Truqap® ▼
capivasertib
160 mg • 200 mg tablets

In combination with fulvestrant for your eligible patients with HR+/HER2– aBC with one or more *PIK3CA/AKT1/PTEN* alterations[1]

For UK Prescribing Information, scan the QR codes on page 8.

Unlocking the potential of targeting the PI3K/AKT pathway in HR+/HER2– aBC

Efficacy and safety profile summary from the CAPItello-291 trial

TRUQAP is indicated in combination with fulvestrant for the treatment of adult patients with hormone receptor (HR) positive human epidermal growth factor receptor 2 (HER2) negative (defined as IHC 0 or 1+, or IHC 2+/ISH–) locally advanced or metastatic breast cancer with one or more *PIK3CA/AKT1/PTEN*-alterations following recurrence or progression on or after an endocrine based regimen.[1]

Adverse Events should be reported. Reporting forms and information can be found at https://yellowcard.mhra.gov.uk or search for MHRA Yellow Card in Google Play or Apple App store. Adverse events should also be reported to AstraZeneca by visiting https://contactazmedical.astrazeneca.com or by calling 0800 783 0033.

aBC, advanced breast cancer; AKT, serine/threonine protein kinase; *AKT1*, serine/threonine protein kinase 1; HER2, human epidermal growth factor receptor 2; HR+, hormone receptor positive; IHC, immunohistochemistry; ISH, *in situ* hybridisation; *PIK3CA*, phosphatidylinositol-4,5-bisphosphate 3-kinase catalytic subunit alpha; PI3K, phosphoinositide 3-kinase; *PTEN*, phosphatase and tensin homologue.

Please refer to the full Summary of Product Characteristics for additional information prior to prescribing.

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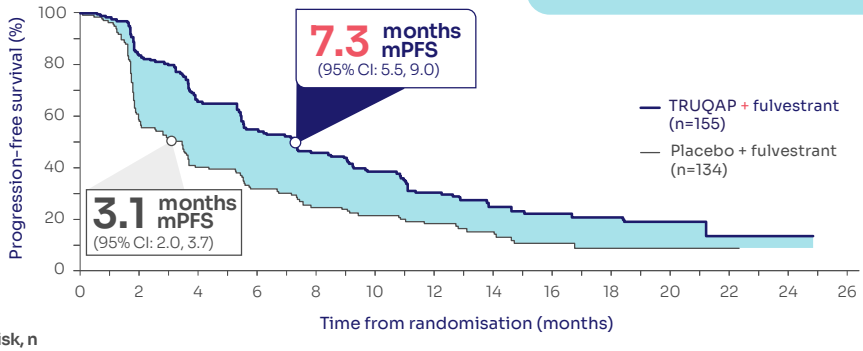
TRUQAP + fulvestrant demonstrated a significant improvement in mPFS* compared with placebo + fulvestrant in the population with *PIK3CA/AKT1/PTEN* alterations[2]

CAPItello-291 is a global, phase III, randomised, double-blind, multicentre trial assessing the efficacy and safety of TRUQAP + fulvestrant compared with placebo + fulvestrant in patients with HR+/HER2– aBC (N=708).[†2]

- Efficacy data presented are from the population of patients with *PIK3CA/AKT1/PTEN*-altered tumours only (n=289)[2]
- Safety data reflect the overall population (N=705)[2]

Primary endpoint: investigator-assessed mPFS† (n=289)[1,2]

HR 0.50 (95% CI: 0.38, 0.65; *P*<0.001)[1]



Patients at risk, n

TRUQAP + fulvestrant	155	127	99	80	65	54	38	26	21	12	3	2	1	0
Placebo + fulvestrant	134	77	48	37	28	24	17	11	6	2	1	1	0	0

Adapted from Turner NC, et al. 2023.[2]

In patients with *PIK3CA/AKT1/PTEN*-altered tumours, TRUQAP + fulvestrant delivered a 50% reduction in risk of disease progression or death vs placebo + fulvestrant, translating to a median extension in PFS of 4.2 months[‡1,2]

Data cut-off: 15 August 2022.[2]

*PFS is defined as the time from the date of randomisation until the date of disease progression, as defined by RECIST v1.1, or death (by any cause in the absence of progression) regardless of whether the patient withdraws from randomised therapy or receives another anticancer therapy prior to progression.[3]

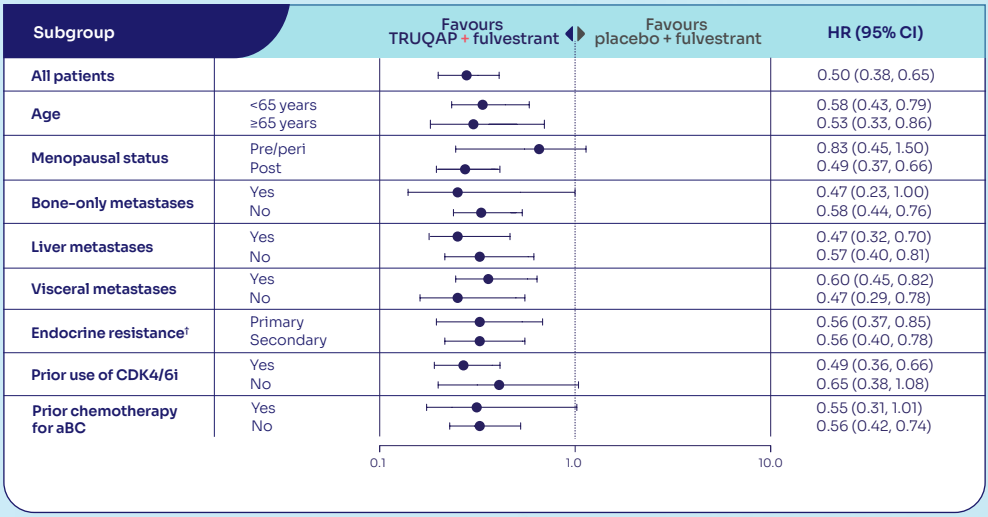
†CAPItello-291 included patients with and without *PIK3CA/AKT1/PTEN*-altered tumours (N=708).[2]

‡Stratified Cox proportional-hazards model. A hazard ratio <1 favours TRUQAP + fulvestrant. In patients with *PIK3CA/AKT1/PTEN*-altered tumours, the log-rank test and Cox model are stratified by the presence of liver metastases (yes vs no) and prior use of CDK4/6i (yes vs no).[2]

aBC, advanced breast cancer; *AKT1*, serine/threonine protein kinase 1; CI, confidence interval; CDK4/6i, cyclin-dependent kinase 4 and 6 inhibitor; HER2–, human epidermal growth factor receptor 2 negative; HR, hazard ratio; HR+, hormone receptor positive; mPFS, median progression-free survival; PFS, progression-free survival; *PIK3CA*, phosphatidylinositol-4,5-bisphosphate 3-kinase catalytic subunit alpha; *PTEN*, phosphatase and tensin homologue; RECIST, Response Evaluation Criteria in Solid Tumours.

In combination with fulvestrant, TRUQAP delivered consistent PFS benefit across subgroups vs placebo + fulvestrant*[2,4]

Investigator-assessed PFS by subgroup in patients with AKT pathway-altered tumours (exploratory analysis)[4]



Adapted from Turner NC, et al. 2023. Supplementary appendix.[4]

In an exploratory analysis of PFS by altered gene within the *PIK3CA/AKT1/PTEN*-altered population, adding TRUQAP to fulvestrant provided a numerical PFS benefit vs placebo + fulvestrant regardless of gene alteration detected (statistical significance not performed)‡[5]

Data cut-off: 15 August 2022.[2]

*Study not powered to show statistical significance across subgroups.[4]

†Per ESO-ESMO guidelines: primary endocrine resistance is considered relapse while on the first 2 years of adjuvant ET, or PD within first 6 months of 1L ET for aBC while on ET. Secondary resistance is considered relapse while on adjuvant ET after the first 2 years, or relapse within 12 months of completing adjuvant ET, or PD ≥6 months after initiating ET for aBC while on ET.[2,6]

‡mPFS in patients with *PIK3CA*, *AKT1* and *PTEN* alterations only was 5.6 months (95% CI: 4.2, 7.4), 9.1 months (95% CI: 2.2, NC) and 9.1 months (95% CI: 5.5, 11.1) with TRUQAP + fulvestrant vs 2.1 months (95% CI: 1.9, 3.6), 3.7 months (95% CI: 1.7, 9.5) and 3.6 months (95% CI: 1.8, 6.7) with placebo + fulvestrant, respectively.[5]

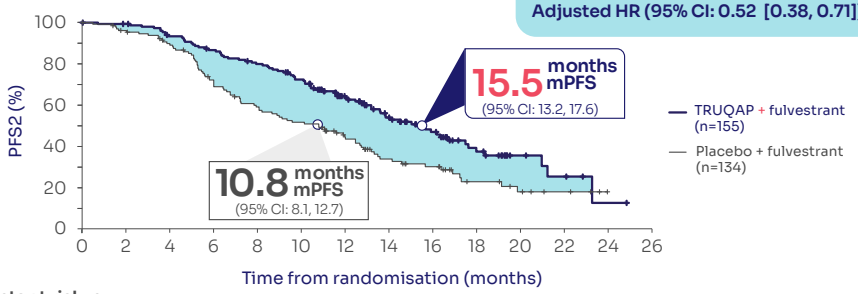
1L, first-line; aBC, advanced breast cancer; AKT, serine/threonine protein kinase; *AKT1*, serine/threonine protein kinase 1; CI, confidence interval; CDK4/6i, cyclin-dependent kinase 4 and 6 inhibitor; ESMO, European Society for Medical Oncology; ESO, European School of Oncology; ET, endocrine therapy; HR, hazard ratio; mPFS, median progression-free survival; NC, not calculable; PD, progressive disease; PFS, progression-free survival; *PIK3CA*, phosphatidylinositol-4,5-bisphosphate 3-kinase catalytic subunit alpha; *PTEN*, phosphatase and tensin homologue.

Secondary endpoint: PFS2*[7]

Extended treatment benefit with TRUQAP + fulvestrant was observed in the *PIK3CA/AKT1/PTEN*-altered population[7]

Extended treatment benefit (PFS2) (n=289)†[7]

Adjusted HR (95% CI: 0.52 [0.38, 0.71])



Patients at risk, n

TRUQAP + fulvestrant	155	151	140	129	118	105	76	53	38	22	8	5	1	0
Placebo + fulvestrant	134	124	113	89	72	63	45	29	22	11	7	4	0	0

Adapted from Rugo H, et al. 2024.[7]

A prespecified analysis of OS in patients with *PIK3CA/AKT1/PTEN* alterations showed an HR of 0.88 (95% CI: 0.65, 1.19)‡[1]

Data cut-off: 15 August 2022.[7]

*PFS2 was defined as the time from randomisation to second progression (i.e. the earliest of either death or a progression event following treatment start after first progression).[7]

†HR was estimated using the Cox proportional hazard model stratified by the presence of liver metastases, prior use of CDK4/6 inhibitor, and geographic region.[7]

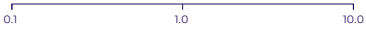
‡Data cut off: 15 April 2024. 59% of patients had died.[1] The key secondary endpoints of OS and ORR will be formally analysed at future data cut-offs.[1]

AKT1, serine/threonine protein kinase 1; CDK4/6, cyclin-dependent kinase 4/6; CI, confidence interval; HR, hazard ratio; mPFS, median progression-free survival; ORR, objective response rate; OS, overall survival; PFS2, time to second progression event or death after a first progression event; *PIK3CA*, phosphatidylinositol-4,5-bisphosphate 3-kinase catalytic subunit alpha; *PTEN*, phosphatase and tensin homologue.

Exploratory ctDNA analyses: TRUQAP + fulvestrant demonstrated numerically consistent PFS benefit vs placebo + fulvestrant post CDK4/6i + AI*[8]

mPFS by ctDNA-defined *PIK3CA/AKT1/PTEN* alteration status (post-1L ET + CDK4/6i[†]) – exploratory analysis

	n	Median PFS (months) TRUQAP + fulvestrant	Placebo + fulvestrant	HR (95% CI)
***PIK3CA/AKT1/PTEN* alteration detected in tissue**	155	7.0	2.6	0.50 (0.35, 0.71)
***PIK3CA/AKT1/PTEN* alteration detected in ctDNA**	161	7.0	2.0	0.41 (0.29, 0.58)
***ESR1*mut co-mutation**	94	7.0	2.0	0.45 (0.29, 0.73)
No *ESR1*mut detected	67	7.4	2.0	0.32 (0.18, 0.57)
***PIK3CA/AKT1/PTEN* alteration detected in either tissue or ctDNA**	196	7.0	2.0	0.44 (0.32, 0.61)



Adapted from Turner NC, et al. 2025.[8]

Study not powered to show statistical significance across subgroups

- Note: NICE recommends TRUQAP + fulvestrant as an option for treating adults with HR+/HER2–[‡] aBC that has one or more *PIK3CA, AKT1* or *PTEN* gene alterations and that has recurred or progressed after a CDK4/6i + AI[9]

In these exploratory ctDNA analyses of baseline blood samples from CAPItello-291, TRUQAP + fulvestrant demonstrated consistent PFS benefit vs placebo + fulvestrant in patients with ctDNA-defined *PIK3CA/AKT1/PTEN* alterations, irrespective of *ESR1*mut status[8]

Data cut-off: 15 August 2022.[8]

*For these exploratory analyses, baseline blood samples were analysed retrospectively. PFS was analysed using Cox regression for subgroups of patients with a baseline tumour alteration (*PIK3CA/AKT1/PTEN*; co-occurring *ESR1*mut) identified by ctDNA.[8]

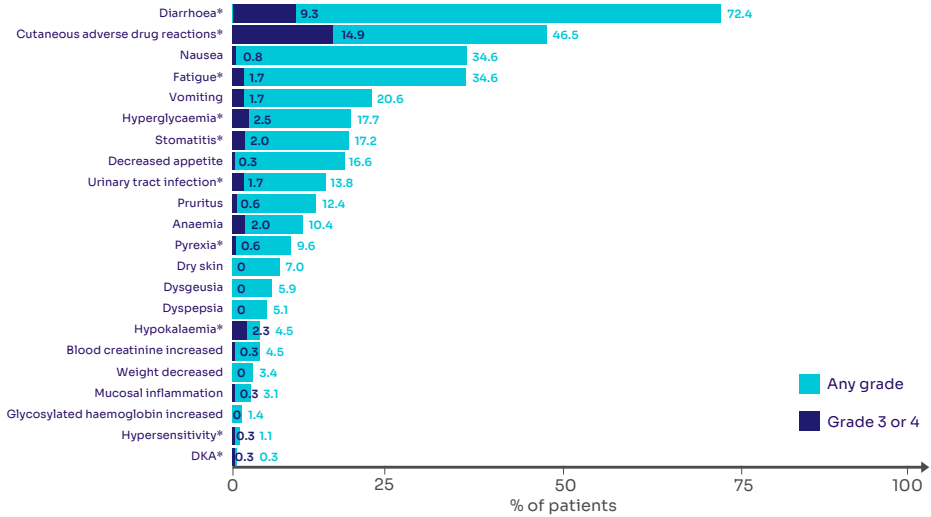
[†]Restricted to patients who received one prior line of AI + CDK4/6i for aBC (except two patients [one in each treatment arm] who received adjuvant AI followed by SERM plus CDK4/6i for aBC) and no prior chemotherapy for aBC.[8]

[‡]HER2-negative (defined as IHC 0 or 1+, or IHC 2+/ISH–).[9]

1L, first-line; aBC, advanced breast cancer; AI, aromatase inhibitor; *AKT1*, serine/threonine protein kinase 1; CDK4/6i, cyclin-dependent kinase 4 and 6 inhibitor; CI, confidence interval; ctDNA, circulating tumour DNA; *ESR1*mut, oestrogen receptor 1 mutation; ET, endocrine therapy; HER2, human epidermal growth factor receptor 2; HR, hazard ratio; HR+, hormone receptor positive; IHC, immunohistochemistry; ISH, *in situ* hybridisation; mPFS, median progression-free survival; NICE, National Institute for Health and Care Excellence; PFS, progression-free survival; *PIK3CA*, phosphatidylinositol-4,5-bisphosphate 3-kinase catalytic subunit alpha; *PTEN*, phosphatase and tensin homologue; SERM, selective oestrogen receptor modulator.

In the overall population of the CAPItello-291 trial, the majority of adverse events were grade 1 or 2[1]

Adverse events observed in the CAPItello-291 study with TRUQAP + fulvestrant (n=355)[1]



Adapted from TRUQAP SmPC.[1]

*The SmPC classifies these adverse events using grouped terms that include multiple underlying events, whereas the primary analysis paper reports them as individual/single terms (excluding rash). Thus, reported values may differ.[1] Please see below for more information on adverse event categorisation.

Diarrhoea
Diarrhoea and frequent bowel movements

Cutaneous adverse drug reactions
Butterfly rash, dermatitis, dermatitis exfoliative generalised, drug eruption, drug reaction with eosinophilia and systemic symptoms (DRESS), erythema, erythema multiforme, papule, rash, rash erythematous, rash follicular, rash macular, rash maculo-papular, rash papular, rash pruritic, skin reaction and toxic skin eruption

Fatigue
Asthenia, fatigue and malaise

Hyperglycaemia
Hyperglycaemia, blood glucose increased, diabetes mellitus, type 2 diabetes mellitus and diabetic metabolic decompensation

Stomatitis
Stomatitis, aphthous ulcer and mouth ulceration

Urinary tract infection
Urinary tract infection, pyuria and cystitis

Pyrexia
Body temperature increased and pyrexia

Hypokalaemia
Blood potassium decreased and hypokalaemia

Hypersensitivity
Hypersensitivity, drug hypersensitivity and anaphylactic reaction

Diabetic ketoacidosis
Ketoacidosis

Refer to the SmPC or your TRUQAP dosing guide for full details on the monitoring requirements for patients on treatment

Hyperglycaemia, diarrhoea, cutaneous adverse drug reactions and co-administration with bisphosphonates or RANK-ligand inhibitors are listed as special warnings in the TRUQAP SmPC.[1] Please see the TRUQAP SmPC for full information on adverse events and their management.

DKA, diabetic ketoacidosis; RANK, receptor activator of nuclear factor kappa-B; SmPC, Summary of Product Characteristics.

TRUQAP + fulvestrant is a PI3K/AKT pathway-directed combination that shows:

Significant improvement in mPFS[1]

7.3 months mPFS (95% CI: 5.5, 9.0) with TRUQAP + fulvestrant (n=155)[1]

– VS –

3.1 months mPFS (95% CI: 2.0, 3.7) with placebo + fulvestrant (n=134)[1]

HR 0.50 (95% CI: 0.38, 0.65; *P*<0.001)[1]

Dual inhibition

Delivers the dual effects of AKT inhibition and ER downregulation (*based on preclinical studies; clinical significance is not known*)[2,10,11]

In an exploratory analysis of PFS by altered gene, adding TRUQAP to fulvestrant provided a numerical PFS benefit vs placebo + fulvestrant regardless of gene alteration detected (*statistical significance not performed*)*[5]

The majority of AEs were grade 1 or 2[1]

The most common any-grade AEs with TRUQAP + fulvestrant were:[1]

- Diarrhoea (72.4% any grade; 9.3% grade ≥3)
- Cutaneous adverse drug reactions (46.5% any grade; 14.9% grade ≥3)
- Nausea (34.6% any grade; 0.8% grade ≥3)

*mPFS in patients with *PIK3CA*, *AKT1* and *PTEN* alterations only was 5.6 months (95% CI: 4.2, 7.4), 9.1 months (95% CI: 2.2, NC) and 9.1 months (95% CI: 5.5, 11.1) with TRUQAP + fulvestrant vs 2.1 months (95% CI: 1.9, 3.6), 3.7 months (95% CI: 1.7, 9.5) and 3.6 months (95% CI: 1.8, 6.7) with placebo + fulvestrant, respectively.[5]

AE, adverse event; AKT, serine/threonine protein kinase; *AKT1*, serine/threonine protein kinase 1; CI, confidence interval; ER, oestrogen receptor; HR, hazard ratio; mPFS, median progression-free survival; NC, not calculable; PFS, progression-free survival; PI3K, phosphoinositide 3-kinase; *PIK3CA*, phosphatidylinositol-4,5-bisphosphate 3-kinase catalytic subunit alpha; *PTEN*, phosphatase and tensin homologue.

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For your eligible patients with HR+/HER2– aBC with *PIK3CA/AKT1/PTEN*-altered tumours, explore extending time on endocrine therapy:[1,2]

- Test for *PIK3CA/AKT1/PTEN* alterations at aBC diagnosis and disease progression
- Consider treatment with TRUQAP + fulvestrant

Click or scan the QR code to view the TRUQAP UK Prescribing Information

Click or scan the QR code to view fulvestrant UK Prescribing Information

aBC, advanced breast cancer; *AKT1*, serine/threonine protein kinase 1; HER2, human epidermal growth factor receptor 2; HR+, hormone receptor positive; *PIK3CA*, phosphatidylinositol-4,5-bisphosphate 3-kinase catalytic subunit alpha; *PTEN*, phosphatase and tensin homologue.

References: ***1.*** TRUQAP (capivasertib). Summary of Product Characteristics. ***2.*** Turner NC, et al. *N Engl J Med.* 2023;388:2058–70. ***3.*** Turner NC, et al. *N Engl J Med.* 2023;388:2058–70. Protocol. ***4.*** Turner NC, et al. *N Engl J Med.* 2023;388:2058–70. Supplementary appendix. ***5.*** Howell S, et al. Presented at: San Antonio Breast Cancer Symposium (SABCS), 5–9 December 2023; San Antonio, TX, USA. Poster PS17-03. ***6.*** Gennari A, et al. *Ann Oncol.* 2021;32:1475–95. ***7.*** Rugo H, et al. Presented at: European Society for Medical Oncology (ESMO) Breast Cancer Annual Congress; 15–17 May 2024; Berlin, Germany. ***8.*** Turner NC, et al. Presented at: San Antonio Breast Cancer Symposium (SABCS); 9–12 December 2025; San Antonio, TX, USA. Oral presentation GS3-10. ***9.*** NICE. Capivasertib with fulvestrant for treating hormone receptor-positive HER2–negative advanced breast cancer after endocrine treatment [TA1063]. May 2025. ***10.*** Fulvestrant. Summary of Product Characteristics. ***11.*** Ribas R, et al. *Mol Cancer Ther.* 2015;14:2035–48.