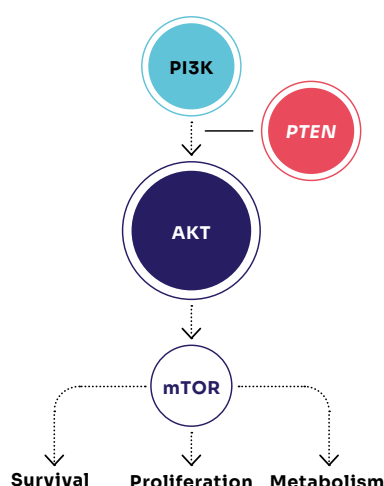


Unlocking the potential of targeting the PI3K/AKT pathway in HR+/HER2- aBC

Identifying patients who may be eligible for treatment with TRUQAP (capivasertib) + fulvestrant

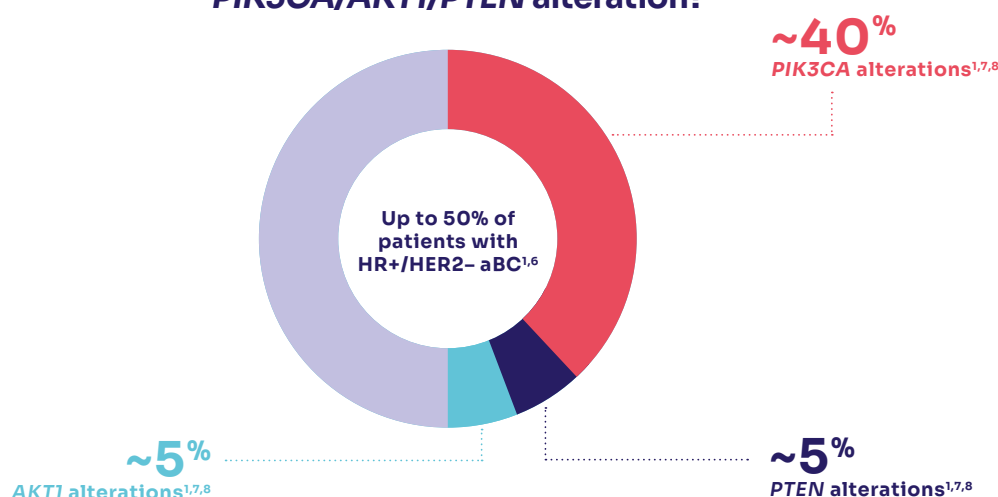


Why is the PI3K/AKT pathway important in breast cancer?

- The PI3K/AKT pathway mediates critical cellular processes, such as cell proliferation, survival and metabolism^{1,2}
- Activation of the PI3K/AKT pathway in HR+/HER2- breast cancer can occur through alterations in *PIK3CA*, *AKT1* and/or *PTEN* genes^{1,3}
- Abnormal PI3K/AKT pathway activation mediates resistance to multiple therapies in breast cancer, including ETs and CDK4/6is^{2,4}

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.⁵

How many of my patients are likely to have a *PIK3CA*/*AKT1*/*PTEN* alteration?



When should *PIK3CA*/*AKT1*/*PTEN* alteration testing be carried out?

M3.6 at diagnosis

Tissue-based, multi-target NGS panel to detect alterations in the *PIK3CA*, *AKT1* and *PTEN* genes to **aid diagnosis and management**⁹

Tissue testing at diagnosis is the **gold standard**¹⁰ & **ESMO recommended**¹¹

Check prior NGS reports

Consult with your genomic testing provider if further information on *PIK3CA*/*AKT1*/*PTEN* status can be made available from the previous NGS test

M3.13 at progression

If no previous *PIK3CA*, *AKT1* or *PTEN* alteration was identified, test using ctDNA, multi-target NGS panel at progression to **guide treatment decisions**^{*9}

ESMO endorsed in routine practice^{12,13}

How should testing be performed?



Test using **tissue biopsy** at initial aBC **diagnosis**⁹



If diagnostic biopsy is unavailable, **archival tissue** may be used instead¹⁴



Test using ctDNA from a **blood sample** at **disease progression**⁹

*Patients with HR+ HER 2-locally advanced or metastatic breast cancer that are progressing on 1L treatment with an aromatase inhibitor plus CDK4/6i.

Identify your patients' ***PIK3CA***, ***AKT1*** and ***PTEN*** alteration status as early as possible to inform future treatment decisions

1L, first-line; aBC, advanced breast cancer; AKT, serine/threonine protein kinase; *AKT1*, serine/threonine protein kinase 1; CDK4/6i, cyclin-dependent kinases 4 and 6 inhibitor; DNA, deoxyribonucleic acid; CNV, copy number variant; ctDNA, circulating tumour DNA; ESMO=European Society for Medical Oncology; ET, endocrine therapy; HER2, human epidermal growth factor receptor 2; HR+, hormone receptor positive; IHC, immunohistochemistry; ISH, *in situ* hybridisation; MDT, multidisciplinary team; mTOR, mammalian target of rapamycin; NGS, next-generation sequencing; *PIK3CA*, phosphatidylinositol-4,5-bisphosphate 3-kinase catalytic subunit alpha; PI3K, phosphoinositide 3-kinase; *PTEN*, phosphatase and tensin homologue.

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