

## Product datasheet

# Androgen Receptor peptide ab174742

### Overview

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**Product name** Androgen Receptor peptide

### Description

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**Nature** Synthetic

### Specifications

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Our [Abpromise guarantee](#) covers the use of **ab174742** in the following tested applications.

The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

**Applications** Blocking - Blocking peptide for Anti-Androgen Receptor antibody [EP670Y] ([ab52615](#))

**Form** Liquid

**Additional notes**

- First try to dissolve a small amount of peptide in either water or buffer. The more charged residues on a peptide, the more soluble it is in aqueous solutions.
- If the peptide doesn't dissolve try an organic solvent e.g. DMSO, then dilute using water or buffer.
- Consider that any solvent used must be compatible with your assay. If a peptide does not dissolve and you need to recover it, lyophilise to remove the solvent.
- Gentle warming and sonication can effectively aid peptide solubilisation. If the solution is cloudy or has gelled the peptide may be in suspension rather than solubilised.
- Peptides containing cysteine are easily oxidised, so should be prepared in solution just prior to use.

### Preparation and Storage

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**Stability and Storage** Shipped at 4°C. Store at -20°C.  
Information available upon request.

### General Info

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**Function** Steroid hormone receptors are ligand-activated transcription factors that regulate eukaryotic gene expression and affect cellular proliferation and differentiation in target tissues. Transcription factor activity is modulated by bound coactivator and corepressor proteins. Transcription activation is

down-regulated by NR0B2. Activated, but not phosphorylated, by HIPK3 and ZIPK/DAPK3. Isoform 3 and isoform 4 lack the C-terminal ligand-binding domain and may therefore constitutively activate the transcription of a specific set of genes independently of steroid hormones.

**Tissue specificity**

Isoform 2 is mainly expressed in heart and skeletal muscle (PubMed:15634333). Isoform 3 is expressed by basal and stromal cells of prostate (at protein level) (PubMed:19244107).

**Involvement in disease**

Androgen insensitivity syndrome

Spinal and bulbar muscular atrophy X-linked 1

Defects in AR may play a role in metastatic prostate cancer. The mutated receptor stimulates prostate growth and metastases development despite of androgen ablation. This treatment can reduce primary and metastatic lesions probably by inducing apoptosis of tumor cells when they express the wild-type receptor.

Androgen insensitivity, partial

**Sequence similarities**

Belongs to the nuclear hormone receptor family. NR3 subfamily.

Contains 1 nuclear receptor DNA-binding domain.

**Domain**

Composed of three domains: a modulating N-terminal domain, a DNA-binding domain and a C-terminal ligand-binding domain. In the presence of bound steroid the ligand-binding domain interacts with the N-terminal modulating domain, and thereby activates AR transcription factor activity. Agonist binding is required for dimerization and binding to target DNA. The transcription factor activity of the complex formed by ligand-activated AR and DNA is modulated by interactions with coactivator and corepressor proteins. Interaction with RANBP9 is mediated by both the N-terminal domain and the DNA-binding domain. Interaction with EFCAB6/DJBP is mediated by the DNA-binding domain.

**Post-translational modifications**

Sumoylated on Lys-388 (major) and Lys-521. Ubiquitinated. Deubiquitinated by USP26. 'Lys-6' and 'Lys-27'-linked polyubiquitination by RNF6 modulates AR transcriptional activity and specificity.

Phosphorylated in prostate cancer cells in response to several growth factors including EGF. Phosphorylation is induced by c-Src kinase (CSK). Tyr-535 is one of the major phosphorylation sites and an increase in phosphorylation and Src kinase activity is associated with prostate cancer progression. Phosphorylation by TNK2 enhances the DNA-binding and transcriptional activity and may be responsible for androgen-independent progression of prostate cancer.

Phosphorylation at Ser-83 by CDK9 regulates AR promoter selectivity and cell growth.

Phosphorylation by PAK6 leads to AR-mediated transcription inhibition.

Palmitoylated by ZDHHC7 and ZDHHC21. Palmitoylation is required for plasma membrane targeting and for rapid intracellular signaling via ERK and AKT kinases and cAMP generation.

**Cellular localization**

Nucleus. Cytoplasm. Predominantly cytoplasmic in unligated form but translocates to the nucleus upon ligand-binding. Can also translocate to the nucleus in unligated form in the presence of RACK1.

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