WORLD JOURNAL OF PHARMACEUTICAL RESEARCH
Coden USA: WJPRAP Impact Factor 8.63
Volume 15, Issue 22, 263-275. Review Article ISSN 2277–7105

ELINZANETANT IN MANAGING MENOPAUSAL SYMPTOMS: PHARMACODYNAMICS, SAFETY AND ADVANTAGES OVER HORMONE THERAPY

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Article Received on 17 Oct. 2026,
Article Revised on 07 Nov. 2026,
Article Published on 16 Nov. 2026

https://doi.org/10.5281/zenodo.24272368

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How to cite this Article: Yazhisai M.[1]*, Sakthi Poorani K.[1], Rekha S.[2]. (2026). Elinzanetant In Managing Menopausal Symptoms: Pharmacodynamics, Safety And Advantages Over Hormone Therapy. World Journal of Pharmaceutical Research, 15(22), 263–275.

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ABSTRACT

Menopause is associated with vasomotor symptoms (VMS), including hot flashes and sleep disturbances, affecting up to 80% of women with hot flashes and 60% with sleep diturbance, respectively. For 10–20% of women, symptoms are severe and impair quality of life. While hormone replacement therapy (HRT) is the most common treatment, its use is limited by contraindications such as vaginal bleeding, breast cancer, and endometrial hyperplasia. Becausof these limitations, there is a growing need for effective non-hormonal alternatives. Elinzanetant (NT-814), branded as Lynkuet™, is a non-hormonal dual NK-1 and NK-3 receptor antagonist for menopausal VMS. As the first dual NK-1/NK-3 receptor antagonist, it targets hypothalamic pathways in thermoregulation. Elinzanetant was approved by Health Canada on July 23, 2025, followed by approval from the U.S. FDA later that year. Clinical efficacy was demonstrated in the Phase III OASIS-1 and OASIS-2 trials (n=796), while long-term safety was evaluated across OASIS-1, OASIS-2, and OASIS-3 studies (n=1,420). These trials showed significant reductions in hot flash frequency and severity with favorable tolerability. This review examines its pharmacodynamics, safety, pharmacokinetics, and therapeutic advantages over hormone therapy.

KEYWORDS: Elinzanetant, Menopause, Vasomotor symptoms, Hot flashes,

Thermoregulation, Hormone Replacement Therapy, Nk-1/Nk-3 receptor antagonist, Non-Hormonal Therapy.

INTRODUCTION

Elinzanetant (NT-814), marketed as **Lynkuet™**, is a **novel, non-hormonal, selective dual neurokinin-1 (NK-1)** and **neurokinin-3 (NK-3)** receptor antagonist developed by **Bayer AG** for the treatment of **moderate-to-severe vasomotor symptoms (VMS)** associated with menopause. It is the **first dual NK-1/NK-3** receptor antagonist, offering a novel mechanism-based approach for symptom control in postmenopausal women.[1]

During the **menopausal transition**, up to **80% of women experience vasomotor symptoms** such as **hot flashes (HFs)** and **60% report sleep disturbances**. Between **10% and 20%** of women experience these symptoms as severe and **intolerable**, significantly impacting their quality of life.[2] While **hormone replacement therapy (HRT)** remains the most common treatment, its use is **contraindicated** in women with conditions such as **unexplained vaginal bleeding**, **past or suspected breast cancer**, or **untreated endometrial hyperplasia**. Therefore, there is a growing need for effective **non-hormonal therapeutic alternatives**.[2]

On **July 23, 2025**, **Health Canada** approved Elinzanetant for the treatment of moderate-to severe VMS associated with menopause.[11] Later, the **U.S. Food and Drug Administration (FDA)** granted approval for **Lynkuet™**, marking an important milestone in the availability of non-hormonal therapies for menopausal women.[12]

Efficacy of Lynkuet™ was shown in 12 weeks of two trials (OASIS 1 and 2) with 796 menopausal women. Lond term safety was established in the OASIS 1, OASIS 2, and OASIS 3 trials which included 1,420 women, of whom 627 who received Lynkuet or placebo for up to 52 weeks.[12,15] These studies confirmed that elinzanetant significantly reduced the frequency and the severity of hot flashes and was generally well tolerated during long-term treatment.[1,5,15] Supporting this advancement, **Dr. JoAnn Pinkerton**, Professor and Director of Midlife Health at the University of Virginia Health System and lead investigator of the **OASIS-2 clinical trial**, stated: These studies investigated the safety and efficacy of Elinzanetant for the treatment of moderate to severe hot flashes associated with menopause and demonstrated improvements in the quality of life in menopausal women and women receiving endocrine therapy for breast cancer.[1]

Lynkuet™ should not be used by pregnant women due to risk of pregnancy loss. Other adverse effects include CNS effects, daytime impairment, increased liver enzymes, and seizure risk in women with neurological disorders.[12,15]

In this review article, a comprehensive discussion is presented on **Elinzanetant (Lynkuet™)**, focusing on its **drug profile**, **mechanism of action**, **pharmacokinetic and pharmacodynamic properties**, **drug interactions**, and its **advantages over conventional hormone replacement therapy (HRT)**. Clinical trial evidence from the OASIS studies is summarized to assess its efficacy and safety.[1,5,6]

CURRENT CLINICAL STATUS OF ELINZANETANT

Elinzanetant is a non-hormonal neurokinin receptor antagonist developed for the management of menopausal vasomotor symptoms (VMS).[4]

- **Regulatory Authority:** U.S. Food and Drug Administration (FDA)[12]
- **Approval Status:** FDA approved.[12]
- **Date of Approval:** October 24, 2025.[12]
- **Brand Name:** Lynkuet.[11,12]
- **Generic Name:** Elinzanetant[12]
- **Dosage Form:** Oral capsules[11,12]
- **Indication:** Treatment of moderate to severe vasomotor symptoms (hot flashes) associated with menopause.[11,12]

CHEMICAL STRUCTURE

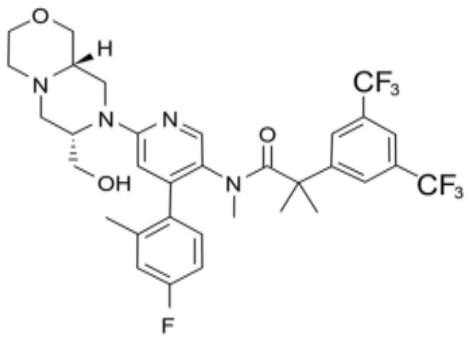


Figure 1: Chemical structure of Elinzanetant (NT-814).[4]

IUPAC name: *N-[6-[(7S,9aS)-7-(hydroxymethyl)-3,4,6,7,9,9a-hexahydro-1H-pyrazino[2,1-c][1,4]oxazin-8-yl]-3-(trifluoromethyl)phenyl]-N,2-dimethylpropanamide.*[6]

Elinzanetant was initially developed by GlaxoSmithKline as GSK1144814 and was later acquired by Bayer, where it was further developed under the code NT-814.[4,6] The chemical structure of Elinzanetant is illustrated in Figure 1. A key pharmacological characteristic of Elinzanetant is its dual antagonistic activity at neurokinin-1 (NK-1) and neurokinin-3 (NK-3) receptors.[4,8] Antagonism of both NK-3 and NK-1 receptors is considered crucial for the clinical efficacy of Elinzanetant in the management of menopausal vasomotor symptoms.[4,8] By simultaneously targeting NK-1 and NK-3, Elinzanetant may provide additional therapeutic benefits for postmenopausal women experiencing climacteric syndrome, including hot flashes and related symptoms.[1,4,6]

PHARMACOKINETICS OF ELINZANETANT

Elinzanetant (BAY 3427080; formerly NT-814 and GSK1144814) is a potent and selective dual neurokinin-1 (NK-1) and neurokinin-3 (NK-3) receptor antagonist developed for the treatment of women with moderate to severe vasomotor symptoms (VMS) associated with menopause.[10]

Soft-gel capsule formulations of elinzanetant demonstrate a dose-proportional increase in maximum plasma concentration (Cmax) over the dose range of 25–600 mg.[16] Although elinzanetant is recommended for administration at night before bedtime, the potential for residual central nervous system (CNS) effects the following morning has been identified as a potential safety consideration.[16]

The area under the plasma concentration–time curve (AUC) increases dose-proportionally between 25 and 120 mg and shows greater-than-dose-proportional increases at higher doses (160–600 mg).[16] To better characterize exposure–response relationships, a population pharmacokinetic–pharmacodynamic (popPK/PD) model was developed for elinzanetant and its three major metabolites (M27, M30/34, and M18/21). This model was used to estimate plasma exposure and NK-1/NK-3 receptor occupancy in women with VMS following repeated dosing.[9]

ABSORPTION

Elinzanetant is **rapidly absorbed** after oral administration, reaching **maximum plasma concentration (Tmax) approximately 1.5 hours** post-dose.[11,16]

The **absolute oral bioavailability** of elinzanetant is approximately **52%**.[11]

Administration with a **high-fat meal reduces the absorption rate** of elinzanetant, although the drug may be administered **with or without food**.[11]

DISTRIBUTION

Elinzanetant exhibits extensive plasma protein binding (>99%), with binding influenced by circadian fluctuations.[11,16]

The blood-to-plasma concentration ratio ranges from 0.6 to 0.7.[11]

Following intravenous administration at steady state, the mean volume of distribution (Vss) is approximately 137 L, indicating extensive extravascular tissue distribution.[11]

The estimated central distribution volume is 23.7 L, while the peripheral distribution volume is 168 L.[11]

METABOLISM

Elinzanetant is primarily metabolized by the cytochrome P450 enzyme CYP3A4.[10,11] A minor metabolic pathway involves glucuronidation, and a small proportion of elinzanetant detected in feces may result from biliary excretion of glucuronide conjugates, which are subsequently hydrolyzed by gut microflora.[10]

The absorption and elimination profiles of metabolites M27, M30/34, and M18/21 are similar to those of the parent compound, whereas metabolite M22 was not quantifiable.[10] All three major metabolites exhibit pharmacological activity comparable to elinzanetant.[10]

ELIMINATION

The geometric mean terminal half-life of elinzanetant ranges between 31 and 41 hours, while the overall elimination half-life is approximately 35 hours.[11,17]

Steady-state concentrations are achieved within 5–7 days following once-daily dosing.[11,17]

Systemic clearance is estimated at 7.26 L/h.[12] After repeated once-daily administration, median receptor occupancy exceeds 99% for NK-1 receptors and 95% for NK-3 receptors during both daytime and nighttime periods, supporting sustained pharmacodynamic activity.[9]

MECHANISM OF ACTION OF ELINZANETANT

Neurokinins are a class of peptide neurotransmitters that include neurokinin A, substance P (SP), and neurokinin B (NKB), and they are widely distributed throughout the central nervous system. The biological actions of neurokinins are mediated through three receptor

subtypes: neurokinin-1 (NK-1), neurokinin-2 (NK-2), and neurokinin-3 (NK-3) receptors.[8]

Specifically, NK-1 receptors primarily interact with substance P, a neuropeptide involved in various physiological functions, including pain perception, inflammation and sleep response.[8] In contrast, NK-3 receptors play a central role in hypothalamic thermoregulatory pathways involved in menopausal vasomotor symptoms. NK-3 receptors play a key role in the body's thermoregulatory center located in the hypothalamus.[4,8] During menopause, fluctuations and eventual decline in estrogen levels disrupt normal thermoregulation, leading to episodes of hot flashes and night sweats.[2,4]

Hypothalamic kisspeptin/neurokinin B/dynorphin (KNDy) neurons express multiple receptor–ligand pairs, including NK-1 and NK-3 receptors and their respective ligands, substance P (SP) and neurokinin B (NKB).[4,8]

During and after the menopausal transition, declining estrogen levels lead to hypertrophy and hyperactivity of KNDy neurons, accompanied by overexpression of neurotransmitters such as NKB and SP.[4,8] This hyperactivation has been shown to coincide with thermoregulatory dysfunction, resulting in vasomotor symptoms (VMS).[4]

Additionally, Substance P acting through NK-1 receptors may contribute to peripheral vasodilation and may also play a role in pathophysiology primary insomnia and sleep disturbances commonly experienced during menopause.[8]

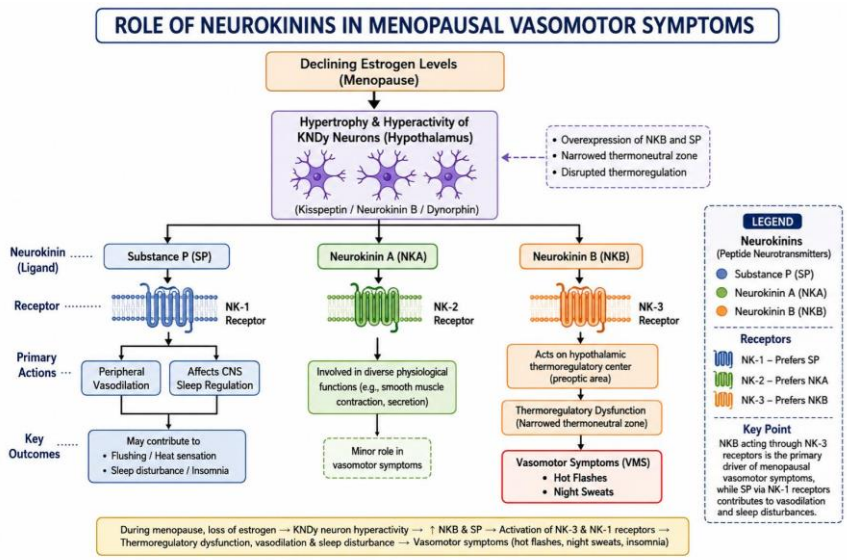


Figure 2: Proposed mechanism of menopausal vasomotor symtoms and the therapeutic action of elinzanetant through dual Nk-1 and Nk-3 receptor antagonism.[4,6,8]

DRUG INTERACTIONS

Elinzanetant is primarily metabolized by cytochrome P450 (CYP) 3A4. Therefore, concomitant administration with CYP3A4 inhibitors or inducers may significantly alter its systemic exposure. Strong CYP3A4 inhibitors increase elinzanetant plasma concentration and should be avoided, whereas strong CYP3A4 inducers reduce drug exposure and may decrease therapeutic efficacy. In addition, elinzanetant acts as a weak CYP3A4 inhibitor and may increase the exposure of sensitive CYP3A4 substrates.[11,12]

Table 1: Clinical Relevant Drug Interactions of Elinzanetant.[11,12]

Interacting drug /class	Example	Effect on Elinzanetant	Clinical Recommendation
Strong CYP3A4 inhibitors	Itraconazole	Increased AUC 4.6-6.3 fold and Cmax 3.3-fold	Contraindicated due to increased exposure and adverse effect risk
Moderate CYP3A4 inhibitors	Erythromycin	Increased AUC~3-fold and Cmax~2-fold	Use with caution; monitor for adverse effects
Weak CYP3A4 inhibitors	Cimetidine	Increased AUC 1.5-fold and Cmax 1.3-fold	Usually no dose adjustment; monitor if necessary
Strong CYP3A4 inducers	Carbamazepine	Decreased AUC 64% and Cmax 44%	Avoid concomitant use due to reduced efficacy
Sensitive CYP3A4 substrates	Midazolam	Increased AUC 1.8-fold and Cmax 1.5-fold	Monitor for toxicity; caution with narrow therapeutic index drugs.

CYP3A4 plays a major role in the metabolism of elinzanetant, clinicians should carefully review concomitant medications before initiating therapy and monitor patients for clinically significant drug interaction.[11,12]

ADVERSE DRUG REACTIONS

|The incidence of adverse drug reactions observed in clinical trials may vary depending on study design, patient population, and duration of exposure. The safety of LYNKUET (elinzanetant) was evaluated across three randomized, double-blind, placebo-controlled, multicenter phase III clinical trials (OASIS-1, OASIS-2, and OASIS-3) involving a total of 1,420 postmenopausal women.[12,15]

The primary efficacy endpoints of these trials included the mean change in the frequency and severity of moderate to severe vasomotor symptoms (VMS).[1,6]

SHORT-TERM SAFETY (OASIS-1 AND OASIS-2)

In the combined OASIS-1 and OASIS-2 trials, 793 women received either LYNKUET or placebo for 12 weeks.[1] Following this initial period, 341 women who were randomized to LYNKUET continued treatment for an additional 14 weeks.[12]

During the first 12 weeks of treatment, elinzanetant demonstrated a safety profile comparable to placebo, with no clinically meaningful differences in overall adverse event rates.[15]

LONG-TERM SAFETY (OASIS-3)

The OASIS-3 trial evaluated the long-term safety of elinzanetant over up to 52 weeks in 627 women.[15] Based on pooled 52-week safety data from all patients who received elinzanetant, the independent liver safety monitoring board concluded that there was no signal of hepatotoxicity. This finding was consistent across OASIS-1, OASIS-2, and OASIS-3 trials.[15]

TREATMENT-EMERGENT ADVERSE EVENTS

Across the clinical development program, treatment-emergent adverse events (TEAEs) were generally mild to moderate in severity.[12,15] The most frequently reported adverse events were headache and fatigue, dizziness, somnolence, abdominal pain, diarrhea, nausea and gastroesophageal reflux disease.[12]

In the combined OASIS-1 and OASIS-2 trials during the first 12 weeks, adverse reactions reported in >2% of patients and at a higher incidence than placebo in the LYNKUET group included headache, fatigue, gastroesophageal reflux disease, dizziness, nausea, and somnolence.[12]

Table 2: Common Adverse Reactions Reported in >2% of patients and at a Higher Frequency than Placebo (Weeks 1–52, OASIS-3).[12]

Adverse Reaction	LYNKUET n (%)	Placebo n (%)
Headache	30 (9.6)	22 (7.0)
Fatigue	23 (7.3)	9 (2.9)
Dizziness	19 (6.1)	6 (1.9)
Somnolence	16 (5.1)	4 (1.3)
Abdominal pain	14 (4.5)	8 (2.5)
Diarrhea	12 (3.8)	3 (1.0)

Photosensitivity

In the OASIS trials, photosensitivity reactions of mild to moderate intensity were reported in

0.5% of patients receiving LYNKUET and 0.1% of patients receiving placebo.[13] The onset of photosensitivity ranged from day 1 to day 290.[13] Discontinuation due to photosensitivity occurred in one patient, while reactions in other patients resolved despite continued treatment with LYNKUET.[12]

The favorable safety and tolerability profile of elinzanetant has also been observed in women receiving endocrine therapy for breast cancer.[14]

Comparison of Menopausal Hormone Therapy and Non-Hormonal Therapy in the Management of Menopause:

Table 3: Comparative overview of menopausal hormone therapy and non-hormone therapy in the management of menopause.

Parameter	Menopausal Hormone Therapy (MHT)	Non-Hormonal Therapy
Definition	Administration of exogenous estrogen alone (in women without a uterus) or in combination with a progestogen (in women with an intact uterus) to relieve menopausal symptoms.[19,20]	Use of pharmacologic and non-pharmacologic interventions that do not involve systemic hormonal replacement.[20,22]
Primary Indications	Moderate to severe vasomotor symptoms (VMS), prevention of postmenopausal bone loss, premature ovarian insufficiency.[19,20]	Mild to moderate VMS, or in women with contraindications to MHT or those who decline hormone therapy.[18,20]
Efficacy on Vasomotor Symptoms	Most effective therapy; reduces hot flash frequency and severity by approximately 75–90%.[19,20]	Moderate efficacy; typically reduces VMS frequency by 30–60% depending on agent.[18,20]
Onset of Action	Rapid onset; clinically significant improvement often within 2–4 weeks.[17,20]	Variable onset; improvement generally observed within 2–8 weeks depending on pharmacologic class.[22]
Common Pharmacologic Options	Oral or transdermal estrogen therapy; combined estrogen-progestogen therapy; levonorgestrel intrauterine system (LNG-IUS) with systemic estrogen; low-dose combined oral contraceptives (perimenopause).[19]	SSRIs/SNRIs (e.g., paroxetine, venlafaxine), gabapentinoids, clonidine, neurokinin receptor antagonists (e.g., elinzanetant), oxybutynin; lifestyle modification.[18,22]
Effect on Sleep and Mood	Improves sleep disturbances secondary to nocturnal VMS; may stabilize mood fluctuations during perimenopause.[20]	Certain agents (SSRIs/SNRIs, gabapentin) may improve sleep and mood; effects are variable and not universal.[7,22]

Effect on Bone Health	Prevents bone loss and significantly reduces risk of osteoporotic fractures.[19,20]	No clinically meaningful improvement in bone mineral density.[18,22]
Effect on Genitourinary Syndrome of Menopause (GSM)	Systemic and local estrogen effectively improve vaginal dryness, dyspareunia, and vulvovaginal atrophy.[19,20]	Limited benefit unless specific local non-hormonal or vaginal therapies are used (e.g., moisturizers, lubricants).[20]
Cardiovascular Risk	Small increased risk of stroke and venous thromboembolism (VTE), particularly with oral formulations and advancing age; risk influenced by timing of initiation ("timing hypothesis").[19,21]	No demonstrated increase in thromboembolic or stroke risk with currently approved agents.[18,22]
Breast Cancer Risk	Combined estrogen-progestin therapy associated with increased breast cancer risk with long-term use; estrogen-alone therapy shows lower or neutral risk in selected populations.[19,22]	No hormone-related breast cancer risk identified.[18,22]
Venous Thromboembolism (VTE)	Increased risk, particularly during first 1–2 years; transdermal estrogen associated with lower risk compared to oral formulations.[19,20]	No increased VTE risk.[18,22]
Use in High-Risk Patients	Contraindicated in women with active or prior breast cancer, estrogen-dependent malignancy, unexplained vaginal bleeding, active thromboembolic disease, or severe liver dysfunction.[19,20]	Preferred in women with contraindications to MHT.[18,22]
Duration of Therapy	Recommended at the lowest effective dose for the shortest duration necessary, with periodic risk–benefit reassessment.[19,20]	May be continued long term based on tolerability and clinical response.[18]
Non-Pharmacologic Interventions	Not primary modality.[22]	Lifestyle modification, weight management, exercise, cognitive behavioral therapy, yoga, meditation, and stress reduction techniques.[20,22]
Overall Role in Therapy	Gold standard for moderate to severe menopausal symptoms in appropriately selected women.[19,20]	Alternative or adjunctive treatment, particularly in high-risk populations or patient preference against hormones.[17,18,22] Serve as an effective alternative in women contraindicated to hormonal therapy, women with history of hormone-sensitive cancer or those who prefer to avoid hormonal therapy. Emerging agent such as elinzanetant provide

		targeted symptom relief with a favorable safety profile and may improve both vasomotor symptoms and sleep disturbance.[6,14,18,22]

CONCLUSION

Menopausal vasomotor symptoms(VMS) remain one of the most common and distressing manifestations of menopause, significantly affecting the quality of life, sleep and overall well-being of postmenopausal women. Although menopausal hormone therapy(MHT) remains the most effective treatment for VMS, it's use is limited in women with contraindications or those who prefer non-hormonal treatment options. Elinzanetant, the first dual neurokinin-1 (NK-1) and neurokinin-3 (NK-3) receptor antagonist, represents a significant advancement in menopause management by targeting the underlying neurobiological mechanisms responsible for vasomotor symptoms. Clinical evidence from the Phase III OASIS trials has demonstrated that elinzanetant significantly reduces the frequency and severity of hot flashes, improves sleep disturbances, and is generally well tolerated, making it a valuable non-hormonal therapeutic option for women with moderate-to-severe menopausal symptoms.

Despite these encouraging findings, the available evidence is largely derived from controlled clinical trials, and long-term real-world data remain limited. Additional studies are needed to evaluate long-term safety, effectiveness in diverse patient populations, comparative efficacy with other non-hormonal therapies, and cost effectiveness in routine clinical practice. Future research should also explore it's role in women with multiple comorbidities, breast cancer survivors receiving endocrine therapy, and other special populations. As further clinical and real-world evidence becomes available, elinzanetant has the potential to become an important component of individualized, evidence-based, non-hormonal management of menopausal vasomotor symptoms.

In conclusion, elinzanetant represents an important therapeutic innovation in menopause management and may play a key role in addressing the unmet needs of women seeking effective, evidence-based, non-hormonal treatment options for vasomotor symptoms.

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