

BACKGROUND

Hydroxysteroid (11-beta) dehydrogenase (HSD11B1) is a microsomal enzyme that catalyses the intracellular regeneration of active glucocorticoids (cortisol, corticosterone) from circulating inert 11-keto forms (cortisone, 11dehydrocorticosterone). The biologically active glucorticoids are ligand for the receptor subfamily 3, group C, member 1 (glucocorticoid receptor) (NR3C1). The enzyme acts to amplify glucocorticoid action locally.¹ This enzyme is ubiquitously expressed in human at a high level in liver and adipose tissue. The HSD11B1 expression can be regulated differently by hormones, growth factors, and cytokines. TNF-alpha increases HSD11B1 expression and activity. The activity of HSD11B1 is also stimulated by P4, prostaglandins, and cortisol. Interestingly, HSD11B1 is also highly expressed in the adult CNS in rodents and humans, notably in the hippocampus, frontal cortex and other regions key to cognitive processes. Transgenic mice lacking HSD11B1 are protected from the deficits in cognitive function with ageing that are seen in many control animals. Moreover, treatment with HSD11B1 inhibitors in humans appears to improve cognitive function in healthy elderly men and patients with type 2 diabetes.² Thus, HSD11B1 inhibition is a target for cognitive protection against ageing. In addition, the reductase activity of HSD11B1 plays an important role in the growth and differentiation of adipose tissue via the activation of glucocorticoids. Increased HSD11B1 activity has been proposed as a mechanism underlying the association between multiple cardiovascular risk factors, whereas inhibition of HSD11B1 has been proposed for the treatment of diabetes and central obesity. Furthermore, it was also shown that that HSD11B1-regenerated cortisol acts via NR3C1 to regulate ovine endometrial functions during early pregnancy.³ The polymorphism in the HSD11B1 gene was reported to associate with an increased risk of several diseases including Alzheimer's disease, Left ventricular mass (LVM) and may be useful genetic markers for bone metabolism.⁴

References:

1. Paterson, J.M. et al: Am. J. Physiol. Regul. Integr. Comp. Physiol. 289: R642-R652, 2005 2. Deary, I.J. et al: Neurosci. Lett. 393:74-7, 2006 3. Simmons, R.M. et al: Biol Reprod. 82:35-43, 2010 3. Hwang, J.Y. et al: Bone 45:1098-103, 2009

TECHNICAL INFORMATION

Source:

Produced in rabbits immunized with a synthetic peptide corresponding to a sequence near the N-terminal of human HSD11B1, identical to the related rat and mouse sequence. HSD11B1 specific antibody was purified by peptide affinity chromatography.

Specificity and Sensitivity:

It reacts specifically with HSD11B1 of human, rabbit, mouse, and rat origin in immunostaining and western blotting, no cross-reactivity with other members of the family.

Storage Buffer: 10mM HEPES (pH 7.5), 150mM NaCl, 100µg/ml BSA, 100µg/ml Sodium Azide and 50% Glycerol

Storage:

Store at -20°C for at least one year. Store at 4°C for frequent use. Avoid repeated freeze-thaw cycles.

APPLICATIONS

Application:	*Dilution:
WB	1:100 - 400
IP	n/d
IHC	1:50 - 200
ICC	n/d
FACS	n/d
*Optimal dilutions must be determined by end user.	

QUALITY CONTROL DATA





Top: Detection of HSD11B1 from rat liver tissue lysate in Western blot assay, using Anti-HSD11B1. **Bottom:** Immunohistochemical staining of paraffin-embedded rat liver tissue, using Anti-HSD11B1.

