

DESCRIPTION

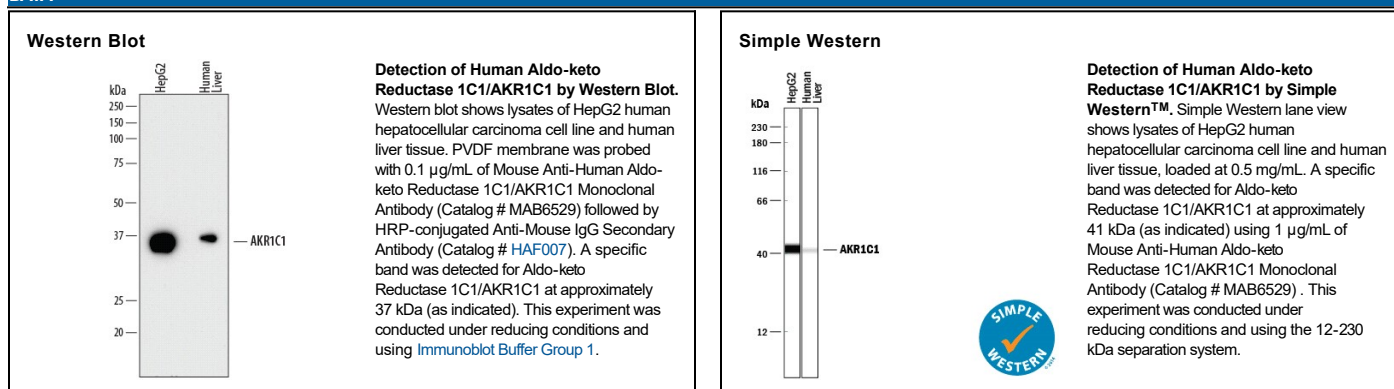
Species Reactivity	Human
Specificity	Detects human Aldo-keto Reductase 1C1/AKR1C1 in ELISAs and Western blots. In direct ELISAs, less than 5% cross-reactivity with recombinant human (rh) Aldo-keto Reductase 1C3 and 1C4 is observed.
Source	Monoclonal Mouse IgG ₁ Clone # 859026
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	<i>E. coli</i> -derived recombinant human Aldo-keto Reductase 1C1/AKR1C1 Met1-Tyr323 Accession # Q04828
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details. *Small pack size (-SP) is supplied either lyophilized or as a 0.2 µm filtered solution in PBS.

APPLICATIONS

Please Note: Optimal dilutions should be determined by each laboratory for each application. *General Protocols* are available in the *Technical Information* section on our website.

	Recommended Concentration	Sample
Western Blot	0.1 µg/mL	See Below
Simple Western	1 µg/mL	See Below

DATA



PREPARATION AND STORAGE

Reconstitution	Sterile PBS to a final concentration of 0.5 mg/mL.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below. *Small pack size (-SP) is shipped with polar packs. Upon receipt, store it immediately at -20 to -70 °C
Stability & Storage	Use a manual defrost freezer and avoid repeated freeze-thaw cycles. <ul style="list-style-type: none"> • 12 months from date of receipt, -20 to -70 °C as supplied. • 1 month, 2 to 8 °C under sterile conditions after reconstitution. • 6 months, -20 to -70 °C under sterile conditions after reconstitution.

BACKGROUND

AKR1C1 (20- α -hydroxysteroid dehydrogenase, 20- α -HSD) is a member of aldo-keto reductase (AKR) superfamily. AKRs perform the NAD(P)H-dependent reduction of carbonyl groups (1). Four AKR1C isoforms (AKR1C1-C4) are known to exist in humans. They are all highly expressed in the liver. Three isoforms, excluding AKR1C4, have a wider expression pattern including prostate, testes, uterus, mammary gland, and haemopoietic progenitors (2). These enzymes are able to accept various natural steroids as substrates, including 3-, 7-, and 20-ketosteroids (3). They can also activate prodrugs such as synthetic steroid hormone tibolone by converting it into active 3 α / β -hydroxy form (4). They are recognized as phase I drug-metabolizing enzymes involved in the maintenance of steroid homeostasis, prostaglandin metabolism, and metabolic activation of polycyclic aromatic hydrocarbons (5). Their reactions introduce a hydroxyl group into the product making it available for sulfonation and glucuronidation by phase II enzyme. Elevated expression of these enzymes is related to cancer with hormone-dependent malignancies (6, 7). Increased levels of expression of AKR1C1 parallels increased cell proliferation activity in human colon cancer cells. It has been shown to be associated with oncogenic potential and proproliferative effects. It is also involved in cancer cell chemoresistance.

References:

1. Jez, J.M. *et al.* (1997) *Biochem. J.* **326**:499.
2. Penning, T.M. *et al.* (2000) *Biochem. J.* **351**:67.
3. Rizner, T.L. *et al.* (2003) *Endocrinology.* **144**:2922.
4. Steckelbroeck, S. *et al.* (2006) *J. Pharmacol. Exp. Ther.* **316**:1300.
5. Penning, T.M. *et al.* (2004) *Mol. Cell. Endocrinol.* **1784**:1342.
6. Penning, T.M. and M.C. Byrns (2009) *Ann. N. Y. Acad. Sci.* **1155**:33.
7. Baumann, D.R. *et al.* (2004) *Drug News Perspect.* **17**:563.