

Human/Mouse Proinsulin Alexa Fluor® 488-conjugated Antibody

Monoclonal Mouse IgG_{2A} Clone # 253627 Catalog Number: IC13361G

100 µg

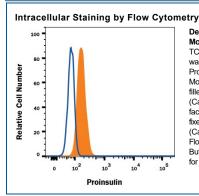
DESCRIPTION			
Species Reactivity	Human/Mouse		
Specificity	Detects human and mouse Proinsulin. Does not detect mature insulin.		
Source	Monoclonal Mouse IgG _{2A} Clone # 253627		
Purification	Protein A or G purified from hybridoma culture supernatant		
Immunogen	E. coli-derived recombinant human Proinsulin Phe25-Asn110 Accession # P01308		
Conjugate	Alexa Fluor 488 Excitation Wavelength: 488 nm Emission Wavelength: 515-545 nm		
Formulation	Supplied in a saline solution containing BSA and Sodium Azide. See Certificate of Analysis for details.		
	*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.

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	Recommended Concentration	Sample	
Intracellular Staining by Flow Cytometry	0.25-1 µg/10 ⁶ cells	See Below	

DATA



Detection of Proinsulin in beta TC-6
Mouse Cell Line by Flow Cytometry. Beta
TC-6 Mouse beta cell insulinoma cell line
was stained with Mouse Anti-Human/Mouse
Proinsulin Alexa Fluor® 488-conjugated
Monoclonal Antibody (Catalog # IC13361G,
filled histogram) or isotype control antibody
(Catalog # IC003G, open histogram). To
facilitate intracellular staining, cells were
fixed with Flow Cytometry Fixation Buffer
(Catalog # FC004) and permeabilized with
Flow Cytometry Permeabilization/Wash
Buffer I(Catalog # FC005). View our protocol
for Staining Intracellular Molecules.

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.





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BACKGROUND

Proinsulin is synthesized as a single chain, 110 amino acid (aa) preproprecursor that contains a 24 aa signal sequence and an 86 aa proinsulin propeptide. Following removal of the signal peptide, the proinsulin peptide undergoes further proteolysis to generate mature insulin, a 51 aa disulfide-linked dimer that consists of a 30 aa B chain (aa 25-54) bound to a 21 aa A chain (aa 90-110). The 34 aa intervening peptide (aa 55-89) that connects the B and A chains is termed the C-peptide. Human proinsulin shares 84% and 80% aa sequence identity with rat and bovine proinsulin, respectively. Most of the sequence variation between species occurs in the region of the C-peptide (1). This peptide generates a structural conformation that allows for the correct formation of the intrachain disulphide bonds (1). Insulin is a molecule that facilitates the cellular uptake of glucose. This is accomplished by regulating the appearance of membrane glucose transporters. Low insulin levels or lack of insulin are associated with type 2 and type 1 diabetes mellitus, respectively. These conditions are associated with an increased risk for microvascular complications such as retinopathy, nephropathy, and peripheral neuropathy (3). Proinsulin also circulates, but its physiologic role is less well understood. It does possess about 25% of the activity of mature insulin, but it would seem unlikely to be a natural substitute for insulin (4). In type 2 diabetes, an elevated proinsulin to insulin ratio in the circulation is a well-known abnormality (5-9). Perhaps this abnormality represents either compromised proteolytic processing or a general inability to process increased levels of insulin precursor (5). In any event, proinsulin will stimulate amylin secretion by β-cells, and amyloid formation in pancreatic islets that promotes decreased β cell function (10). Studies also suggest that fasting serum proinsulin may be a better predictor of future type 2 diabetes than fasting insulin levels in obese children (11).

References:

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- 3. Forst, T. et al. (2008) Exp. Diabetes Res. 2008:176245.
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- 5. Roder, M.E. et al. (1999) Diabetes Care 22:609
- 6. Porte, D. Jr. (1991) Diabetes 40:166.
- 7. Gordon, P. et al. (1974) Diabetologia 34:483.
- 8. Saad, M.F. et al. (1990) J. Clin. Endocrinol. Metab. 70:1247.
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- 10. Dworacka, M. et al. (2006) Int. J. Clin. Pharmacol. Ther. 44:14.
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