

DRUG-DRUG INTERACTIONS BETWEEN DANDELION ROOT (*Taraxacum officinale*) AND TENOFOVIR: A COMPREHENSIVE REVIEW

A Review Article

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ABSTRACT

The concurrent use of herbal supplements and antiretroviral drugs is increasingly prevalent, particularly among individuals living with HIV/HBV, raising important concerns about herb-drug interactions (HDIs). Dandelion root (*Taraxacum officinale*) is one of the most widely consumed herbal products worldwide, valued for its diuretic, hepatoprotective, anti-inflammatory, and antioxidant properties. Tenofovir, available as tenofovir disoproxil fumarate (TDF) and tenofovir alafenamide (TAF), is a cornerstone nucleotide reverse transcriptase inhibitor used in the management of HIV and hepatitis B virus (HBV) infections. This review critically evaluates the pharmacokinetic and pharmacodynamic mechanisms by which dandelion root may interact with tenofovir, with emphasis on renal transporter-mediated interactions, diuretic effects on drug elimination, P-glycoprotein modulation, and shared nephrotoxic potential. Available evidence from in vitro studies, animal models, and clinical case reports is synthesized to provide clinicians with a comprehensive risk framework. The review concludes with clinical recommendations and proposes areas for future investigation.

Keywords: *Taraxacum officinale*, tenofovir, drug-drug interactions, herb-drug interactions, pharmacokinetics, nephrotoxicity, P-glycoprotein, OAT transporters, antiretroviral therapy, HIV.

1. INTRODUCTION

The global use of herbal and complementary medicines has grown substantially over the past two decades. Surveys across multiple countries indicate that 20–30% of patients taking prescription medications concurrently use herbal remedies, often without disclosing this to their healthcare providers [1,2]. This practice poses significant risks of herb-drug interactions (HDIs), particularly in vulnerable populations such as those undergoing antiretroviral therapy (ART) for HIV infection.

Dandelion (*Taraxacum officinale* Weber ex Wiggers, family Asteraceae) is among the most extensively used medicinal herbs worldwide. Its root, leaf, and flower possess diverse bioactive compounds including sesquiterpenoids, flavonoids (luteolin, kaempferol), phenolic acids, triterpenoids (taraxasterol, beta-sitosterol), inulin, and potassium-rich minerals [3,4]. Historically employed across European, Chinese, and Ayurvedic traditions, dandelion root is taken for liver and digestive support, diuresis, blood sugar modulation, and anti-inflammatory purposes [5].

Tenofovir, introduced as tenofovir disoproxil fumarate (TDF) in 2001 and subsequently reformulated as tenofovir alafenamide (TAF), represents one of the most widely prescribed antiretroviral drugs globally. It acts as a nucleotide reverse transcriptase inhibitor (NtRTI) and is a backbone component in first-line ART regimens for HIV and treatment of hepatitis B virus (HBV) [6]. Unlike many antiretrovirals, tenofovir is not significantly metabolized by cytochrome P450 (CYP450) enzymes; instead, it is primarily eliminated unchanged via renal tubular secretion through organic anion transporters (OAT1, OAT3) and efflux by multidrug resistance proteins (MRP2, MRP4) [7,8].

The interaction potential between dandelion root and tenofovir has not been formally studied in clinical trials. However, mechanistic understanding of their pharmacological properties reveals several plausible interaction pathways. This review synthesizes available evidence to characterize these interactions and provide guidance to clinicians managing patients who concurrently use both.

2. BOTANY AND PHYTOCHEMISTRY OF DANDELION ROOT

2.1 Botanical Description

Taraxacum officinale is a perennial herb of the Asteraceae family, native to Europe and widely naturalized across temperate regions of the northern hemisphere. The plant features deeply toothed basal leaves, hollow stems exuding a milky latex, and solitary yellow capitula inflorescences. The taproot is fleshy, brownish, and rich in bioactive constituents that vary with season, growing conditions, and geographic origin [9].

2.2 Active Phytochemical Constituents

The dandelion root is characterized by a rich array of pharmacologically active compounds that underpin both its therapeutic uses and interaction potential. The major constituent classes include:

Sesquiterpenoid lactones (taraxinic acid beta-D-glucopyranosyl ester, taraxacolide glucoside) impart the characteristic bitter taste and exhibit anti-inflammatory activity. Triterpenoids and sterols (taraxasterol, beta-sitosterol, stigmasterol) possess antioxidant and anti-inflammatory properties [4,10]. Flavonoids including luteolin and luteolin-7-O-glucoside suppress iNOS and COX-2 expression [11]. Phenolic acids (caffeic acid, chlorogenic acid) contribute hepatoprotective and antioxidant effects. Inulin, a prebiotic polysaccharide abundant in the root, may influence gut absorption processes. Potassium is present in significant quantities, relevant to diuretic drug interactions [12]. Coumarins and carotenoids round out a complex phytochemical profile.

These constituents interact with multiple pharmacological targets, including diuretic pathways, hepatic metabolizing enzymes, and renal transporters — all potentially relevant to tenofovir pharmacokinetics.

3. PHARMACOLOGY OF TENOFOVIR

3.1 Mechanism of Action

Tenofovir is a nucleotide analog of adenosine 5'-monophosphate. After cellular uptake, it undergoes phosphorylation to tenofovir diphosphate, which competitively inhibits HIV reverse transcriptase and HBV polymerase by acting as a chain terminator in DNA synthesis. This mechanism is unique to NtRTIs and distinguishes tenofovir from nucleoside reverse transcriptase inhibitors (NRTIs), which require two rather than one phosphorylation step for activation [6].

3.2 Pharmacokinetic Profile of TDF and TAF

Tenofovir disoproxil fumarate (TDF) is an orally bioavailable prodrug hydrolyzed by plasma and tissue esterases to tenofovir. Following oral administration, TDF achieves peak plasma concentrations in 1–2 hours, with bioavailability of approximately 25% in the fasted state (enhanced to ~40% with a high-fat meal). Plasma protein binding is less than 1%, and the apparent volume of distribution is approximately 1.2 L/kg [13].

Tenofovir alafenamide (TAF) is a more recent phosphonamidate prodrug that delivers tenofovir more selectively to lymphoid tissues and hepatocytes via intracellular metabolism by cathepsin A and carboxylesterase-1. TAF achieves 90% lower plasma concentrations of tenofovir compared to TDF at its respective therapeutic dose (25 mg vs. 300 mg), conferring a better renal and bone safety profile [14].

3.3 Elimination and Renal Transport

A defining pharmacokinetic feature of tenofovir is its predominantly renal elimination. Unlike most antiretrovirals, tenofovir is NOT metabolized by CYP450 enzymes and does not induce or inhibit CYP3A4, CYP2D6, or other major hepatic enzymes [7,15]. Approximately 70–80% of tenofovir is excreted unchanged in urine via a combination of glomerular filtration and active proximal tubular secretion [8].

The proximal tubular secretion of tenofovir involves two major transport steps: (i) basolateral uptake from peritubular capillaries into proximal tubular cells mediated primarily by OAT1 (SLC22A6) and secondarily by OAT3 (SLC22A8); and (ii) apical efflux into the tubular lumen mediated by MRP2 (ABCC2) and MRP4 (ABCC4) [16,17]. Disruption of either transporter step — by accumulation within tubular cells (increased OAT1 uptake) or decreased apical secretion (MRP4 inhibition) — can result in elevated intracellular tenofovir concentrations, mitochondrial DNA depletion, and proximal tubular toxicity [18].

For TAF, absorption involves P-glycoprotein (P-gp/ABCB1) and breast cancer resistance protein (BCRP/ABCG2) as substrates; therefore, P-gp inducers reduce TAF bioavailability, whereas P-gp inhibitors may increase it [14].

4. MECHANISMS OF DRUG-DRUG INTERACTION

4.1 Pharmacokinetic Interactions

4.1.1 Diuretic Effects and Altered Renal Clearance

Dandelion root exhibits clinically documented diuretic activity, principally attributed to its high potassium content, sesquiterpenoids, and flavonoids. A human study by Clare et al. demonstrated significant increases in urinary frequency and volume following administration of aqueous dandelion leaf extract, validating its traditional use as a diuretic [19]. Because tenofovir is eliminated primarily through the kidney, any substantial alteration in urine volume or glomerular filtration rate (GFR) may affect tenofovir plasma concentrations and renal exposure.

Increased urine output induced by dandelion root could theoretically accelerate tenofovir urinary excretion, potentially reducing its plasma concentrations and compromising antiviral efficacy. Conversely, the diuretic-induced reduction in effective circulating volume may, paradoxically,

reduce GFR, slowing tenofovir elimination and raising plasma levels. The net effect would depend on the degree of diuresis, baseline renal function, and hydration status of the patient.

4.1.2 Renal Transporter-Mediated Interactions

The most clinically significant potential interaction between dandelion root constituents and tenofovir involves the OAT and MRP renal transporter systems. Several dandelion phytochemicals — including flavonoids (luteolin, kaempferol) and phenolic acids — have been documented to modulate organic anion transporter function [20]. Luteolin, in particular, has been shown to inhibit OAT1-mediated transport in vitro. If dandelion constituents inhibit OAT1 in the renal proximal tubule, uptake of tenofovir into tubular cells may be reduced, potentially decreasing renal secretion and increasing plasma tenofovir concentrations.

Alternatively, if dandelion phytochemicals inhibit the apical efflux transporters MRP4 or MRP2, tenofovir may accumulate intracellularly in proximal tubular cells. Kohler et al. demonstrated that OAT1 and MRP4 are key regulators of tenofovir renal proximal tubular toxicity [16]. Intracellular tenofovir accumulation resulting from impaired efflux has been mechanistically linked to mitochondrial DNA depletion and tubular dysfunction [18]. Co-administration of dandelion root in patients on TDF could therefore amplify nephrotoxic risk if these transporter pathways are affected.

4.1.3 P-Glycoprotein Modulation

P-glycoprotein (P-gp) is an ATP-binding cassette efflux transporter expressed at the intestinal epithelium, blood-brain barrier, renal tubules, and hepatocytes. TAF (but not TDF) is a substrate of P-gp; therefore, compounds modulating P-gp activity can significantly affect TAF absorption. Dandelion flavonoids, particularly quercetin and luteolin, have been documented to inhibit P-gp in vitro [21]. P-gp inhibition by dandelion root could increase intestinal absorption of TAF, leading to higher systemic plasma concentrations and potentially increased risk of adverse effects. Conversely, dandelion triterpenoids and bitter sesquiterpenoids may influence intestinal motility in ways that alter drug transit time and absorption.

4.1.4 CYP450 Enzyme Modulation

Tenofovir itself undergoes negligible CYP450 metabolism and neither inhibits nor induces CYP enzymes [7]. Therefore, direct CYP-mediated drug interactions with dandelion are of limited direct relevance to tenofovir clearance. However, dandelion root constituents including luteolin, caffeic

acid, and chlorogenic acid have been demonstrated to inhibit CYP2C9, CYP2C19, CYP2D6, and CYP3A4 in vitro [22]. This could be clinically relevant in patients taking tenofovir-based combination regimens that include CYP-metabolized drugs such as protease inhibitors or NNRTIs, where dandelion-mediated CYP inhibition could alter plasma levels of co-administered antiretrovirals.

4.1.5 Absorption-Level Interactions

Animal pharmacokinetic studies involving *Taraxacum mongolicum* (a close relative of *T. officinale*) co-administered with ciprofloxacin demonstrated a significant reduction in ciprofloxacin absorption, attributed to the high mineral content (particularly magnesium, calcium, and iron) of dandelion chelating the antibiotic and forming insoluble complexes [23]. Although tenofovir does not significantly chelate divalent cations in the same manner as fluoroquinolones, high mineral content in dandelion supplements warrants consideration, particularly for simultaneous administration with TAF, which undergoes P-gp-dependent intestinal absorption. It is recommended that dandelion supplements not be taken within 2 hours of any antiretroviral drug to avoid potential formulation-level interactions.

4.2 Pharmacodynamic Interactions

4.2.1 Potassium and Electrolyte Imbalance

Dandelion root and leaf are notably rich in potassium [12]. The diuretic effect of dandelion combined with its potassium load may produce clinically significant electrolyte alterations. Tenofovir-associated proximal tubulopathy (Fanconi syndrome) is characterized by urinary wasting of phosphate, glucose, amino acids, and potassium [18]. In patients on TDF with subclinical tubular dysfunction, additional potassium from dandelion may partially compensate for urinary potassium loss; however, if renal function deteriorates and potassium excretion is impaired, hyperkalemia could develop, especially in patients also taking potassium-sparing diuretics or ACE inhibitors.

4.2.2 Additive Renal Stress

While dandelion root is generally regarded as nephroprotective in traditional use and some animal studies have demonstrated hepatic and renal antioxidant effects [24], its diuretic action represents a form of renal functional stress. In patients with pre-existing renal impairment or those on TDF long-term (a risk factor for chronic kidney disease), the additional diuretic burden from dandelion

root could compromise residual renal function. TDF-associated nephrotoxicity involves direct mitochondrial toxicity in proximal tubular cells; additional renal stress from diuretic-induced volume depletion or transporter interference may lower the threshold for overt nephrotoxic injury [25].

4.2.3 Hypoglycemic Synergy

Animal studies have demonstrated that dandelion root extracts reduce blood glucose through insulin-secretagogue mechanisms [26]. A clinical case report documented symptomatic hypoglycemia in a diabetic patient concurrently using insulin and dandelion supplements [3]. HIV-infected patients on protease inhibitor-based ART (often combined with tenofovir) are at risk for insulin resistance and glucose dysregulation. In this context, additive hypoglycemic effects of dandelion root with antidiabetic medications co-prescribed alongside tenofovir-based ART could represent a clinically meaningful pharmacodynamic interaction.

4.2.4 Anticoagulant Interactions

Dandelion root contains vitamin K and compounds that may inhibit platelet aggregation and slow blood clotting [5]. In patients receiving tenofovir-based ART who are also anticoagulated with warfarin for thrombotic complications, concurrent dandelion root use could alter warfarin response. Dandelion's vitamin K content might antagonize warfarin, while its antiplatelet properties could synergistically increase bleeding risk. INR monitoring is warranted.

5. SUMMARY OF INTERACTION EVIDENCE

Interaction Type	Mechanism	Potential Clinical Effect	Level of Evidence
Diuretic / Renal Clearance	Dandelion root diuresis alters urine flow and GFR	Altered tenofovir plasma concentration and renal exposure	Human clinical study (diuretic effect); indirect inference for tenofovir [19]
OAT1/OAT3 Transporter Modulation	Dandelion flavonoids inhibit OAT-mediated renal tubular uptake	Reduced tenofovir secretion; increased plasma levels	In vitro studies; mechanistic extrapolation [16,20]
MRP2/MRP4 Efflux Inhibition	Phytochemicals impair apical tubular efflux of tenofovir	Intracellular TFV accumulation; enhanced nephrotoxicity	Animal model + in vitro data [16,17,18]
P-Glycoprotein Inhibition	Dandelion flavonoids inhibit intestinal P-gp	Increased TAF absorption; elevated plasma TAF levels	In vitro data; clinical significance uncertain [14,21]
CYP450 Inhibition (indirect)	Luteolin inhibits CYP2C9, CYP3A4 in vitro	Elevated levels of CYP-metabolized co-ARVs	In vitro evidence; indirect for tenofovir [22]

Interaction Type	Mechanism	Potential Clinical Effect	Level of Evidence
Mineral Chelation / Absorption	Divalent cations in dandelion reduce oral drug absorption	Reduced absorption of concomitant drugs	Animal PK study (ciprofloxacin) [23]
Electrolyte / Potassium Imbalance	Dandelion high-K content + diuresis + TDF tubulopathy	Hypo- or hyperkalemia; electrolyte disturbance	Pharmacodynamic inference; case reports [3,12,18]
Additive Nephrotoxic Potential	Diuretic-induced volume depletion compounds TDF renal stress	Increased risk of tubular dysfunction / AKI	Mechanistic; case-series data for TDF nephrotoxicity [25,27]
Hypoglycemic Synergy	Dandelion insulin-secretagogue effects + ART glucose changes	Symptomatic hypoglycemia in susceptible patients	Case report; animal data [3,26]
Anticoagulant Interaction	Vitamin K content antagonizes warfarin; antiplatelet effect	Altered INR; bleeding risk in anticoagulated patients	Pharmacodynamic inference [5,12]

6. CLINICAL IMPLICATIONS AND MANAGEMENT RECOMMENDATIONS

6.1 Risk Stratification

The interaction risk between dandelion root and tenofovir is likely to be modest under normal circumstances, given that formal clinical interaction studies are absent. However, specific patient subgroups are at heightened risk and warrant particular vigilance: (i) patients with pre-existing renal impairment or CKD; (ii) elderly patients with reduced renal reserve; (iii) patients on TDF-based regimens (higher plasma tenofovir exposure compared to TAF); (iv) patients co-administered nephrotoxic agents (NSAIDs, aminoglycosides, ritonavir-boosted protease inhibitors); and (v) patients using dandelion root in high doses or as concentrated supplements rather than dietary quantities.

6.2 Monitoring Parameters

Clinicians should implement the following monitoring measures in patients who concurrently use dandelion root supplements with tenofovir-based antiretroviral therapy:

Renal function should be monitored regularly, including serum creatinine, estimated GFR (eGFR), serum phosphate, urine dipstick for glucose and protein, and urine phosphate-to-creatinine ratio. Electrolytes including potassium, sodium, and bicarbonate should be checked at baseline and periodically. In diabetic patients on ART, blood glucose monitoring should be intensified. If

warfarin is co-prescribed, INR should be monitored more frequently upon initiation or discontinuation of dandelion supplements.

6.3 Administration Guidance

If a patient chooses to use dandelion root alongside tenofovir, the following practical measures are recommended: First, dandelion supplements and antiretrovirals should be taken at least 2 hours apart to avoid any absorption-level interactions. Second, patients should maintain adequate hydration to counteract the diuretic effect of dandelion and minimize renal concentration of tenofovir. Third, consistent dosing (rather than episodic high-dose use) is preferable, as variable phytochemical exposure complicates drug interaction prediction. Fourth, disclosure to the prescribing clinician is essential, and all herbal supplement use should be documented in the patient's medical record.

6.4 Contraindications and Precautions

Dandelion root supplements should be avoided or used only under close medical supervision in HIV or HBV patients who: (i) have eGFR below 50 mL/min; (ii) are on TDF with documented proximal tubular dysfunction or Fanconi syndrome; (iii) are co-prescribed multiple nephrotoxic agents; or (iv) have documented hypersensitivity to Asteraceae/Compositae plants, as cross-reactivity can trigger anaphylaxis.

7. LIMITATIONS AND FUTURE RESEARCH DIRECTIONS

This review is limited by the near-complete absence of controlled pharmacokinetic studies examining dandelion-tenofovir interactions in humans. All mechanistic inferences are derived from in vitro studies, animal pharmacokinetic experiments with ciprofloxacin (not tenofovir), and extrapolations from the pharmacological properties of dandelion constituents and tenofovir transport pathways.

Future research should prioritize: (i) formal pharmacokinetic interaction studies in healthy volunteers examining the effect of standardized dandelion root extracts on tenofovir plasma concentrations (AUC, C_{max}, renal clearance); (ii) in vitro OAT1, OAT3, MRP2, MRP4, and P-gp interaction assays using purified dandelion phytochemicals at clinically relevant concentrations; (iii) epidemiological studies examining rates of TDF-associated nephrotoxicity in populations with high dandelion supplement use; and (iv) standardization of dandelion root supplements to ensure

consistent phytochemical composition for interaction studies.

8. CONCLUSION

Dandelion root (*Taraxacum officinale*) and tenofovir represent a clinically important herb-drug pair given their widespread concurrent use in populations living with HIV and HBV. While tenofovir's lack of CYP450 metabolism precludes many classical herb-drug interactions, its unique dependence on renal OAT and MRP transporters for elimination, combined with dandelion root's diuretic, potassium-rich, flavonoid-mediated transporter-modulating properties, creates a mechanistically plausible interaction scenario. The most important areas of concern include: modulation of renal OAT-mediated tenofovir secretion, P-gp-mediated alteration of TAF absorption, electrolyte imbalances amplifying TDF nephrotoxicity risk, and pharmacodynamic synergy with co-administered medications.

Until rigorous pharmacokinetic studies are conducted, clinicians should counsel HIV and HBV patients to disclose herbal supplement use, monitor renal function closely, maintain appropriate timing between dandelion and antiretroviral administration, and exercise heightened vigilance in at-risk patient subgroups. The growing intersection of traditional medicine and modern antiretroviral therapy underscores the urgent need for systematic pharmacokinetic evaluation of widely used herbal supplements in this population.

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