


Product datasheet

Anti-GLP1 antibody [8G9] (Biotin) ab121085

Overview

Product name	Anti-GLP1 antibody [8G9] (Biotin)
Description	Mouse monoclonal [8G9] to GLP1 (Biotin)
Host species	Mouse
Conjugation	Biotin
Tested applications	Suitable for: Sandwich ELISA
Species reactivity	Reacts with: Rat Predicted to work with: Mammals 
Immunogen	Synthetic peptide: HAEGTFTSNVSSYLEGQAAKEFIAWLVKGR , corresponding to amino acids 7-36 amide of GLP1, coupled to carrier and adsorbed onto aluminum hydroxide gel. Run BLAST with Run BLAST with
Epitope	C-terminal epitope of GLP1(7-36)amide.

Properties

Form	Liquid
Storage instructions	Shipped at 4°C. Store at +4°C short term (1-2 weeks). Store at -20°C or -80°C. Avoid freeze / thaw cycle.
Storage buffer	pH: 7.40 Preservative: 0.02% Sodium azide Constituents: 99% Phosphate Buffer, 0.82% Sodium chloride
Purity	Protein A purified
Clonality	Monoclonal
Clone number	8G9
Myeloma	x63-Ag8.653
Isotype	IgG1
Light chain type	kappa

Applications

Our [Abpromise guarantee](#) covers the use of **ab121085** in the following tested applications.

The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

Application	Abreviews	Notes
Sandwich ELISA		1/4000. as capture antibody. The dilution guideline for ELISA is based on use as detection antibody for antigen coated at 0.1-1 µg/ml.

Target

Function

Glucagon plays a key role in glucose metabolism and homeostasis. Regulates blood glucose by increasing gluconeogenesis and decreasing glycolysis. A counterregulatory hormone of insulin, raises plasma glucose levels in response to insulin-induced hypoglycemia. Plays an important role in initiating and maintaining hyperglycemic conditions in diabetes.

GLP-1 is a potent stimulator of glucose-dependent insulin release. Play important roles on gastric motility and the suppression of plasma glucagon levels. May be involved in the suppression of satiety and stimulation of glucose disposal in peripheral tissues, independent of the actions of insulin. Have growth-promoting activities on intestinal epithelium. May also regulate the hypothalamic pituitary axis (HPA) via effects on LH, TSH, CRH, oxytocin, and vasopressin secretion. Increases islet mass through stimulation of islet neogenesis and pancreatic beta cell proliferation. Inhibits beta cell apoptosis.

GLP-2 stimulates intestinal growth and up-regulates villus height in the small intestine, concomitant with increased crypt cell proliferation and decreased enterocyte apoptosis. The gastrointestinal tract, from the stomach to the colon is the principal target for GLP-2 action. Plays a key role in nutrient homeostasis, enhancing nutrient assimilation through enhanced gastrointestinal function, as well as increasing nutrient disposal. Stimulates intestinal glucose transport and decreases mucosal permeability.

Oxyntomodulin significantly reduces food intake. Inhibits gastric emptying in humans.

Suppression of gastric emptying may lead to increased gastric distension, which may contribute to satiety by causing a sensation of fullness.

Glicentin may modulate gastric acid secretion and the gastro-pyloro-duodenal activity. May play an important role in intestinal mucosal growth in the early period of life.

Tissue specificity

Glucagon is secreted in the A cells of the islets of Langerhans. GLP-1, GLP-2, oxyntomodulin and glicentin are secreted from enteroendocrine cells throughout the gastrointestinal tract. GLP1 and GLP2 are also secreted in selected neurons in the brain.

Sequence similarities

Belongs to the glucagon family.

Post-translational modifications

Proglucagon is post-translationally processed in a tissue-specific manner in pancreatic A cells and intestinal L cells. In pancreatic A cells, the major bioactive hormone is glucagon cleaved by PCSK2/PC2. In the intestinal L cells PCSK1/PC1 liberates GLP-1, GLP-2, glicentin and oxyntomodulin. GLP-1 is further N-terminally truncated by post-translational processing in the intestinal L cells resulting in GLP-1(7-37) GLP-1(7-36)amide. The C-terminal amidation is neither important for the metabolism of GLP-1 nor for its effects on the endocrine pancreas.

Cellular localization

Secreted.

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