

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human Placental Lactogen/CSH1 in direct ELISAs.
Source	Monoclonal Mouse IgG _{2A} Clone # 658234
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Chinese Hamster Ovary cell line CHO-derived human Placental Lactogen/CSH1 Val27-Phe217 Accession # P01243
Conjugate	Alexa Fluor 594 Excitation Wavelength: 590 nm Emission Wavelength: 617 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.

Neutralization 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

Human Placental Lactogen (abbreviated PL or hPL), also called Chorionic Somatomammotropin Hormone 1 (abbreviated CSH1), is a member of the prolactin/growth hormone (PRL/GH) family (1). It is found in a cluster of growth hormones on chromosome 17 that appear to have a common ancestry. Identical 191 amino acid (aa) mature hPL proteins may be formed from one of two genes (2). PL contains a pair of C-terminal cysteines that may form either intra- or interchain disulfides. Human PL shares 98% aa identity with chimpanzee PL and >85% aa sequence identity with other human growth hormones, but only ~25% aa identity with mouse, ovine or bovine PL. PL is mainly expressed by cells in the syncytiotrophoblast layer of the placenta, which produce increasing amounts of PL as pregnancy proceeds. The major portion enters the maternal circulation, where it joins GH2 (placenta-specific GH) in replacing the functions of pituitary GH during pregnancy. A smaller amount of PL circulates in the fetus. Primate PL shows high affinity for the PRL receptor and low affinity for the GH receptor (1). Reduced stimulation of PL by angiotensin 2 correlates with intrauterine growth restriction (3). There is some evidence that mature angiogenic PL may be cleaved to form an anti-angiogenic N-terminal fragment (4). Although PL promotes pancreatic beta cell survival, it does not appear to be altered in gestational diabetes. It helps prepare mammary for lactation, but probably does not influence lactation itself. PL may be a ligand of stabilin-1, which has been proposed to regulate PL internalization and degradation or re-expression (6).

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