

# HCN4 Monoclonal Antibody (SHG 1E5)

Product Details	
Size	100 µL
Species Reactivity	Human, Mouse, Rat
Published Species	Rat
Host/Isotype	Rat / IgG1
Class	Monoclonal
Type	Antibody
Clone	SHG 1E5
Conjugate	Unconjugated
Immunogen	Synthetic peptide sequence corresponding to residues S H G S L L L P P A S S P P P P Q V P Q R R G T P P L T P G R L T Q D L K L of HCN4.
Form	Liquid
Concentration	Conc. Not Determined
Storage buffer	ascites
Contains	0.05% sodium azide
Storage conditions	-20° C, Avoid Freeze/Thaw Cycles
RRID	AB_2120037

Applications	Tested Dilution	Publications
Western Blot (WB)	1:5,000	1 Publication
Immunohistochemistry (Paraffin) (IHC (P))	1:10-1:100	-
Immunohistochemistry (Frozen) (IHC (F))	1:1,000	-
Flow Cytometry (Flow)	1/50	-

## Product Specific Information

MA3-903 detects HCN4 from human, mouse, and rat samples.

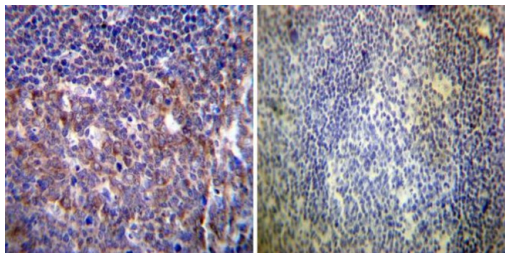
MA3-903 has been successfully used in Western blotting and immunohistochemistry procedures. By Western blot MA3-903 detects a ~132 KDa band representing HCN4.

The MA3-903 immunogen is a synthetic peptide sequence corresponding to residues S H G S L L L P P A S S P P P P Q V P Q R R G T P P L T P G R L T Q D L K L of HCN4.

MA3-903 clone SHG 1E5 is a rat monoclonal hybridoma. This antibody was produced by immunizing a rat, isolating the spleen cells, creating the hybridoma and successively injecting these cells into a mouse to produce ascites.

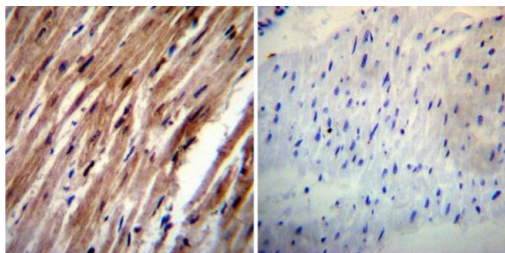
**HCN4 Antibody (MA3-903) in IHC (P)**

Immunohistochemistry was performed on normal deparaffinized Human tonsil tissue tissues. To expose target proteins, heat induced antigen retrieval was performed using 10mM sodium citrate (pH6.0) buffer, microwaved for 8-15 minutes. Following antigen retrieval tissues were blocked in 3% BSA-PBS for 30 minutes at room temperature. Tissues were then probed at a dilution of 1:20 with a rat monoclonal antibody recognizing HCN4 (Product # MA3-903) or without primary antibody (negative control) overnight at 4°C in a humidified chamber. Tissues were washed extensively with PBST and endogenous peroxidase activity was quenched with a peroxidase suppressor. Detection was performed using a biotin-conjugated secondary antibody and SA-HRP, followed by colorimetric detection using DAB. Tissues were counterstained with hematoxylin and prepped for mounting.



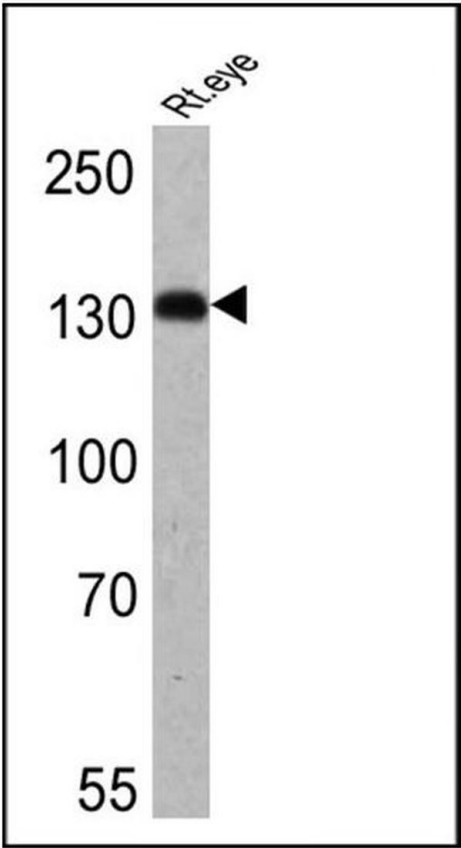
**HCN4 Antibody (MA3-903) in IHC (P)**

Immunohistochemistry was performed on normal deparaffinized Human heart tissue tissues. To expose target proteins, heat induced antigen retrieval was performed using 10mM sodium citrate (pH6.0) buffer, microwaved for 8-15 minutes. Following antigen retrieval tissues were blocked in 3% BSA-PBS for 30 minutes at room temperature. Tissues were then probed at a dilution of 1:20 with a rat monoclonal antibody recognizing HCN4 (Product # MA3-903) or without primary antibody (negative control) overnight at 4°C in a humidified chamber. Tissues were washed extensively with PBST and endogenous peroxidase activity was quenched with a peroxidase suppressor. Detection was performed using a biotin-conjugated secondary antibody and SA-HRP, followed by colorimetric detection using DAB. Tissues were counterstained with hematoxylin and prepped for mounting.



**HCN4 Antibody (MA3-903) in WB**

Western blot analysis of HCN4 was performed by loading 25 µg of rat eye lysate onto an SDS polyacrylamide gel. Proteins were transferred to a PVDF membrane and blocked at 4°C overnight. The membrane was probed with a HCN4 monoclonal antibody (Product # MA3-903) at a dilution of 1:200 overnight at 4°C, washed in TBST, and probed with an HRP-conjugated secondary antibody for 1 hr at room temperature in the dark. Chemiluminescent detection was performed using Pierce ECL Plus Western Blotting Substrate (Product # 32132). Results show a band at ~132 kDa.



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Western Blot (1)

<p>Pflugers Archiv : European journal of physiology</p> <p><b>Thalamocortical neurons display suppressed burst-firing due to an enhanced Ih current in a genetic model of absence epilepsy.</b></p> <p>"MA3-903 was used in western blot to study burst-firing in TR neurons and spike-and-wave discharges in the Genetic Absence Epilepsy Rats from Strasbourg model"</p> <p>Authors: Cain SM,Tyson JR,Jones KL,Shutch TP</p>	<p><b>Year</b> 2015</p> <p><b>Species</b> Rat</p> <p><b>Dilution</b> 1:2000</p>
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