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Beyond Prescription Pad Safeguarding Nutritional Health in Long-Term Medication use through Pharmacist-Led Intervention

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Abstract: Long-term use of prescription and over-the-counter medications can lead to both subclinical and clinically significant micronutrient deficiencies, often developing gradually over time. Despite the widespread use of medications, research on drug-nutrient interactions remains limited. This review explores the potential effects of commonly prescribed drugs on nutrient absorption, metabolism, and utilization. Medications such as proton pump inhibitors (PPIs), non-steroidal anti-inflammatory drugs (NSAIDs), metformin, diuretics, ACE inhibitors, and bronchodilators can contribute to deficiencies in essential vitamins and minerals, including B12, calcium, magnesium, and iron. Pharmacists play a crucial role in identifying these interactions, monitoring at-risk individuals, and providing guidance on supplementation and dietary adjustments to prevent deficiencies. For instance, patients using PPIs may require B12 and calcium supplementation, while NSAID users may need iron due to a higher risk of anaemia. Likewise, diuretics necessitate regular monitoring of potassium and magnesium levels, whereas ACE inhibitors may require monitoring of potassium and zinc. While routine supplementation is not generally recommended, pharmacists help ensure adequate nutrient intake through diet and targeted supplementation when necessary. This review highlights the essential role of pharmacists in optimizing drug therapy and maintaining nutritional balance, ultimately improving patient health by reducing drug-induced nutrient deficiencies.

Keywords*: Drug - Nutrient Interaction, Pharmacist-Led Interventions, Nutrient Deficiencies.*

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I. INTRODUCTION

The long-term use of prescription and over-the-counter (OTC) medications can gradually cause mild or significant deficiencies in essential nutrients. These deficiencies often develop over months or years and rarely show typical symptoms. Unfortunately, aside from the most well-known cases, many healthcare providers are not well-versed in identifying or understanding micronutrient imbalances. As a result, these deficiencies are sometimes mistaken for symptoms of aging or other health conditions, leading to delays in diagnosis [1] Deficiencies caused by medications may also explain unexplained symptoms and, in some instances, affect patients' willingness to continue their medications [2] Drug-nutrient interactions refer to the physical, chemical, or physiological relationships between medications and nutrients. These interactions often involve various factors [3] Drugs can impact how nutrients are

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consumed, absorbed, digested, distributed in the body, activated, metabolized, or excreted [4] Additionally, the interaction between specific proteins, receptors, and enzymes in tissues adds another layer of complexity, leading to many possible ways these interactions can occur [5]. This complexity makes it hard to predict how they may affect patients. Substances like alcohol and tobacco can also impact nutrient levels similarly, though this topic is beyond the scope of this review The National Center for Health Statistics, through the National Ambulatory Care Service Survey, identifies the most common chronic conditions in patients over 45 that necessitate long-term medication use. These conditions include hypertension, high cholesterol (hyperlipidemia), arthritis, diabetes, depression, asthma, coronary artery disease, and chronic obstructive pulmonary disease (COPD) [6] pharmacist play a critical role in managing drug-nutrient interactions and supporting patients with chronic conditions requiring long-term medication use. Their expertise allows them to identify and address medication-induced nutrient deficiencies, optimize treatment outcomes, and improve patient well-being. By providing education, recommending dietary adjustments or supplements, and collaborating with healthcare providers, pharmacists ensure safer and more effective medication use, especially in populations at risk of nutrient imbalances or polypharmacy.

II. MEDICATIONS MOST LIKELY TO AFFECT NUTRITIONAL STATUS:

A. Proton Pump Inhibitors (PPIS)

The main action of PPIs is to reduce gastric acid production. Thus, decreased absorption of micronutrients that depend on low pH for uptake into intestinal cells may occur with PPI use.

- *Vitamin B12:*

Vitamin B12 absorption depends on gastric acid to release it from dietary proteins, though the B12 in fortified foods and supplements does not require gastric acid. Research shows mixed evidence regarding PPIs and B12 deficiency. Long-term PPI use (≥12 months) has been linked to higher deficiency risk, even when accounting for multivitamin use [7]. However, some studies found no significant differences in B12 levels between PPI users and non-users, likely due to exclusions such as individuals receiving parenteral B12 supplements. Variability in study methods and populations may explain these inconsistencies. Certain risk factors increase vulnerability to PPI-related B12 deficiency, such as aging, H. pylori infection, and atrophic gastritis, which exacerbate impaired acid secretion [8]. Genetic polymorphisms affecting omeprazole metabolism can also raise gastric pH and decrease B12 levels in some individuals. Consuming acidic beverages with B12 has been shown to enhance absorption in PPI users [9].Although PPIs may impair protein-bound B12 absorption and elevate deficiency risk in certain groups, inconsistent findings led the American Gastroenterological Association to conclude that routine screening or supplementation is unnecessary[10].

- *Vitamin C:*

Research shows that PPI use reduces the concentration of biologically active ascorbic acid (AA) in gastric juice, especially in individuals infected with H. pylori. This is likely due to an increase in intragastric pH, which diminishes the bioavailability of AA, converting it into dehydroascorbic acid (DHAA), which is not absorbable in the intestine. Studies indicate that long-term PPI use may result in significantly lower circulating vitamin C levels, which may contribute to vitamin C deficiency in vulnerable individuals [11].

- *Iron:*

PPI therapy has the potential to impair the absorption of non-heme iron, the primary form of iron in plant-based foods. Evidence of iron deficiency with PPI use is mixed, with some studies linking chronic use to reduced iron status, especially in patients with existing iron deficiency or related conditions. PPI use is associated with a higher risk of iron deficiency over time, particularly in those already predisposed. However, short-term studies have not shown significant changes in iron absorption [12].

- *Calcium:*

Similar to vitamin C, the absorption of calcium is affected by the change in gastric pH caused by PPI use. Some studies suggest that prolonged PPI therapy may be linked to an increased risk of bone fractures, particularly in individuals already at risk, such as the elderly. Observational research shows that individuals using PPIs long-term have a modest increase in fracture risk, though the effect of PPI on calcium absorption and bone mineral density (BMD) is still debated. The National Osteoporosis Foundation considers PPI use a potential contributing factor to osteoporosis, but routine monitoring of bone health is not recommended [13].

B. NSAIDs: Aspirin

Aspirin use, particularly at high or prolonged doses, has been shown to influence vitamin C and iron levels, especially in individuals with specific health conditions, such as rheumatoid arthritis or elderly individuals.

- *Vitamin C and Aspirin Interaction:*

Early studies from the 1970s revealed that high doses of aspirin led to significantly lower platelet ascorbic acid levels in RA patients compared to controls. Follow-up studies found that 500 mg of vitamin C combined with 900 mg of aspirin reduced the vitamin C increase in leukocytes, suggesting that aspirin impedes the storage rather than the intestinal absorption of vitamin C [14]. Moreover, long-term aspirin use decreased vitamin C concentrations in plasma, urine, and gastric mucosa. This reduction was attributed to aspirin-induced gastric mucosal damage rather than impaired absorption, as aspirin increased antioxidant defenses. These findings suggest that vitamin C supplementation might help mitigate aspirin-induced gastric damage [15].

- *Aspirin and Iron Deficiency Risk:*

Chronic aspirin use is known to cause gastrointestinal damage, such as gastric ulcers and bleeding, potentially reducing iron levels and increasing the risk of iron deficiency

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anemia. Several studies have demonstrated a connection between aspirin and reduced hemoglobin (Hb) levels, particularly in older adults. However, anemia did not always develop, and Hb remained within the normal range[16]. Retrospective studies in elderly patients found that aspirin use was more prevalent among those with iron deficiency anemia. Additionally, studies revealed significantly lower serum ferritin levels (a marker of iron stores) in aspirin users, especially those infected with Helicobacter pylori, suggesting that aspirin may interfere with iron metabolism. However, more research is needed to fully determine the clinical impact of aspirin on iron deficiency[17].

C. Oral Hypoglycemic Drugs:

Metformin, a frequently prescribed oral hypoglycemic for Type 2 Diabetes, has been consistently linked with a reduction in vitamin B12 levels, with this effect being dose- and duration-dependent.

➢ *Vitamin B12 Deficiency and Metformin Use:*

Metformin is believed to interfere with calcium-dependent processes in the intestine, impairing the absorption of vitamin B12, which requires the intrinsic factor for its absorption. In one study, calcium supplementation (1.2 g/dl for one month) in metformin users helped reverse the observed B12 malabsorption, indicated by increases in serum B12 and Holotranscobalamin levels [18]. Several cross-sectional studies across diverse countries, such as the U.S., Korea, the Netherlands, and Brazil, have established that T2D patients on metformin tend to have lower serum or plasma B12 levels when compared to both healthy controls and T2D patients not using the medication. This reduction in B12 was particularly marked when metformin was used in combination with sulfonylurea as opposed to metformin alone, with deficiency prevalence ranging from 6% to 28%, depending on the deficiency criteria used [19]. Furthermore, studies that incorporated biomarkers such as methylmalonic acid (MMA) and homocysteine (Hcy) indicated higher levels of MMA and Hcy in patients on metformin, signifying impaired vitamin B12 utilization. Nevertheless, not all studies found significant shifts in MMA levels in response to metformin [20].

➢ *Effects on Homocysteine and Folate*

A randomized controlled trial (RCT) conducted over 16 weeks demonstrated a 14% reduction in serum B12 levels and a 4% increase in Hcy levels in metformin users, while folate levels also declined by 7% [21]. These findings suggest that metformin's impact on elevated Hcy may not solely be attributable to vitamin B12 depletion but also to decreased folate status. In a longer study, metformin use continued to lower B12 and folate levels in Type 2 diabetes patients using insulin [22].Considering the association between metformin use and vitamin B12 deficiency, routine monitoring of B12 levels is recommended for patients on long-term metformin therapy, especially those at an elevated risk of deficiency, such as the elderly and vegetarians. The concomitant use of a multivitamin alongside metformin may help prevent B12 depletion [23]. More studies incorporating functional markers of B12 status are necessary to further clarify the clinical relevance of this interaction.

D. Anti-Hypertensives:

➢ *Diuretics:*

- *Calcium and Loop Diuretics:*

Loop diuretics, including furosemide and bumetanide, significantly enhance calcium excretion. These diuretics achieve this by inhibiting calcium reabsorption in the thick ascending limb of the loop of Henle. This process disrupts the trans epithelial voltage, which typically drives calcium-ion transport in the kidney [24]. As a result, loop diuretics lead to an increase in urinary calcium loss and often a subsequent rise in plasma parathyroid hormone (PTH).In a study involving postmenopausal osteopenic women, the urinary calcium excretion and plasma PTH levels were shown to increase in response to varying doses of bumetanide (0.5–2.0 mg/d). This relationship indicates that loop diuretics have a dose-dependent effect on calcium homeostasis [25].The effects of loop diuretics on bone mineral density (BMD) have been extensively studied, with mixed results. In one observational study, elderly women who used loop diuretics exhibited significantly reduced hip BMD after adjusting for age, menopause duration, and body weight [26]. However, a contrasting case-control study found no difference in BMD between long-term loop diuretic users and nonusers, despite increased urinary calcium excretion. The study suggested that increased 1,25-dihydroxyvitamin D levels in diuretic users might offset renal calcium loss through enhanced intestinal calcium absorption, thereby preventing major bone metabolism disturbances [27].over one year led to a 2% decrease in BMD and an increase in bone turnover markers, despite supplementation with calcium (800 mg/day) and vitamin D (10 µg/day). This suggests a negative impact on bone health from loop diuretic use, despite attempts at compensation through dietary supplementation [28]. Several studies have linked loop diuretic use to an increased risk of fractures, particularly in elderly populations. A case-control study involving patients hospitalized for hip fractures found a 9-fold greater risk for users of furosemide compared to non users . Additionally, cohort studies have found long-term loop diuretic use to be associated with an elevated risk of osteoporotic fractures, particularly hip fractures in older adults [29].

- *Calcium and Thiazide Diuretics***:**

Thiazide diuretics mainly affect the early distal tubule in the kidney, enhancing calcium reabsorption and reducing its excretion into the urine. This mechanism of action is primarily responsible for the increase in calcium retention observed during thiazide use. In addition, thiazides exert their effects through a PTH dependent pathway, with studies showing that thiazide administration reduces urinary calcium in individuals with hyperparathyroidism, but not in those with hypoparathyroidism [30].Thiazide diuretics, including hydrochlorothiazide, show a dose-dependent reduction in urinary calcium excretion. Specifically, a range of 2.5–10 mg/day has been found effective in reducing calcium in the urine. Additionally, thiazide treatment results in elevated plasma osteocalcin, a marker associated with bone formation, although no substantial changes were observed in BMD in some clinical studies, such as one in healthy older women

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[31].Although RCTs examining the effects of thiazides on BMD and fracture risk are limited, observational studies suggest a reduced risk of hip fractures among long-term thiazide users, with reductions reported to be between 18%–24%. One study indicated that long-term use of thiazides offers more substantial protection against fractures than short-term use, though the data remain inconclusive and further RCTs are necessary to confirm the bone-protective effects of these medications.Chronic use of thiazide diuretics, particularly in older women, has been associated with an increased risk of elevated serum calcium levels. Although hypercalcemia is uncommon, an age- and sex-adjusted population study found that thiazide use leads to mild and non-progressive hypercalcemia in a substantial number of elderly individuals. Conversely, a study investigating thiazide use with vitamin D supplementation showed that users had higher serum calcium levels, but only one participant developed serum calcium levels severe enough to be classified as hypercalcemic [32].

- *Thiamin and Diuretics:*

Loop diuretics, such as furosemide, have been shown to increase urinary thiamin excretion, a phenomenon correlated with urine flow rate, suggesting that the loss of thiamin is not unique to a specific diuretic but rather due to the sustained diuresis induced by these drugs [33]. Thiamin deficiency, especially in individuals with congestive heart failure who are on diuretics, has been observed to be significantly more prevalent compared to age-matched controls, and this deficiency risk escalates with higher doses of furosemide [34].The elderly population is particularly vulnerable to thiamin deficiency due to both diuretic usage and insufficient dietary intake. One study found that older adults on furosemide therapy had a noticeable reduction in thiamin status, and this effect was associated with the cumulative dose of furosemide administered during their hospital stay [35]. Moreover, a study involving 324 homebound elderly individuals revealed that diuretic users were considerably more likely to have intakes below the recommended dietary allowance for thiamin, regardless of other variables such as meal patterns and sociodemographic factors [36].Thus, prolonged use of loop diuretics, especially furosemide, poses a substantial risk of thiamin deficiency, particularly in the elderly, which may necessitate intervention with thiamin supplementation to maintain adequate nutritional status.

- *Potassium and Diuretics:*

Loop and thiazide diuretics both lead to increased urinary potassium excretion, with thiazides often causing hypokalemia more frequently. Thiazide diuretics induce renal potassium secretion through various mechanisms, whereas loop diuretics inhibit potassium reabsorption in the loop of Henle [37]. Research suggests that the risk of hypokalemia is positively associated with thiazide dosage. For example, in a study from the UK, hypokalemia occurred in 8.5% of patients on thiazides, and a dose-response relationship between thiazides and low serum potassium was identified. Among the thiazides, bendroflumethiazide was found to be the most potent in reducing potassium, while hydrochlorothiazide was the least potent. Thiazide-induced hypokalemia has been linked to increased blood glucose and, in some cases, ventricular arrhythmias [38]. Despite potassium supplementation, diuretic users have shown significantly lower muscle potassium concentrations compared to controls, and even when potassium supplements are provided, they help prevent hypokalemia but don't necessarily restore normal potassium levels completely. However, potassium supplements can be beneficial in preventing hypokalemia symptoms from chronic diuretic use, though they may also cause additional blood pressure reduction, potentially leading to hypotension [39]

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E. Zinc and Potassium in ACE Inhibitor use

➢ *Zinc*:

Long-term use of ACE inhibitors, particularly captopril, has been implicated in hypogeusia, which is associated with zinc deficiency. Studies indicate that hypertensive patients on high-dose (266 mg/day) captopril treatment (>6 months) displayed higher taste detection and recognition thresholds, lower plasma zinc levels, and increased urinary zinc excretion compared to controls, suggesting that captopril affects zinc status. Conversely, short-term (less than 6 months) use of lower doses (100 mg/day) did not significantly alter zinc levels. Furthermore, in individuals with kidney disease or heart failure, lower doses (50 mg/day) of captopril have shown to diminish zinc levels [40]. The thiol-radical group in captopril may chelate serum zinc and promote its excretion, contributing to zinc deficiency. Evidence suggests that captopril has a more significant effect on zinc depletion than other ACE inhibitors like enalapril, with implications for patients with co-existing conditions such as heart failure, renal disease, or malabsorption. However, serum zinc levels alone may not reflect tissue distribution, necessitating further research on captopril's broader impact on zinc [41].

➢ *Potassium*:

ACE inhibitors, through their inhibition of aldosterone secretion, promote potassium retention in the kidneys, which increases the likelihood of hyperkalemia. Though the incidence of hyperkalemia is low (1–2%) in hypertensive patients on ACE inhibitors, certain factors exacerbate the risk. Studies report that older adults, those with renal failure, diabetes, congestive heart failure, and individuals consuming high-potassium diets or potassium supplements are more susceptible to hyperkalemia while on ACE inhibitors[42]. A study found that patients on enalapril for 40 months exhibited a three-fold increased rate of hyperkalemia compared to placebo. The use of potassium-sparing diuretics or potassium-rich foods supplements can further elevate this risk. Thus, while ACE inhibitors effectively manage hypertension, their impact on potassium homeostasis requires careful monitoring, particularly in at-risk populations [43].

F. Bronchodilators: Beta2-Agonists and Inhaled Corticosteroids (ICS)

➢ *Calcium and Vitamin D:*

The impact of beta2-agonists on bone health has been inadequately explored in human studies, though one population-based case-control study linked higher doses with

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an increased risk of hip and femur fractures. This risk was diminished once oral glucocorticoid use and underlying disease were accounted. A randomized trial on mild asthmatics using beta2-agonists revealed a negative association between inhaled corticosteroids (ICS) dose and lumbar spine BMD, while no such link was found in the non-steroid group[44].Concerning long-term ICS use (≥12 months), the research remains inconclusive. One systematic review found that ICS therapy may influence markers of bone metabolism and BMD in asthmatic and COPD patients, as well as healthy adults [45]. A meta-analysis affirmed that higher ICS doses can elevate bone turnover, though no clear relationship with fracture risk was observed. Lower doses, however, did not exhibit these effects. An industry-sponsored meta-analysis revealed that BMD loss among asthma and COPD patients did not significantly differ from healthy individuals . Meanwhile, a more recent systematic review excluding COPD patients found no significant correlation between long-term ICS use and BMD or fracture risk in asthmatic adults and children [46].ICS usage may affect bone metabolism and BMD, especially in patients with COPD, who have pre-existing risk factors such as smoking, systemic inflammation, and cachexia. Importantly, no studies have investigated the role of calcium and vitamin D supplementation in preventing bone deterioration for ICS users, representing a significant gap in research. Notably, only 17% of ICS users over 50 report taking calcium and vitamin D supplements [47].

III. PHARMACIST'S ROLE IN MANAGING DRUG-NUTRIENT INTERACTIONS

Pharmacist-led interventions are important in managing drug-nutrient interactions to optimize patient health outcomes, particularly when patients are on multiple medications that can cause nutrient imbalances. Several common medications such as proton pump inhibitors (PPIs), non-steroidal anti-inflammatory drugs (NSAIDs), oral hypoglycemics (like metformin), diuretics, ACE inhibitors, and bronchodilators are known to interfere with the absorption, metabolism, and utilization of essential nutrients, potentially leading to deficiencies. As healthcare professionals, pharmacists play a vital role in identifying these interactions and providing proactive advice to mitigate nutrient depletion.For instance, PPIs, commonly used for gastric acid-related conditions, have been shown to reduce the absorption of vitamin B12, calcium, and magnesium. In such cases, pharmacists must monitor the patient's nutrient status and advise on the supplementation of vitamin B12 and calcium, as well as increasing intake of magnesium-rich foods to avoid long-term deficiencies [48]. Furthermore, NSAIDs, frequently prescribed for pain and inflammation, can cause gastrointestinal bleeding, which may lead to iron deficiency anemia. Pharmacists should recommend routine screening for anemia and suggest appropriate supplementation with iron and vitamin C to enhance absorption and support red blood cell production [49].Metformin, a first-line treatment for type 2 diabetes, is known to decrease vitamin B12 levels, leading to possible neuropathy and other neurological issues over time. For patients using this medication, pharmacists must regularly monitor B12 levels and consider recommending B12 supplementation to prevent complications associated with its deficiency [50]. In patients taking diuretics, such as furosemide or hydrochlorothiazide, potassium, magnesium, and thiamine levels may drop significantly. Diuretic therapy has been associated with an increased risk of electrolyte imbalances, and pharmacists should monitor potassium and magnesium levels regularly, suggesting appropriate supplementation and advising on dietary adjustments to maintain balance [51].ACE inhibitors, such as captopril and enalapril, are often prescribed to manage hypertension and heart failure. However, these medications can lead to hyperkalemia and a potential zinc deficiency, which can compromise immune function. Pharmacists should monitor serum potassium levels closely and provide counseling on dietary modifications to prevent excessive potassium intake. Moreover, they can recommend zinc supplementation where necessary, particularly in patients at risk of malnutrition [52]. Finally, long-term use of bronchodilators (including beta-agonists) and inhaled corticosteroids has been linked to osteoporosis and reduced bone mineral density. Pharmacists should assess bone health in such patients and advise supplementation with calcium and vitamin D, as well as recommend weight-bearing exercises to counteract bone loss associated with chronic medication use [53].Through personalized assessments and recommendations, pharmacists are essential in preventing nutrient deficiencies, managing drug interactions, and improving medication adherence, which ultimately enhances patient outcomes. Their expertise ensures that drug therapy is optimized not only for its therapeutic effect but also for maintaining the overall nutritional health of patients, minimizing potential complications due to nutrient deficiencies.The below table-1 provides comprehensive nutritional recommendations to address common vitamin and mineral deficiencies that impact overall health. It highlights key sources of Vitamin B12, Vitamin C, Calcium, Iron, Zinc, and Vitamin D, including animal-based, plant-based, fortified options, and supplementation. For patients at risk of drug induced vitamin deficiencies, this table can guide dietary choices and interventions tailored to their specific needs. By incorporating these evidence-based dietary recommendations, patients can improve nutrient intake, support immune function, strengthen bones, and boost overall well-being.

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Table 1 Nutritional Sources

Nutrient	Animal- Based Sources	Plant-Based Sources	Fortified Foods	Other Sources	References
Vitamin B12	Beef liver, chicken, salmon, trout, eggs, dairy	None (except fermented foods like tempeh, unreliable)	Fortified cereals, nutritional yeast, fortified plant-based milk (soy, almond, oat)	Supplements: Cyanocobalamin or Methylcobalamin	NIH Office of Dietary Supplements - Vitamin B12
Vitamin C	None (Vitamin C is plant-based)	Citrus fruits (oranges, lemons), bell peppers, kiwi, strawberries, kale	Some fortified juices	None	NIH Office of Dietary Supplements - Vitamin C
Calcium	Dairy (milk, yogurt, cheese), sardines (with bones), salmon	Kale, bok choy, almonds, sesame seeds, tofu, broccoli	Fortified plant-based milk, orange juice, cereals	Supplements or calcium-fortified water	NIH Office of Dietary Supplements - Calcium
Iron	Red meat, poultry, seafood (clams, oysters, shrimp)	Lentils, beans, quinoa, tofu, spinach, pumpkin seeds	Fortified cereals	Pair non-heme sources with Vitamin C for better absorption	NIH Office of Dietary Supplements - Iron
Zinc	Oysters, crab, lobster, red meat, poultry	Chickpeas, lentils, cashews, pumpkin seeds, whole grains (oats, quinoa)	Fortified breakfast cereals	Zinc supplements (if needed in deficiencies)	NIH Office of Dietary Supplements - Zinc
Vitamin D	Fatty fish (salmon, mackerel, sardines), cod liver oil	Limited (some mushrooms like maitake provide Vitamin D2)	Fortified milk, orange juice, cereals, plant-based milk	Sunlight (15-30 mins daily); Vitamin D2 or D3 supplements	NIH Office of Dietary Supplements - Vitamin D

Table of nutritional recommendations to address common vitamin and mineral deficiencies that impact overall health

IV. CONCLUSION

In conclusion, drug-nutrient interactions are a significant but often overlooked aspect of managing long-term medication use. As many patients require chronic treatments for conditions such as hypertension, diabetes, and arthritis, understanding how medications influence the absorption, metabolism, and excretion of vital nutrients is crucial. Over time, common medications like PPIs, NSAIDs, oral hypoglycemics such as metformin, and diuretics can deplete essential nutrients, leading to deficiencies that may remain undiagnosed. These deficiencies can manifest in the form of anemia, bone fractures, neurological issues, and cardiovascular complications, complicating the management of the primary health condition.The role of pharmacists in addressing these drug-nutrient interactions is essential. Pharmacists are positioned to monitor nutrient status in patients using long-term medications, identify potential deficiencies, and advise on corrective measures, such as dietary modifications and supplementation. By working closely with other healthcare providers and patients, pharmacists ensure that drug therapy is optimized to not only treat the medical condition but also maintain nutritional balance, ultimately improving patient outcomes. In doing so, pharmacists help mitigate the risks of chronic medication use, preventing unnecessary health complications and promoting overall well-being. This highlights the importance of an integrated healthcare approach that considers both medical and nutritional factors, ensuring that patients receive the most comprehensive care.

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