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# AKR1C3 Rabbit mAb

Catalog # AP79045

## Product Information

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|---|---|
| **Application** | WB, IF, FC, ICC, IP |
| **Primary Accession** | P42330 |
| **Reactivity** | Human |
| **Host** | Rabbit |
| **Clonality** | Monoclonal Antibody |
| **Isotype** | IgG |
| **Conjugate** | Unconjugated |
| **Immunogen** | A synthesized peptide derived from human AKR1C3 |
| **Purification** | Affinity Chromatography |
| **Calculated MW** | 36853 |

## Additional Information

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|---|---|
| **Gene ID** | 8644 |
| **Other Names** | AKR1C3 |
| **Dilution** | WB~~1/500-1/1000 IF~~1/50-1/200 FC~~1:10~50 ICC~~N/A IP~~N/A |
| **Format** | Liquid in 10mM PBS, pH 7.4, 150mM sodium chloride, 0.05% BSA, 0.02% sodium azide and 50% glycerol. |
| **Storage** | Store at 4°C short term. Aliquot and store at -20°C long term. Avoid freeze/thaw cycles. |

## Protein Information

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|---|---|
| **Name** | AKR1C3 |
| **Function** | Cytosolic aldo-keto reductase that catalyzes NADPH-dependent reduction of ketosteroids to hydroxysteroids. Displays broad substrate specificity with distinct positional and stereochemistry, primarily generating 17beta-hydroxysteroids, but also 3alpha- and 20alpha- hydroxysteroids (PubMed:10998348, PubMed:11165022, PubMed:20036328, PubMed:9415401, PubMed:9927279, PubMed:10998348, PubMed:9927279). Produces potent androgens via classical and 'backdoor'/alternative pathways. In the classical androgen metabolic pathway (biosynthesis of 5alpha-dihydrotestosterone (5alpha-DHT) via testosterone), catalyzes the reduction of delta4-androstenedione to form testosterone (PubMed:10998348, PubMed:11165022, PubMed:20036328, PubMed:9415401, PubMed:9927279). In the 'backdoor' androgen metabolic pathway (biosynthesis of 5alpha-dihydrotestosterone (5alpha-DHT) via pregnanes), reduces androsterone to 5alpha-androstane-3alpha,17beta- diol |

preceding 5alpha-DHT secretion (PubMed:10557352, PubMed:10998348, PubMed:9415401). Reduces 5alpha-DHT to less potent androgen 5alpha-androstane-3alpha,17beta-diol, likely regulating ligand availability for androgen receptors (PubMed:10557352, PubMed:10998348, PubMed:11165022, PubMed:14672942, PubMed:7650035, PubMed:9415401). May contribute to the metabolism of adrenal-derived androgen precursors. Reduces 11-keto-4-androstene-3,17-dione (11KA4) and 11-keto-5alpha-androstane-3,17-dione (11K-Adione) into potent androgens 11-ketotestosterone (11KT) and 11-ketodihydrotestosterone (11KDHT), respectively (PubMed:31926269). In estrogen metabolism, catalyzes the conversion of estrone to potent estrogen 17beta-estradiol (PubMed:10998348, PubMed:11165022, PubMed:20036328). Acts as a prostaglandin (PG) F2alpha synthase. Displays 11-ketoreductase and 9,11-endoperoxide reductase activities and reduces PGD2 to 11beta-PGF2alpha and PGH2 to PGF2alpha (PubMed:10622721, PubMed:11165022, PubMed:15047184, PubMed:19010934, PubMed:20036328, PubMed:7650035, PubMed:9415401, PubMed:9927279). Also displays retinaldehyde reductase activity toward 9-cis-retinal (PubMed:21851338). In vitro can efficiently catalyze bidirectional conversion between ketosteroids and hydroxysteroids using NADPH/NADP(+) or NADH/NAD(+) as cofactors. In vivo however, the reductase activity prevails since the major reducing cofactor NADPH inhibits NAD(+)-dependent oxidase activity (PubMed:11165022, PubMed:14672942). In addition, it is able to reduce in vitro various carbonyl compounds like menadione, phenanthrenequinone and nitrobenzaldehyde (By similarity).

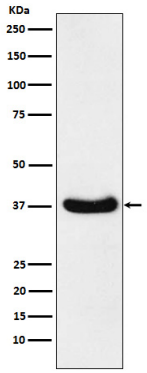
**Cellular Location**

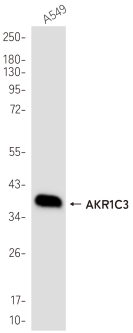
Cytoplasm.

**Tissue Location**

Expressed in many tissues including adrenal gland, brain, kidney, liver, lung, mammary gland, placenta, small intestine, colon, spleen, prostate and testis. High expression in prostate and mammary gland. In the prostate, higher levels in epithelial cells than in stromal cells. In the brain, expressed in medulla, spinal cord, frontotemporal lobes, thalamus, subthalamic nuclei and amygdala. Weaker expression in the hippocampus, substantia nigra and caudate

## Images



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