

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human Dimethylarginine Dimethylaminohydrolase 1/DDAH1 in direct ELISAs.
Source	Polyclonal Sheep IgG
Purification	Antigen Affinity-purified
Immunogen	<i>E. coli</i> -derived recombinant human Dimethylarginine Dimethylaminohydrolase 1/DDAH1 Ala2-Ser285 Accession # O94760
Conjugate	Alexa Fluor 350 Excitation Wavelength: 346 nm Emission Wavelength: 442 nm
Formulation	Supplied 0.2mg/ml in 1X PBS with RDF1 and 0.09% Sodium Azide
*Contains <0.1% Sodium Azide, which is not hazardous at this concentration according to GHS classifications. Refer to the Safety Data Sheet (SDS) for additional information and handling instructions.	

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.

Immunohistochemistry Optimal dilution of this antibody should be experimentally determined.

PREPARATION AND STORAGE

Shipping	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	Protect from light. Do not freeze. 12 months from date of receipt, 2 to 8 °C as supplied

BACKGROUND

Dimethylarginine Dimethylaminohydrolase (DDAH) metabolizes asymmetric dimethyl arginine (ADMA) to L-citrulline and dimethylamine, and N^G-monomethyl arginine (MMA) to L-citrulline and monomethylamine (1). Two members of the DDAH family have been identified in humans. DDAH1 is widely expressed, especially in liver and kidney. DDAH2 predominates in vascular endothelium and expressed selectively in kidney (2). It is also expressed in immune tissues including spleen, thymus, peripheral leukocytes, lymph nodes, and bone marrow. Over 90 % of endogenous ADMA is metabolized by DDAH with the remainder excreted (3). ADMA and MMA are endogenous inhibitors of nitric oxide synthase (NOS). Thus, enzymes of the DDAH family play a key role in vascular function through the turnover of methylated arginine (4). It has been observed that genetic variation in the DDAH1 and DDAH2 genes is significantly associated with serum ADMA levels (5).

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