



National Comprehensive Cancer Network® (NCCN®) differentiates ribociclib (KISQALI®) as the only Category 1 Preferred 1L treatment option in combination with an AI for appropriate patients with HR+/HER2- mBC.¹

There is controversy on the choice of CDK4/6i as there are no head-to-head comparisons between the agents and there are some differences in the study populations in the phase III randomized studies. NCCN makes no warranties of any kind whatsoever regarding their content, use, or application and disclaims any responsibility for their application or use in any way.

Systemic therapy for ER- and/or PR-positive recurrent unresectable (local or regional) or stage IV (M1) disease^a

HER2-Negative and Postmenopausal or Premenopausal Receiving Ovarian Ablation or Suppression	
See BINV-P 1 of 3 for general considerations for therapy selection for HR-positive, HER2-negative disease.	
FIRST-LINE THERAPY	SECOND- AND/OR SUBSEQUENT-LINE THERAPY
<p>Preferred Regimens</p> <ul style="list-style-type: none"> • Aromatase inhibitor + CDK4/6 inhibitor^b <ul style="list-style-type: none"> – Aromatase inhibitor + ribociclib (category 1)^c – Aromatase inhibitor + abemaciclib – Aromatase inhibitor + palbociclib <p>If disease progression on adjuvant endocrine therapy or relapse within 12 months of adjuvant endocrine therapy completion consider:</p> <ul style="list-style-type: none"> • Fulvestrant^d + CDK4/6 inhibitor^b <ul style="list-style-type: none"> – Fulvestrant + ribociclib (category 1)^e – Fulvestrant + abemaciclib (category 1)^e – Fulvestrant + palbociclib 	<p>Preferred Regimens</p> <ul style="list-style-type: none"> • Fulvestrant + CDK4/6 inhibitor (abemaciclib, palbociclib, or ribociclib) if CDK4/6 inhibitor not previously used (category 1)^{f,g} • For HER2-negative tumors with <i>PIK3CA</i> or <i>AKT1</i> activating mutations or PTEN alterations, see BINV-Q (6)^h • Everolimus + endocrine therapy (exemestane, fulvestrant, tamoxifen)^{i,j} • Targeted therapy, see BINV-Q (6) and BINV-Q (7), and emerging biomarker options, see BINV-Q (8)
<p>Useful in Certain Circumstances</p> <ul style="list-style-type: none"> • For HER2-negative tumors with <i>PIK3CA</i> activating mutations and disease progression on adjuvant endocrine therapy or relapse within 12 months of adjuvant endocrine therapy completion, see BINV-Q (6) 	<p>Useful in Certain Circumstances</p> <ul style="list-style-type: none"> • Megestrol acetate • Estradiol • Abemaciclib^l • Targeted therapy, see BINV-Q (6) and BINV-Q (7), and emerging biomarker options, see BINV-Q (8)
<p>Other Recommended Regimens for first and/or subsequent lines of therapy</p> <ul style="list-style-type: none"> • For HER2-negative disease and <i>ESR1</i> mutated tumors and after progression on one or two prior lines of endocrine therapy, including one line containing a CDK4/6 inhibitor, see BINV-Q (6) <ul style="list-style-type: none"> • Fulvestrant • Anastrozole • Letrozole • Fulvestrant + aromatase inhibitor (anastrozole, letrozole) (category 1)^k <ul style="list-style-type: none"> • Tamoxifen • Exemestane 	

^aBaseline assessment of bone density recommended for patients receiving an aromatase inhibitor who are at risk of osteoporosis (eg, age >65, family history, chronic steroids).
^bThere is controversy on the choice of CDK4/6 inhibitor as there are no head to head comparisons between the agents and there are some differences in the study populations in the phase 3 randomized studies.
^cIn phase 3 randomized controlled trials, ribociclib + endocrine therapy have shown OS benefit in the first-line setting.
^dConsider for disease progression on adjuvant endocrine therapy or with early disease relapse within 12 months of adjuvant endocrine therapy completion.
^eIn phase 3 randomized controlled trials, fulvestrant + ribociclib or abemaciclib has shown OS benefit in the first-line setting.
^fIn phase 3 randomized controlled trials, fulvestrant in combination with a CDK4/6 inhibitor (abemaciclib, palbociclib, and ribociclib) has shown OS benefit in the second-line setting.
^gIf there is disease progression while on palbociclib, there are limited phase II data to support the use of ribociclib in the second line setting.
^hIf there is progression while on a PI3K inhibitor, there are limited data to support another line of therapy with a PI3K-pathway inhibitor-containing regimen.
ⁱIf there is disease progression while on an everolimus-containing regimen, there are no data to support an additional line of therapy with another everolimus regimen.
^jA combination of exemestane with everolimus can be considered for patients who meet the eligibility criteria for BOLERO-2 (progressed within 12 mo or on nonsteroidal aromatase inhibitor).
^kA single study (S0226) in patients with HR-positive breast cancer and no prior chemotherapy, biological therapy, or endocrine therapy for metastatic disease demonstrated that the addition of fulvestrant to anastrozole resulted in prolongation of time to progression and OS. Subset analysis suggested that patients without prior adjuvant tamoxifen and more than 10 years since diagnosis experienced the greatest benefit. Two studies with similar design (FACT and SOFEA) demonstrated no advantage in time to progression with the addition of fulvestrant to anastrozole.
^lIndicated after progression on prior endocrine therapy and prior chemotherapy in the metastatic setting.
Note: All recommendations are category 2A unless otherwise indicated.

Note: Ribociclib (KISQALI) is not indicated for concomitant use with tamoxifen.

Indications

KISQALI is indicated:

- in combination with an aromatase inhibitor for the adjuvant treatment of adults with hormone receptor (HR)-positive, human epidermal growth factor receptor 2 (HER2)-negative stage II and III early breast cancer (eBC) at high risk of recurrence
- for the treatment of adults with HR-positive, HER2-negative advanced or metastatic breast cancer (mBC) in combination with:
 - an aromatase inhibitor as initial endocrine-based therapy; or
 - fulvestrant as initial endocrine-based therapy or following disease progression on endocrine therapy

IMPORTANT SAFETY INFORMATION

Interstitial lung disease/pneumonitis. Severe, life-threatening, or fatal interstitial lung disease (ILD) and/or pneumonitis can occur in patients treated with KISQALI and other CDK4/6 inhibitors. In patients with eBC (NATALEE) who received 400 mg KISQALI plus a nonsteroidal aromatase inhibitor (NSAI), 1.5% of patients had ILD/pneumonitis (grade 1/2). In patients with advanced or mBC (MONALEESA-2, MONALEESA-3, MONALEESA-7), 1.6% of patients had ILD/pneumonitis of any grade, 0.4% had grade 3/4, and 0.1% had a fatal outcome. Additional cases of ILD/pneumonitis have occurred in the postmarketing setting, some resulting in death.



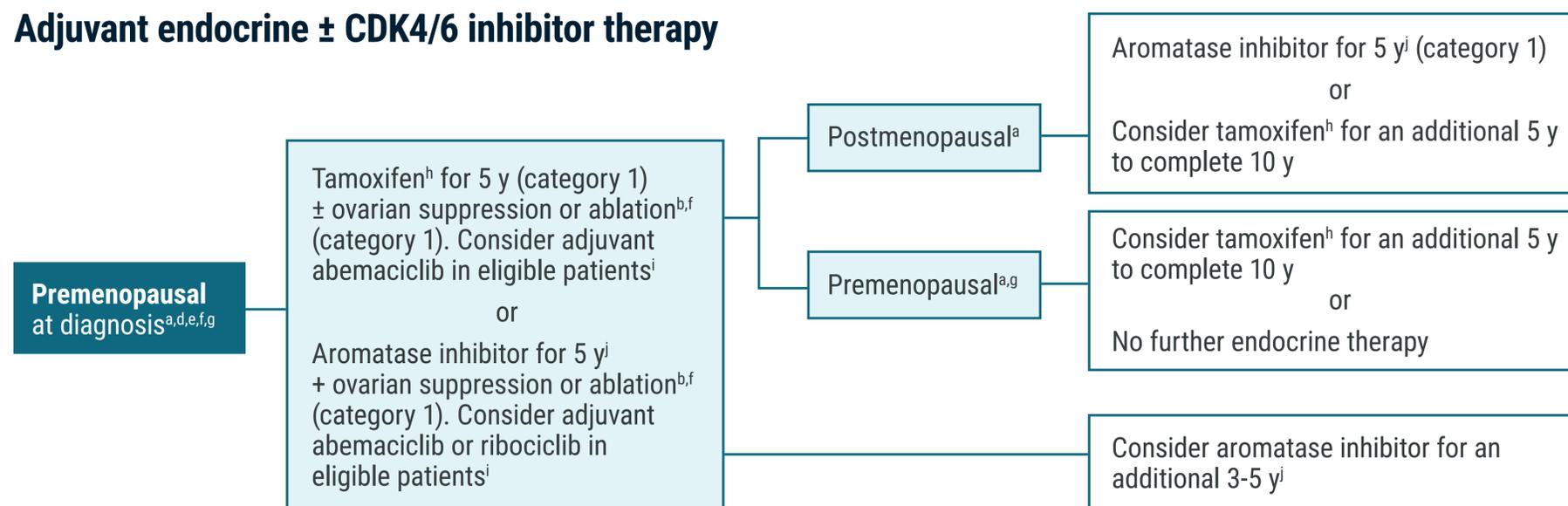
NCCN recognizes ribociclib (KISQALI®) as a **Category 1 Preferred CDK4/6 inhibitor** in combination with an AI for appropriate patients with HR+/HER2- eBC—the only one to receive this designation for both high-risk node-negative and any node-positive disease.¹

KISQALI is approved for use in combination with an AI; node-positive disease excludes patients with microscopic nodal involvement.²

High-risk node-negative disease is defined as either tumor size >5 cm, or if tumor size 2-5 cm, either grade 2 (with high genomic risk or Ki-67 ≥20%), or grade 3.^{1,2}

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Adjuvant endocrine ± CDK4/6 inhibitor therapy



Premenopausal at diagnosis^{a,d,e,f,g}

Tamoxifen^h for 5 y (category 1) ± ovarian suppression or ablation^{b,f} (category 1). Consider adjuvant abemaciclib in eligible patientsⁱ

or

Aromatase inhibitor for 5 y^j + ovarian suppression or ablation^{b,f} (category 1). Consider adjuvant abemaciclib or ribociclib in eligible patientsⁱ

Postmenopausal^a

Aromatase inhibitor for 5 y^j (category 1)
or
Consider tamoxifen^h for an additional 5 y to complete 10 y

Premenopausal^{a,g}

Consider tamoxifen^h for an additional 5 y to complete 10 y
or
No further endocrine therapy

Consider aromatase inhibitor for an additional 3-5 y^j

Note: Ribociclib (KISQALI) is not indicated for concomitant use with tamoxifen.

^aDefinition of Menopause (BINV-0).

^bA balanced discussion of the risks and benefits associated with ovarian suppression therapy is critical, including the potential side effects of premature menopause. Aromatase inhibitor or tamoxifen for 5 years plus ovarian suppression should be considered, based on SOFT and TEXT clinical trial outcomes, for premenopausal patients at higher risk of recurrence (ie, young age, high-grade tumor, lymph node involvement).

^dBaseline assessment of bone density recommended for patients receiving an aromatase inhibitor who are at risk of osteoporosis (eg, age >65, family history, chronic steroids).

^eThe use of a bisphosphonate (oral/IV) or denosumab is acceptable to maintain or to improve bone mineral density and reduce risk of fractures in postmenopausal (natural or induced) patients receiving adjuvant aromatase inhibitor therapy.

^fEvidence suggests that the magnitude of benefit from surgical or radiation ovarian ablation in premenopausal patients with HR-positive breast cancer is similar to that achieved with CMF alone.

^gSafety data support administration of GnRH agonists before or with chemotherapy, especially if there is a goal to enhance fertility preservation. They can also be initiated after chemotherapy in patients who remain premenopausal.

^hSome SSRIs like fluoxetine and paroxetine decrease the formation of endoxifen, 4-OH tamoxifen, and active metabolites of tamoxifen, and may impact its efficacy. Caution is advised about coadministration of these drugs with tamoxifen. However, SNRIs (citalopram and venlafaxine) appear to have minimal impact on tamoxifen metabolism. At this time, based on current data the panel recommends against CYP2D6 gene testing for patients being considered for tamoxifen therapy.

ⁱIn patients with HR-positive/HER2-negative breast cancer, 1) for those with high-risk breast cancer (ie, those with ≥4 positive lymph nodes (confirmed preoperatively and/or at surgery), or 1-3 positive lymph nodes with either grade 3 disease or tumor size ≥5 cm (on pre-operative imaging and/or at surgery), 2 years of adjuvant abemaciclib can be considered in combination with endocrine therapy (category 1, preferred). **VTE risk should be considered when combining abemaciclib with tamoxifen.** 2) For those with any lymph node involvement (excluding microscopic nodal involvement), or if no nodal involvement either tumor size >5 cm, or if tumor size 2-5 cm, either grade 2 (and high genomic risk or Ki-67 ≥20%), or grade 3, 3 years of ribociclib with aromatase inhibitor may be considered (Category 1, preferred). Notably adjuvant CDK4/6 inhibitors have been studied in patients at high risk who mostly received adjuvant/neoadjuvant chemotherapy; **there are limited data in those who did not receive chemotherapy.** In patients eligible for adjuvant olaparib (see BINV-L) and abemaciclib or ribociclib, the benefit and optimal sequence is not known.

^jThe three selective aromatase inhibitors (ie, anastrozole, letrozole, exemestane) have shown similar anti-tumor efficacy and toxicity profiles in randomized studies in the adjuvant and preoperative settings. The optimal duration of aromatase inhibitors in adjuvant therapy is uncertain. Patients with lymph node involvement may benefit from extended aromatase inhibitor duration (7.5-10 years total).

Note: All recommendations are category 2A unless otherwise indicated.

IMPORTANT SAFETY INFORMATION (continued)

Interstitial lung disease/pneumonitis (continued). Monitor patients for pulmonary symptoms indicative of ILD/pneumonitis, which may include hypoxia, cough, and dyspnea. In patients who have new or worsening respiratory symptoms suspected to be due to ILD or pneumonitis, interrupt KISQALI immediately and evaluate the patient. Permanently discontinue treatment with KISQALI in patients with severe ILD/pneumonitis or any recurrent symptomatic ILD/pneumonitis.

Severe cutaneous adverse reactions. Severe cutaneous adverse reactions (SCARs), including Stevens-Johnson syndrome (SJS), toxic epidermal necrolysis (TEN), and drug-induced hypersensitivity syndrome (DiHS)/drug reaction with eosinophilia and systemic symptoms (DRESS) can occur in patients treated with KISQALI.

If signs or symptoms of SCARs occur, interrupt KISQALI until the etiology of the reaction has been determined. Early consultation with a dermatologist is recommended to ensure greater diagnostic accuracy and appropriate management.

If SJS, TEN, or DiHS/DRESS is confirmed, permanently discontinue KISQALI. Do not reintroduce KISQALI in patients who have experienced SCARs or other life-threatening cutaneous reactions during KISQALI treatment.

QT interval prolongation. KISQALI has been shown to prolong the QT interval in a concentration-dependent manner.

Avoid KISQALI in patients who are at significant risk of developing torsades de pointes (TdP), including those with:

- congenital long QT syndrome;
- uncontrolled or significant cardiac disease, recent myocardial infarction, heart failure, unstable angina, bradyarrhythmias, uncontrolled hypertension, high degree atrioventricular block, severe aortic stenosis, or uncontrolled hypothyroidism;
- electrolyte abnormalities;
- taking drugs known to prolong QT interval and/or strong CYP3A inhibitors as this may lead to prolongation of the QTcF interval.

Based on the observed QT prolongation during treatment, KISQALI may require dose interruption, reduction, or discontinuation.

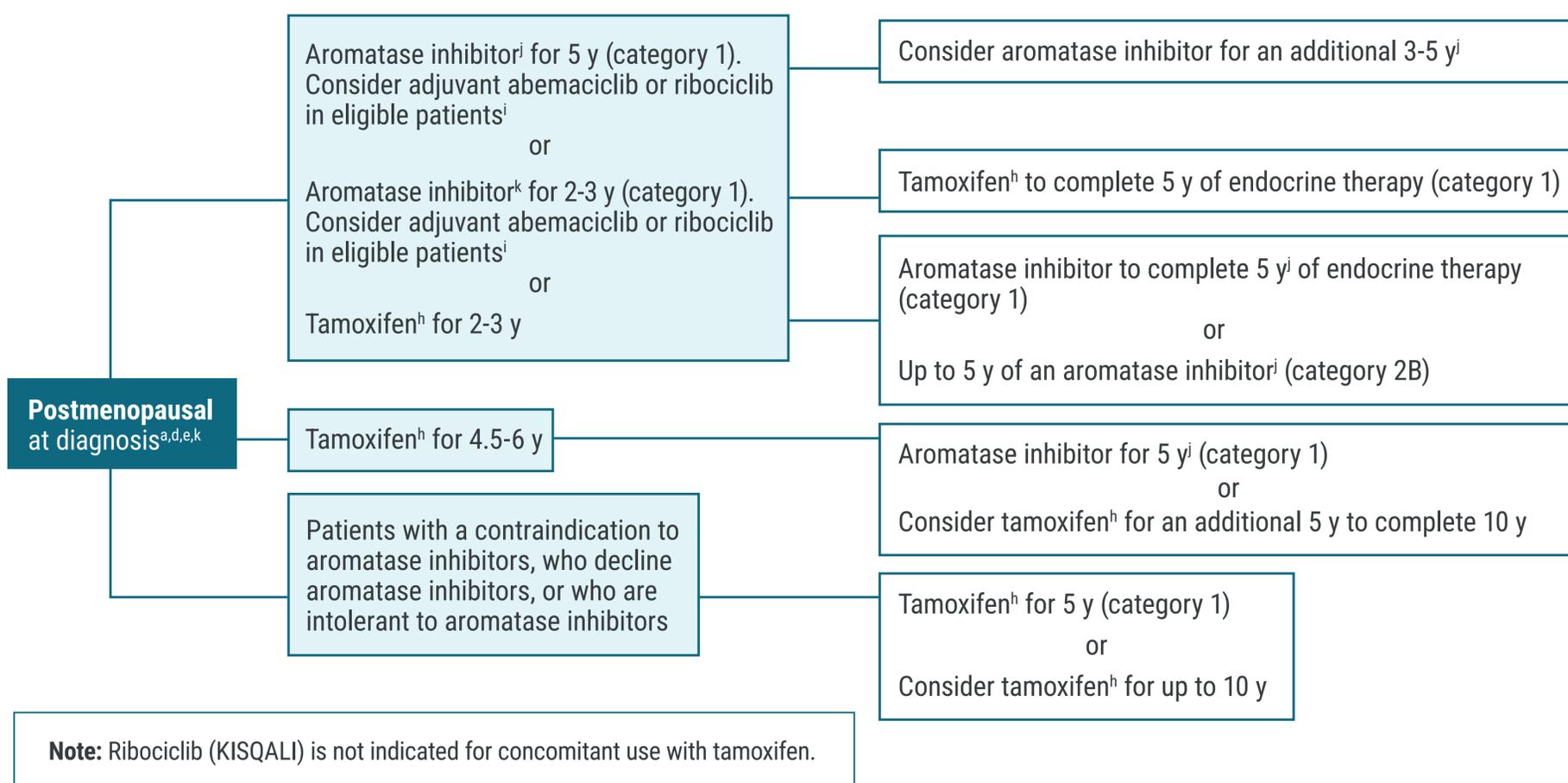
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^fSome SSRIs like fluoxetine and paroxetine decrease the formation of endoxifen, 4-OH tamoxifen, and active metabolites of tamoxifen, and may impact its efficacy. Caution is advised about coadministration of these drugs with tamoxifen. However, SNRIs (citalopram and venlafaxine) appear to have minimal impact on tamoxifen metabolism. At this time, based on current data the panel recommends against CYP2D6 gene testing for patients being considered for tamoxifen therapy.

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^hThe three selective aromatase inhibitors (ie, anastrozole, letrozole, exemestane) have shown similar anti-tumor efficacy and toxicity profiles in randomized studies in the adjuvant and preoperative settings. The optimal duration of aromatase inhibitors in adjuvant therapy is uncertain. Patients with lymph node involvement may benefit from extended aromatase inhibitor duration (7.5-10 years total).

ⁱIf patient is not postmenopausal, sequential evaluation of hormonal status is recommended to consider an alternative endocrine agent.

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IMPORTANT SAFETY INFORMATION (continued)

QT interval prolongation (continued). In patients with eBC (NATALEE) who received 400 mg KISQALI plus NSAID, 8 out of 2494 patients (0.3%) had > 500 ms post-baseline QTcF interval value and 50 out of 2494 patients (2%) had > 60 ms QTcF increase from baseline. QTcF prolongation was reversible with dose interruption. The majority of QTcF prolongation occurred within the first 4 weeks of KISQALI. There were no reported cases of torsades de pointes.

In patients with advanced or mBC (MONALEESA-2, MONALEESA-3, and MONALEESA-7) who received 600 mg KISQALI plus NSAID or fulvestrant, 15 of 1054 patients (1.4%) had >500 ms postbaseline QTcF value, and 61 of 1054 (6%) had a >60 ms QTcF increase from baseline. QTcF prolongation was reversible with dose interruption. The majority of QTcF prolongation occurred within the first 4 weeks of KISQALI. There were no reported cases of torsades de pointes. In MONALEESA-2, in the KISQALI + letrozole treatment arm, there was 1 (0.3%) sudden death in a patient with grade 3 hypokalemia and grade 2 QT prolongation. No cases of sudden death were reported in MONALEESA-7 or MONALEESA-3.

Perform electrocardiogram (ECG) in all patients prior to starting KISQALI. Initiate treatment with KISQALI only in patients with QTcF values <450 ms. Repeat ECG at approximately Day 14 of the first cycle, and as clinically indicated.

Monitor serum electrolytes (including potassium, calcium, phosphorus and magnesium) prior to the initiation of KISQALI, at the beginning of the first 6 cycles, and as clinically indicated. Correct any abnormality before starting KISQALI.

IMPORTANT SAFETY INFORMATION (continued)

Increased QT prolongation with concomitant use of tamoxifen. KISQALI is not indicated for concomitant use with tamoxifen. Avoid use of tamoxifen with KISQALI. In MONALEESA-7, the observed mean QTcF increase from baseline was >10 ms higher in the tamoxifen + placebo subgroup compared with the nonsteroidal aromatase inhibitor (NSAI) + placebo subgroup. In the placebo arm, an increase of >60 ms from baseline occurred in 6/90 (7%) of patients receiving tamoxifen, and in no patients receiving an NSAI. An increase of >60 ms from baseline in the QTcF interval was observed in 14/87 (16%) of patients in the KISQALI and tamoxifen combination and in 18/245 (7%) of patients receiving KISQALI plus an NSAI.

Hepatotoxicity. In patients with eBC and advanced or mBC, drug-induced liver injury and increases in transaminases occurred with KISQALI.

In patients with eBC (NATALEE) treated with KISQALI, drug-induced liver injury was reported in 9 patients (0.4%), of which 5 were grade ≥ 3 and 8 had resolved as of the data cutoff. There were 8 (0.3%) clinically confirmed Hy's Law cases (including 4 out of 9 drug-induced liver injury mentioned above), 6 of which had resolved within 303 days and 2 were resolving, all after discontinuation of KISQALI. Grade 3/4 increases in alanine aminotransferase (ALT) and aspartate aminotransferase (AST) occurred in 8% and 4.7%, respectively, and grade 4 increases in ALT (1.5%) and AST (0.8%).

In patients with advanced or mBC (MONALEESA-2, MONALEESA-7, and MONALEESA-3) treated with KISQALI, grade 3 or 4 increases in ALT and AST occurred in 11% and 8%, respectively. Among the patients who had grade ≥ 3 ALT/AST elevation, the median time to onset was 92 days for the KISQALI plus aromatase inhibitor or fulvestrant treatment arms. The median time to resolution to grade ≤ 2 was 21 days in the KISQALI plus aromatase inhibitor or fulvestrant treatment arms. In MONALEESA-2 and MONALEESA-3, concurrent elevations in ALT or AST >3x ULN and total bilirubin >2x ULN, with normal alkaline phosphatase, in the absence of cholestasis (Hy's Law) occurred in 6 (1%) patients and all patients recovered after discontinuation of KISQALI.

Perform liver function tests (LFTs) before initiating KISQALI. Monitor LFTs every 2 weeks for the first 2 cycles, at the beginning of each of the subsequent 4 cycles, and as clinically indicated. Based on the severity of the transaminase elevations, KISQALI may require dose interruption, reduction, or discontinuation.

Neutropenia. KISQALI causes concentration-dependent neutropenia. In patients with eBC (NATALEE) who received KISQALI plus NSAI, 94%, including 45% of grade 3/4, had a decrease in neutrophil counts (based on laboratory findings), 63% had an adverse drug reaction of neutropenia, and 0.3% had febrile neutropenia. The median time to grade ≥ 2 neutropenia was 18 days. The median time to resolution of grade ≥ 3 neutropenia to grade <3 was 10 days. Treatment discontinuation due to neutropenia was required in 1.1% of patients.

In patients with advanced or metastatic breast cancer (MONALEESA-2, MONALEESA-7, and MONALEESA-3) who received KISQALI plus NSAI or fulvestrant, 75% had neutropenia, 62% had grade 3/4 decrease in neutrophil count (based on laboratory findings), and 1.7% had febrile neutropenia. The median time to grade ≥ 2 neutropenia was 17 days. The median time to resolution of grade ≥ 3 neutropenia to grade <3 was 12 days. Treatment discontinuation due to neutropenia was required in 1% of patients.

Perform complete blood count (CBC) before initiating therapy with KISQALI. Monitor CBC every 2 weeks for the first 2 cycles, at the beginning of each of the subsequent 4 cycles, and as clinically indicated. Based on the severity of the neutropenia, KISQALI may require dose interruption, reduction, or discontinuation.

Embryo-fetal toxicity. Based on findings from animal studies and the mechanism of action, KISQALI can cause fetal harm when administered to a pregnant woman. Advise pregnant women of the potential risk to a fetus. Advise women of reproductive potential to use effective contraception during therapy with KISQALI and for at least 3 weeks after the last dose.

Adverse reactions in early breast cancer patients. Most common (incidence $\geq 20\%$) adverse reactions include infections, nausea, headache, and fatigue.

Laboratory abnormalities. In a clinical trial of patients with early breast cancer, the most common laboratory abnormalities reported in the KISQALI arm (all grades, pooled incidence $\geq 20\%$) were **lymphocytes decreased, leukocyte decreased, neutrophil decreased, hemoglobin decreased, alanine aminotransferase increased, aspartate aminotransferase increased, creatinine increased, and platelets decreased.**

Adverse reactions in advanced or metastatic breast cancer patients. Most common (incidence $\geq 20\%$) adverse reactions include infections, nausea, fatigue, diarrhea, vomiting, headache, constipation, alopecia, cough, rash, and back pain.

Laboratory abnormalities. Across clinical trials of patients with advanced or metastatic breast cancer, the most common laboratory abnormalities reported in the KISQALI arm (all grades, pooled incidence $\geq 20\%$) were **leukocytes decreased, neutrophils decreased, hemoglobin decreased, lymphocytes decreased, AST increased, gamma-glutamyl transferase increased, ALT increased, creatinine increased, platelets decreased, and glucose serum decreased.**

Please see additional Important Safety Information throughout and [click here](#) for full Prescribing Information for KISQALI.

1L, first line; AI, aromatase inhibitor; AKT1, AKT serine/threonine kinase 1; CDK, cyclin-dependent kinase; CMF, cyclophosphamide/methotrexate/fluorouracil; eBC, early breast cancer; ER, estrogen receptor; ESR1, estrogen receptor 1; GnRH, gonadotropin-releasing hormone; IV, intravenous; mBC, metastatic breast cancer; mo, month; OS, overall survival; PI3K, phosphatidylinositol-3 kinase; PIK3CA, phosphatidylinositol-4,5-bisphosphate 3-kinase catalytic subunit alpha; PR, progesterone receptor; PTEN, phosphatase and tensin homolog; SNRI, serotonin and norepinephrine reuptake inhibitor; SSRI, selective serotonin reuptake inhibitor; VTE, venous thromboembolism; y, year.

References: 1. Referenced with permission from the NCCN Clinical Practice Guidelines in Oncology (NCCN Guidelines[®]) for Breast Cancer V.4.2025. © National Comprehensive Cancer Network, Inc. 2025. All rights reserved. Accessed April 17, 2025. To view the most recent and complete version of the guideline, go online to NCCN.org. 2. Kisqali. Prescribing information. Novartis Pharmaceuticals Corp.



Scan to see the [data guiding KISQALI recommendations](#)