

EGR2 Rabbit pAb

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Catalog # AP58977

Product Information

Application	WB, IHC-P, IHC-F, IF
Primary Accession	P11161
Reactivity	Human, Mouse
Predicted	Rat
Host	Rabbit
Clonality	Polyclonal
Calculated MW	50302
Physical State	Liquid
Immunogen	KLH conjugated synthetic peptide derived from human EGR2
Epitope Specificity	351-450/476
Isotype	IgG
Purity	affinity purified by Protein A
Buffer	0.01M TBS (pH7.4) with 1% BSA, 0.02% Proclin300 and 50% Glycerol.
SUBCELLULAR LOCATION	Nucleus.
DISEASE	Defects in EGR2 are a cause of congenital hypomyelination neuropathy (CHN) [MIM:605253]. Inheritance can be autosomal dominant or recessive. Recessive CHN is also known as Charcot-Marie-Tooth disease type 4E (CMT4E). CHN is characterized clinically by early onset of hypotonia, areflexia, distal muscle weakness, and very slow nerve conduction velocities. Defects in EGR2 are a cause of Charcot-Marie-Tooth disease type 1D (CMT1D) [MIM:607678]. CMT1D is a form of Charcot-Marie-Tooth disease, the most common inherited disorder of the peripheral nervous system. Charcot-Marie-Tooth disease is classified in two main groups on the basis of electrophysiologic properties and histopathology: primary peripheral demyelinating neuropathy or CMT1, and primary peripheral axonal neuropathy or CMT2. Neuropathies of the CMT1 group are characterized by severely reduced nerve conduction velocities (less than 38 m/sec), segmental demyelination and remyelination with onion bulb formations on nerve biopsy, slowly progressive distal muscle atrophy and weakness, absent deep tendon reflexes, and hollow feet. Defects in EGR2 are a cause of Dejerine-Sottas syndrome (DSS) [MIM:145900]; also known as Dejerine-Sottas neuropathy (DSN) or hereditary motor and sensory neuropathy III (HMSN3). DSS is a severe degenerating neuropathy of the demyelinating Charcot-Marie-Tooth disease category, with onset by age 2 years. DSS is characterized by motor and sensory neuropathy with very slow nerve conduction velocities, increased cerebrospinal fluid protein concentrations, hypertrophic nerve changes, delayed age of walking as well as areflexia. There are both autosomal dominant and autosomal recessive forms of Dejerine-Sottas syndrome.
Important Note	This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.
Background Descriptions	Egr proteins function in transcription regulatory activities surrounding cellular growth, differentiation and function. The deduced amino acid sequences of human Egr-2 and mouse Egr-1 are 92% identical in the zinc finger region but

show no homology elsewhere. Egr-2 is a sequence-specific DNA-binding transcription factor that binds two specific DNA sites located in the promoter region of HoxA4 and localizes to the nucleus. Defects in the Egr-2 protein are a cause of congenital hypomyelination neuropathy (CHN). CHN is characterized clinically by early onset of hypotonia, areflexia, distal muscle weakness and very slow nerve conduction velocities. Mutations in the gene that encodes Egr-2 (EGR2) also cause Dejerine-Sottas syndrome (DSS), which is also known as Dejerine-Sottas neuropathy (DSN) or hereditary motor and sensory neuropathy III (HMSN3). DSS patients exhibit severe early onset motor and sensory neuropathy with very slow nerve conduction velocities and elevated cerebrospinal fluid protein concentrations.

Additional Information

Gene ID	1959
Other Names	E3 SUMO-protein ligase EGR2, 2.3.2.-, AT591, E3 SUMO-protein transferase ERG2, Early growth response protein 2, EGR-2, Zinc finger protein Krox-20, EGR2, KROX20
Dilution	WB=1:500-2000,IHC-P=1:100-500,IHC-F=1:100-500,IF=1:100-500
Storage	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. When reconstituted in sterile pH 7.4 0.01M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.

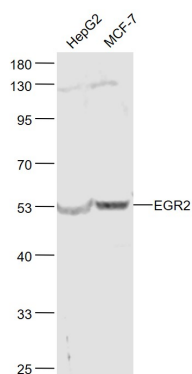
Protein Information

Name	EGR2
Synonyms	KROX20
Function	Sequence-specific DNA-binding transcription factor (PubMed: 17717711). Plays a role in hindbrain segmentation by regulating the expression of a subset of homeobox containing genes and in Schwann cell myelination by regulating the expression of genes involved in the formation and maintenance of myelin (By similarity). Binds to two EGR2- consensus sites EGR2A (5'-CTGTAGGAG-3') and EGR2B (5'-ATGTAGGTG-3') in the HOXB3 enhancer and promotes HOXB3 transcriptional activation (By similarity). Binds to specific DNA sites located in the promoter region of HOXA4, HOXB2 and ERBB2 (By similarity). Regulates hindbrain segmentation by controlling the expression of Hox genes, such as HOXA4, HOXB3 and HOXB2, and thereby specifying odd and even rhombomeres (By similarity). Promotes the expression of HOXB3 in the rhombomere r5 in the hindbrain (By similarity). Regulates myelination in the peripheral nervous system after birth, possibly by regulating the expression of myelin proteins, such as MPZ, and by promoting the differentiation of Schwann cells (By similarity). Involved in the development of the jaw opener musculature, probably by playing a role in its innervation through trigeminal motor neurons (By similarity). May play a role in adipogenesis, possibly by regulating the expression of CEBPB (By similarity).
Cellular Location	Nucleus {ECO:0000250 UniProtKB:P08152}.

Background

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Images



Sample:

HepG2(Human) Cell Lysate at 30 ug

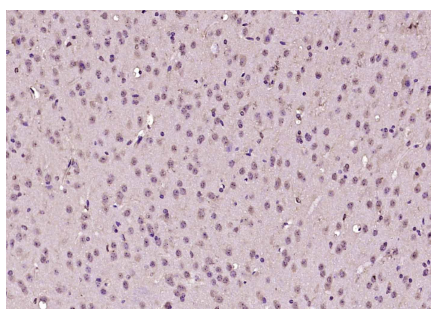
MCF-7(Human) Cell Lysate at 30 ug

Primary: Anti- EGR2 (AP58977) at 1/1000 dilution

Secondary: IRDye800CW Goat Anti-Rabbit IgG at 1/20000 dilution

Predicted band size: 50 kD

Observed band size: 52 kD



Paraformaldehyde-fixed, paraffin embedded (mouse brain); Antigen retrieval by boiling in sodium citrate buffer (pH6.0) for 15min; Block endogenous peroxidase by 3% hydrogen peroxide for 20 minutes; Blocking buffer (normal goat serum) at 37°C for 30min; Antibody incubation with (EGR2) Polyclonal Antibody, Unconjugated (AP58977) at 1:200 overnight at 4°C, followed by operating according to SP Kit(Rabbit) (sp-0023) instructions and DAB staining.

Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.