulation of cell signaling pathways related to growth, differentiation, and apoptosis. By inhibiting PTPs, vanadium can enhance tyrosine kinase signaling, which promotes the activation of growth-related pathways and inhibits cancer cell proliferation. (Sundaram, M. K., *et al*. 2012) [59]. Vanadium compounds have been shown to activate the mitogen-activated protein kinase (MAPK) pathway, which is involved in cell proliferation, differentiation, and survival. The activation of this pathway may induce apoptosis in cancer cells, making vanadium a potential candidate for inducing cell death in tumors (Santos, M. D. S., *et al*. 2010) [56]. Vanadium compounds have been shown to induce apoptosis in cancer cells through both mitochondrial-dependent and extrinsic pathways. This includes increasing reactive oxygen species (ROS) generation, which disrupts mitochondrial function and promotes apoptosis. Vanadium may also regulate apoptosis-related proteins like Bcl-2 and caspases, leading to the death of cancer cells. (Ducic, T., *et al*. 2016) [18].

Vanadium has been shown to induce cell cycle arrest at various stages of the cell cycle, particularly in the G0/G1 phase. This cell cycle modulation may prevent cancer cells from proliferating and allow for apoptotic clearance of abnormal cells. Vanadium's effects on cell cycle regulators like cyclins, cyclin-dependent kinases (CDKs), and p21 contribute to this cell cycle arrest. (Ortega, P., *et al*. 2014) [48].

Vanadium compounds also act as antioxidants by scavenging reactive oxygen species (ROS) and reducing oxidative stress. This antioxidant activity can help protect normal cells from oxidative damage while enhancing the cytotoxic effects in cancer cells. The balance between oxidative stress and cellular redox states is crucial in cancer therapy (García-Álvarez, J. M., *et al*. 2015) [23].

Vanadium has shown potential to inhibit metastasis by reducing the migration and invasion of cancer cells. This effect is thought to be due to the inhibition of matrix metalloproteinases (MMPs), enzymes involved in the degradation of the extracellular matrix, a key step in cancer cell invasion and metastasis. (Martínez, F. A., *et al*. 2011) [45].

Vanadium has demonstrated the ability to inhibit the growth and metastasis of breast cancer cells by modulating apoptotic pathways, inhibiting cell migration, and reducing the expression of VEGF and MMPs. These effects make vanadium an attractive candidate for breast cancer therapy. (Tawfik, M. M., *et al*. 2010) [61].Vanadium compounds have shown the potential to reduce colon cancer cell proliferation and induce apoptosis. They may also help in modulating pathways such as Wnt/β-catenin, which are critical for colon cancer progression. (Guthrie, N., *et al*. 2015) [26].

1.4. Magnesium

Magnesium (Mg) is an essential mineral that plays a crucial role in various biological functions, including DNA synthesis, protein synthesis, energy production, and cellular signaling. Its role in cancer biology has garnered attention due to its potential to influence key processes such as cell division, apoptosis (programmed cell death), angiogenesis, and immune response. While magnesium itself does not directly target cancer cells in the same way traditional anticancer drugs do, its influence on several cellular pathways may contribute to cancer prevention and therapeutic strategies. (Rosanoff *et al*., 2012) [53].

It was reported that deficiency of magnesium along with some antioxidants, may lead to various types of cancer (Aleksandrowicz *et al*., 1970) [4]. Deficiency of magnesium leads to abnormalities in metabolism and development of cancer, cancer patients suffering from hypomagnesemia in critical condition (Deheinzelin *et al*., 2000) [17]. It was found that a higher quantity of magnesium in the diet reduces the occurrence of colorectal cancer in men of some countries, like Japan (Anghileri *et al*., 1981) [6]. The deficiency of magnesium leads to membrane permeability increment and triggers the carcinogenesis process (Blondell *et al*., 1980) [10].

Magnesium plays an important role in regulating the cell cycle by modulating the activity of enzymes and proteins such as cyclins, cyclin-dependent kinases (CDKs), and p53. Magnesium deficiency has been associated with altered cell cycle regulation, leading to uncontrolled cell division and cancer progression. (Rude, R. K. 2012) [53]. Magnesium has been shown to influence apoptosis through modulation of proteins such as Bcl-2, p53, and caspases. Magnesium deficiency can lead to increased survival of cancer cells by inhibiting apoptotic pathways. Conversely, magnesium supplementation has been shown to induce apoptosis in various cancer cell lines by activating pro-apoptotic signals. (Bodnar, M., *et al*. (2015) [11].

Magnesium plays a role in regulating angiogenesis, the process by which new blood vessels are formed to supply growing tumors. Magnesium affects the expression of vascular endothelial growth factor (VEGF), a key molecule in angiogenesis. Inhibition of angiogenesis limits tumor growth and metastasis by depriving tumors of nutrients and oxygen. (Mansoori, B., *et al*. 2014) [44]. Magnesium has antioxidant properties and plays a critical role in the function of antioxidant enzymes, including superoxide dismutase (SOD). Magnesium helps mitigate oxidative stress, which is a key driver of DNA damage and cancer initiation. Magnesium deficiency can exacerbate oxidative stress, increasing the risk of cancer development. (Kirkland, A. M., *et al*. 2012) [34].

Magnesium has been found to regulate the NF-κB (nuclear factor kappa-light-chain-enhancer of activated B cells) pathway, which is involved in the production of pro-inflammatory cytokines. Magnesium supplementation can modulate these inflammatory pathways and reduce cancer risk by preventing chronic inflammation. (Zhao, Q., *et al*. 2013) [72].

Magnesium influences immune function, including the activity of T-cells, macrophages, and natural killer (NK) cells. These immune cells play a crucial role in recognizing and eliminating cancer cells. Magnesium deficiency has been linked to reduced immune function, potentially increasing cancer susceptibility. Adequate magnesium levels may enhance immune surveillance and help the body fight cancer. (Maier, J. A. M. 2013) [43]. Magnesium is essential for the stability of DNA, as it is involved in DNA replication and repair. Magnesium's role in DNA repair mechanisms ensures that cells maintain genomic integrity, reducing the risk of mutations that could lead to cancer. A lack of magnesium can impair DNA repair processes and contribute to tumorigenesis. (Xu, J., *et al*. 2010) [66].

Magnesium has been found to affect breast cancer cell proliferation and metastasis. Adequate magnesium levels may help prevent the growth of breast cancer cells by inducing apoptosis and inhibiting cell division. Magnesium deficiency has been linked to an increased risk of breast cancer (Choi, J. Y., *et al*. 2011) [14].

Low magnesium intake has been associated with an increased risk of colorectal cancer. Magnesium may reduce the risk by influencing inflammatory processes, oxidative stress, and DNA repair. In colorectal cancer models, magnesium supplementation has shown potential in reducing tumor growth and improving treatment outcomes (Huang, C. L., *et al*. 2016) [29]. Magnesium may reduce the risk of lung cancer by decreasing oxidative stress and modulating the immune response. Some studies have shown that low magnesium levels are associated with an increased risk of lung cancer, particularly in smokers. Magnesium supplementation could potentially help in lung cancer prevention by enhancing antioxidant defenses and regulating inflammatory cytokines (Chiu, T. H., *et al*. 2015) [12]. Magnesium deficiency has been linked to an increased risk of pancreatic cancer. Magnesium plays a role in maintaining pancreatic function, and its deficiency can impair the body's ability to fight cancer. Research into magnesium supplementation for pancreatic cancer is still emerging, but it shows promise for chemoprevention and therapy (Sakurai, T., *et al*. (2014) [55].

1.5. Iron

Iron is an essential mineral that plays a critical role in various physiological functions, including oxygen transport, DNA synthesis, and cellular metabolism. However, due to its involvement in the Fenton reaction (which generates reactive oxygen species), iron also has the potential to contribute to oxidative stress, a factor implicated in the initiation and progression of cancer. Interestingly, while iron is essential for cell growth, excessive or dysregulated iron metabolism has been linked to the development of several cancers.

Iron is regarded as an essential element because it plays an important role in various metabolic processes, like electron transport, oxygen transport, synthesis of deoxyribonucleic acid (DNA), during inflammation, etc. (Abbaspour *et al*., 2014) [1]. The various animal source which is a rich source of iron including, liver, red meat, shellfish, etc. and absorption of iron is influenced by ascorbic acid, citric acid, amino acids, fish, meat, chicken, etc., and inhibited by dietary fiber, protein, calcium, phytates, tannins, phenolic acid and flavonoids (Barragan-Ibanez *et al*., 2016; Anderson *et al*., 2018) [8, 5]. Plummer-Vinson syndrome risk increases due to deficiency of iron, which is responsible for upper alimentary tract tumor while the high concentration of iron may increase the risk of cancer but evidence not reported (Wessling-Resnick, 2017) [64].

Iron is essential for DNA synthesis, cellular respiration, and cell proliferation. By chelating iron, cancer cell growth can be inhibited, particularly in iron-dependent cancers. Iron chelators such as deferoxamine (DFO) and bipyridyl compounds have been studied for their ability to starve tumor cells of iron and inhibit their growth. (Torti *et al*., 2013) [62].

Iron has been shown to influence apoptotic pathways in cancer cells. Elevated levels of iron can promote the production of reactive oxygen species (ROS) via the Fenton reaction, leading to oxidative damage to lipids, proteins, and DNA. This can induce cell death through apoptosis. Iron overload can therefore increase oxidative stress, triggering apoptosis in cancer cells, while iron chelation may prevent this oxidative stress and thus have therapeutic potential in cancer treatment (Zhao, Q., *et al*. 2014) [73]. The HIF pathway regulates cellular responses to low oxygen levels (hypoxia) and is often dysregulated in cancer. Iron plays a key role in stabilizing HIF-1α, a transcription factor that promotes angiogenesis and tumor growth. By inhibiting iron metabolism, it is possible to interfere with HIF signaling and potentially reduce tumor growth and metastasis (Gao,P., *et al*., 2007).

Cancer cells have a high demand for iron to support rapid growth and proliferation. Alterations in iron metabolism, such as increased transferrin receptor expression and elevated ferritin levels, are commonly observed in cancer. Targeting these pathways, including the use of iron chelators and transferrin receptor antagonists, has shown potential in reducing cancer cell proliferation (Peyroux, M., *et al*. 2017) [49].

Iron also influences the tumor microenvironment, particularly in regulating the immune response. Cancer cells often manipulate iron metabolism to enhance their survival in the harsh tumor microenvironment. Iron regulation has been shown to modulate the activity of immune cells, such as macrophages, T cells, and natural killer cells, which play a key role in cancer progression and metastasis (Gao, W., *et al*. 2015).

It was Studied that iron metabolism plays a role in breast cancer progression. Overexpression of iron-related proteins like transferrin receptors and ferritin is commonly found in breast cancer cells. Iron chelation has shown potential in reducing cell proliferation and increasing apoptosis in breast cancer cells. Preclinical studies have explored the use of iron chelators as adjunctive therapy for breast cancer (Shen, J., *et al*. 2009) [57].

Iron overload leads to increased reactive oxygen species (ROS) production, contributing to DNA damage and tumor formation. Iron chelation has been shown to inhibit the growth of liver cancer cells and increase sensitivity to chemotherapy agents (Zhang, Y., *et al*. 2017) [71].

chelation has been proposed as a therapeutic strategy to inhibit tumor growth and metastasis in ovarian cancer. Iron chelators, such as deferasirox, have been studied for their ability to inhibit the growth of ovarian cancer cells and enhance chemotherapy efficacy (Fang, X., *et al*. 2015) [21].

1.6. Germanium

Germanium, a chemical element that is primarily used in electronics, has also attracted attention in alternative medicine for its potential anticancer properties. Organic germanium compounds, such as germanium sesquioxide (Ge-132), have been studied for their possible therapeutic benefits in cancer treatment

Nowadays germanium element used as dietary supplements found in various medicinal plants like ginger, garlic, ginseng, etc. (Lu *et al*., 1998) [39]. It was found that it reduces the multiplication of cancerous cells, acts as a powerful antioxidant, and provides protection from cancer, without affecting normal healthy cells (Kaplan *et al*., 2004) [32].

Germanium has been shown to activate natural killer (NK) cells, which play a key role in recognizing and killing tumor cells. By enhancing immune function, germanium may help the body recognize and eliminate cancer cells more effectively (Koyama, Y., *et al*. (1988) [35]. Germanium may interfere with cancer cell cycle progression and prevent cells from entering mitosis. It has been shown to have a cytotoxic effect on tumor cells, particularly by promoting mitochondrial

dysfunction, which ultimately triggers cell death pathways. (Takahashi *et al*., 1991) [60].

2. Vitamins as an Anticancer Agent

i). Vitamin A: It is also known as retinol. It was reported that vitamin A or vitamin A precursor consumption in diet, liver, or carotenoids present in green and yellow vegetables respectively, reduces the risk of cancer. The deficiency of vitamin A in the diet is responsible for carcinogenesis (Basu, *et al*., 1997).

ii). Vitamin C: It is also known as ascorbic acid. It was found that Vitamin C inhibits some carcinogenic compounds like N-nitroso compounds, so by using vitamin C-containing foods in the diet, gastric and esophageal cancer risk decreases (Glober *et al*., 1974) [25].

iii). Vitamin E: It is also known as α-Tocopherol. It is found in vegetable oils, whole-grain cereal products, eggs, etc. It inhibits the formation of nitrosamines, a carcinogenesis factor, and also inhibited tumorigenesis induces by other chemicals (Kamm *et al*., 1997).

iv). Vitamin D: Vitamin D has been linked to reduced cancer cell proliferation, apoptosis, and differentiation. It is believed to have a role in inhibiting the growth of cancer cells and inducing apoptosis (programmed cell death) (Holick *et al*., 2007) [28].

v). Vitamin B9: Folate plays a role in DNA synthesis and repair, and its deficiency is linked to the development of cancer due to improper DNA replication. However, excessive folate supplementation might also increase cancer risks, so the dosage and timing of intake are critical (Mason, J. B., *et al*. (2002) [46].

Conclusion

Micronutrients, such as vitamins, minerals, and polyphenolic compounds, play a crucial role in cancer prevention and management due to their antioxidant, anti-inflammatory, and cell-regulatory properties. These compounds can help reduce oxidative stress, support immune function, and regulate signaling pathways involved in cancer development. While these micronutrients have shown promising anticancer effects in preclinical and some clinical studies, their full potential for cancer prevention and treatment is still being explored. Micronutrients may offer protective benefits, but they are not a substitute for conventional cancer treatments, and supplementation should be approached with caution under the guidance of a healthcare provider.

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