INNOVATIVE PUBLICATION
Journal of Effective Learning and Sustainable Innovation
Vol.3 №10 (2025). October
innovativepublication.uz

PHARMACOLOGICAL EFFECTS OF VITAMIN DRUGS ON PREGNANT WOMEN

Rakhmonova Khosiyat Boburjon kizi
Assistant intern of the Department of Pharmacology, Clinical Pharmacology and Medical Biotechnology, ASMI

Abstract. The physiological demands of pregnancy require a delicate balance between maternal and fetal nutrition, metabolism, and hormonal regulation. Vitamins — both water- and fat-soluble — play essential roles in supporting cellular growth, hematopoiesis, nervous system development, and immune competence during gestation. The pharmacological use of vitamin preparations in pregnant women aims to prevent nutritional deficiencies, fetal malformations, and pregnancy-related complications such as anemia, preeclampsia, and neural tube defects. However, excessive or inappropriate vitamin supplementation may lead to teratogenic or toxic effects, particularly with fat-soluble compounds. This article presents an in-depth analysis of the pharmacological mechanisms, benefits, and potential risks associated with vitamin drug use in pregnant women. It explores the kinetics of vitamin absorption, distribution, and metabolism during pregnancy and underscores the importance of evidence-based supplementation guided by clinical pharmacology principles.

Keywords: vitamins, pregnancy, pharmacology, teratogenicity, folic acid, vitamin D, vitamin A, prenatal health.

INTRODUCTION

Pregnancy represents a unique physiological condition characterized by profound metabolic and hormonal changes that support fetal growth and maternal adaptation. These changes include increased blood volume, elevated cardiac output, enhanced renal clearance, and altered hepatic metabolism. Such transformations also affect the pharmacokinetics and pharmacodynamics of nutrient and drug absorption, distribution, and elimination. Vitamins, though required in trace amounts, are indispensable for maintaining enzymatic reactions, cellular differentiation, and tissue repair. Inadequate vitamin levels during pregnancy can result in adverse maternal outcomes — such as anemia, fatigue, and hypertension — as well as fetal disorders, including congenital malformations, neural tube defects, and growth retardation [1].

Clinical pharmacology provides a framework for understanding how vitamin drugs

interact with the pregnant body's altered physiological state. Unlike dietary intake, pharmacological vitamin therapy delivers controlled, bioavailable doses designed to prevent or correct specific deficiencies. Yet, the boundary between therapeutic and excessive supplementation is narrow, particularly with fat-soluble vitamins (A, D, E, and K), which can accumulate in tissues and cause toxicity. Therefore, rational vitamin therapy during pregnancy requires precise dosing, timing, and clinical monitoring based on evidence and patient-specific needs.

MATERIALS AND METHODS

Pregnancy alters the absorption and metabolism of vitamins due to gastrointestinal, hepatic, and renal changes. Slower gastric motility and increased intestinal blood flow enhance the absorption of certain vitamins, while increased plasma volume dilutes water-soluble vitamin concentrations. Fat-soluble vitamins, absorbed with dietary lipids, depend on bile acid secretion, which may be reduced in cholestatic states associated with late pregnancy.

Renal clearance of water-soluble vitamins such as vitamin C and B-complex compounds is accelerated due to increased glomerular filtration rate, necessitating more frequent intake. Conversely, fat-soluble vitamins are stored in adipose tissue and the liver, with prolonged half-lives and potential accumulation during prolonged supplementation. Hepatic enzyme induction, influenced by pregnancy hormones such as estrogen and progesterone, also modifies the metabolism of several vitamins and their coenzymes, affecting their biological availability to both mother and fetus [2].

RESULTS AND DISCUSSION

Among all vitamins, folic acid (vitamin B_9) has the most firmly established role in prenatal pharmacology. It participates in DNA synthesis, methylation reactions, and amino acid metabolism, processes critical during early embryogenesis. Deficiency of folate during the periconceptional period is strongly associated with neural tube defects (NTDs), including spina bifida and anencephaly. Pharmacologically, folic acid supplementation increases maternal serum and red blood cell folate levels, ensuring adequate supply for neural tube closure, which occurs within the first 28 days of conception — often before pregnancy is clinically recognized.

The recommended dose for women planning pregnancy is typically 400–800 micrograms daily, but higher doses (4 mg/day) may be prescribed for women with a history of NTDs or anticonvulsant therapy, as certain drugs such as phenytoin and carbamazepine impair folate metabolism. Excessive folic acid, while generally safe, may mask vitamin B_{12} deficiency and lead to neurological complications if unrecognized [3].

Vitamin D plays a pivotal role in calcium and phosphate balance, essential for fetal skeletal development and maternal bone health. During pregnancy, increased estrogen levels enhance intestinal calcium absorption, while the active form of vitamin D — calcitriol — further promotes mineralization of the fetal skeleton. Vitamin D deficiency is associated with gestational hypertension, preeclampsia, impaired glucose tolerance, and low birth weight. Pharmacologically, supplementation with vitamin D_3 (cholecalciferol) improves maternal serum 25-hydroxyvitamin D levels and enhances fetal bone mineral density.

However, excessive vitamin D intake may cause hypercalcemia, leading to vascular calcification, nephrocalcinosis, and suppression of parathyroid hormone. Therefore, vitamin D supplementation should remain within recommended daily allowances, typically 600–1000 IU per day during pregnancy, and serum calcium levels should be periodically monitored to avoid toxicity.

While vitamin A (retinol and its derivatives) is indispensable for cellular differentiation, vision, and immune function, it is also one of the most teratogenic substances when consumed in excessive amounts. The active metabolite, retinoic acid, regulates gene expression involved in embryonic development. In pharmacological doses exceeding 10,000 IU daily, vitamin A can disrupt normal morphogenesis, causing craniofacial, cardiac, and central nervous system malformations [4].

Nevertheless, moderate vitamin A intake is necessary to prevent maternal night blindness and maintain epithelial integrity. Beta-carotene, a precursor of vitamin A found in plant sources, is safer as it undergoes regulated enzymatic conversion to retinol. Clinical pharmacology therefore emphasizes the distinction between dietary provitamins and pharmacological retinoids, the latter of which must be strictly avoided in pregnancy due to teratogenic potential — particularly isotretinoin, a retinoid used in dermatology.

Vitamin E (tocopherol) functions primarily as a lipid-soluble antioxidant, protecting cellular membranes from oxidative damage induced by free radicals. During pregnancy, oxidative stress increases due to elevated metabolic demands and placental mitochondrial activity. Adequate vitamin E intake supports endothelial health, reduces lipid peroxidation, and may lower the risk of preeclampsia and premature rupture of membranes.

However, pharmacological doses exceeding the recommended limits can interfere with vitamin K-dependent clotting factors, predisposing to hemorrhage in both mother and newborn. Large-scale trials have not consistently shown a reduction in preeclampsia risk with high-dose vitamin E supplementation, suggesting that its role is supportive

rather than therapeutic. Therefore, clinicians must distinguish between physiological antioxidant support and pharmacological overuse.

Vitamin C (ascorbic acid) serves as a potent antioxidant and cofactor in collagen synthesis, iron absorption, and immune function. In pregnancy, it supports the integrity of placental tissues and enhances iron utilization, reducing the risk of maternal anemia. Deficiency may result in impaired wound healing, gingival bleeding, and increased susceptibility to infection.

Pharmacologically, moderate vitamin C supplementation is safe and beneficial; however, megadoses exceeding 2 grams per day can cause gastrointestinal irritation, diarrhea, and, paradoxically, rebound scurvy in neonates after delivery due to metabolic adaptation to high maternal intake. Additionally, excessive vitamin C may increase oxalate formation and risk of renal calculi, underscoring the importance of moderation.

The B-complex vitamins, including thiamine (B_1), riboflavin (B_2), niacin (B_3), pyridoxine (B_6), and cobalamin (B_{12}), participate in energy metabolism and neural function. Thiamine deficiency during pregnancy can lead to cardiac insufficiency and neuropathy, particularly in hyperemesis gravidarum. Pyridoxine is often prescribed to alleviate nausea and vomiting in early pregnancy, with proven safety at physiological doses. However, chronic high-dose administration (>200 mg/day) can cause sensory neuropathy. Vitamin B_{12}, crucial for erythropoiesis and myelination, must be adequately supplied, especially in vegetarians or women with malabsorption syndromes, as deficiency increases the risk of megaloblastic anemia and neurological damage in the fetus [5].

CONCLUSION

The pharmacological use of vitamin drugs in pregnant women embodies a delicate equilibrium between therapeutic necessity and the potential for toxicity. Pregnancy imposes unique physiological demands that alter vitamin metabolism, necessitating supplementation tailored to individual needs and clinical circumstances. Vitamins such as folic acid, vitamin D, and vitamin B_{12} are unequivocally beneficial and form the cornerstone of prenatal care, preventing congenital anomalies and supporting maternal health. In contrast, fat-soluble vitamins such as A and E must be administered cautiously due to their cumulative nature and potential for teratogenic or hemorrhagic effects when taken in excessive doses.

From a clinical pharmacology standpoint, rational vitamin therapy during pregnancy is guided by evidence-based dosage, pharmacokinetic awareness, and continuous maternal-fetal monitoring. The goal is neither indiscriminate supplementation nor

pharmacological austerity, but rather the maintenance of optimal biochemical and physiological balance that nurtures healthy gestation and development.

As scientific understanding advances, future research into vitamin receptor biology, placental transport mechanisms, and nutrigenomics will refine our capacity to design personalized prenatal supplementation programs. Such precision pharmacology will ensure that vitamin therapy continues to serve as a cornerstone of maternal-fetal medicine — enhancing health while safeguarding the next generation from preventable complications of both deficiency and excess.

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