

AR (Acetyl Lys632) Rabbit Polyclonal Antibody

Catalog# UZ-PA00811

Product information

产品货号	UZ-PA00811
别名	Androgen receptor (Dihydrotestosterone receptor;Nuclear receptor subfamily 3 group C member 4);DHTR;NR3C4
产品名称	AR (Acetyl Lys632) Rabbit Polyclonal Antibody
类别	抗体产品
基因名称	AR
推荐应用	WB,ELISA
反应种属	Human,Mouse
存储缓冲液	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% New type preservative N .
Human Gene ID	367
Human Swissprot No.	P10275
免疫原	Synthesized peptide derived from human AR (Acetyl Lys632)
稀释度	WB 1:1000-2000, ELISA 1:5000-20000
预测分子量	105kDa
运输及保存条件	-20°C/1 year
宿主	Rabbit
同种型	Rabbit,IgG

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背景介绍

disease:Defects in AR are the cause of androgen insensitivity syndrome (AIS) [MIM:300068]; previously known as testicular feminization syndrome (TFM). AIS is an X-linked recessive form of pseudohermaphroditism due end-organ resistance to androgen. Affected males have female external genitalia, female breast development, blind vagina, absent uterus and female adnexa, and abdominal or inguinal testes, despite a normal 46,XY karyotype.,disease:Defects in AR are the cause of androgen insensitivity syndrome partial (PAIS) [MIM:312300]; also known as Reifenstein syndrome. PAIS is characterized by hypospadias, hypogonadism, gynecomastia, genital ambiguity, normal XY karyotype, and a pedigree pattern consistent with X-linked recessive inheritance. Some patients present azoospermia or severe oligospermia without other clinical manifestations.,disease:Defects in AR are the cause of spinal and bulbar muscular atrophy X-linked type 1 (SMAX1) [MIM:313200]; also known as Kennedy disease. SMAX1 is an X-linked recessive form of spinal muscular atrophy. Spinal muscular atrophy refers to a group of neuromuscular disorders characterized by degeneration of the anterior horn cells of the spinal cord, leading to symmetrical muscle weakness and atrophy. SMAX1 occurs only in men. Age at onset is usually in the third to fifth decade of life, but earlier involvement has been reported. It is characterized by slowly progressive limb and bulbar muscle weakness with fasciculations, muscle atrophy, and gynecomastia. The disorder is clinically similar to classic forms of autosomal spinal muscular atrophy. SMAX1 is caused by trinucleotide CAG repeat expansion. In SMAX1 patients the number of Gln ranges from 38 to 62. Longer expansions result in earlier onset and more severe clinical manifestations of the disease.,disease: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.,domain:Composed of three domains: a modulating N-terminal domain, a DNA-binding domain and a C-terminal steroid-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.,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.,miscellaneous:In the absence of ligand, steroid hormone receptors are thought to be weakly associated with nuclear components; hormone binding greatly increases receptor affinity. The hormone-receptor complex appears to recognize discrete DNA sequences upstream of transcriptional start sites.,miscellaneous:The level of tyrosine phosphorylation may serve as a diagnostic tool to predict patient outcome in response to hormone-ablation therapy. Inhibition of tyrosine phosphorylation may be an effective intervention target for hormone-refractory prostate cancer.,miscellaneous:Transcriptional activity is enhanced by binding to RANBP9.,online information:Androgen receptor entry.,polymorphism:The poly-Gln region of AR is highly polymorphic and the number of Gln varies in the population (from 17 to 26). A smaller size of the poly-Gln region may be associated with the development of prostate cancer.,polymorphism:The poly-Gly region of AR is polymorphic and ranges from 24 to 31 Gly. A poly-Gly region shorter or equal to 23 may be associated with the development of androgenetic alopecia.,PTM:Phosphorylated in prostate cancer cells in response to several growth factors including EGF. Phosphorylation is induced by c-Src kinase (CSK). Tyr-534 is one of the major phosphorylation sites and an increase in phosphorylation and Src kinase activity is associated with prostate cancer progression.