

FOX C2 Rabbit pAb

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Product Information

Application WB **Primary Accession Q99958** Reactivity Mouse

Predicted Human, Rat, Chicken

Host Rabbit Clonality Polyclonal **Calculated MW** 53719 **Physical State** Liquid

Immunogen KLH conjugated synthetic peptide derived from human FOX C2

Epitope Specificity 101-200/501

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 **SIMILARITY DISEASE**

Important Note

Nucleus. Contains 1 fork-head DNA-binding domain.

> Defects in FOXC2 are the cause of lymphedema hereditary type 2 (LMPH2) [MIM:153200]; also known as Meige lymphedema. Hereditary lymphedema is a chronic disabling condition which results in swelling of the extremities due to altered lymphatic flow. Patients with lymphedema suffer from recurrent local infections, and physical impairment. Defects in FOXC2 are a cause of lymphedema-yellow nails (LYYN) [MIM:153300]. LYYN is characterized by yellow, dystrophic, thick and slowly growing nails, associated with

lymphedema and respiratory involvement. Lymphedema occurs more often in the lower limbs. It can appear at birth or later in life. Onset generally follows the onset of ungual abnormalities. Defects in FOXC2 are a cause of lymphedema-distichiasis (LYD) [MIM:153400]. LYD is characterized by primary limb lymphedema usually starting at puberty (but in some cases later or at birth) and associated with distichiasis (double rows of eyelashes, with extra

eyelashes growing from the Meibomian gland orifices).

This product as supplied is intended for research use only, not for use in

human, therapeutic or diagnostic applications.

FOXC2 is a member of forkhead/winged helix transcription factor family, **Background Descriptions** whose members serve as key regulators in embryogenesis and cell

differentiation (3). FOXC2 functions as a key regulator of adipocyte

metabolism by increasing the sensitivity of the beta-adrenergic-cAMP-protein

kinase A (PKA) signaling pathway through alteration of adipocyte PKA holoenzyme composition (4). Increased FOXC2 levels, induced by high fat diet, seem to counteract most of the symptoms associated with obesity (4). FOXC2

expression is also associated with the early stage of chondrogenic

differentiation both in vivo and in vitro (3). FOXC2 haploinsufficiency results in

Lymphedema-distichiasis (LD), an autosomal dominant disorder that classically presents as lymphedema of the limbs, and double rows of

eyelashes (distichiasis) (5). Mutant mice null for FOXC2 show defects in axial

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and cranial skeletogenesis, suggesting a requirement of FOXC2 for skeletal tissue development (3). FOXC2 interacts with FOXC1 in the Notch signaling pathway (1) and in kidney and heart development (2).

Additional Information

Gene ID 2303

Other Names Forkhead box protein C2, Forkhead-related protein FKHL14, Mesenchyme fork

head protein 1, MFH-1 protein, Transcription factor FKH-14, FOXC2, FKHL14,

MFH1

Dilution WB=1:500-2000

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 FOXC2

Synonyms FKHL14, MFH1

Function Transcriptional activator.

Cellular Location Nucleus {ECO:0000255 | PROSITE-ProRule:PRU00089,

ECO:0000269 | PubMed:23878394, ECO:0000269 | PubMed:28179430}

Background

FOXC2 is a member of forkhead/winged helix transcription factor family, whose members serve as key regulators in embryogenesis and cell differentiation (3). FOXC2 functions as a key regulator of adipocyte metabolism by increasing the sensitivity of the beta-adrenergic-cAMP-protein kinase A (PKA) signaling pathway through alteration of adipocyte PKA holoenzyme composition (4). Increased FOXC2 levels, induced by high fat diet, seem to counteract most of the symptoms associated with obesity (4). FOXC2 expression is also associated with the early stage of chondrogenic differentiation both in vivo and in vitro (3). FOXC2 haploinsufficiency results in Lymphedema-distichiasis (LD), an autosomal dominant disorder that classically presents as lymphedema of the limbs, and double rows of eyelashes (distichiasis) (5). Mutant mice null for FOXC2 show defects in axial and cranial skeletogenesis, suggesting a requirement of FOXC2 for skeletal tissue development (3). FOXC2 interacts with FOXC1 in the Notch signaling pathway (1) and in kidney and heart development (2).

Images

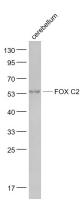
Sample:

Cerebellum (Mouse) Lysate at 40 ug

Primary: Anti- FOX C2 (AP59076) at 1/1000 dilution Secondary: IRDye800CW Goat Anti-Rabbit IgG at 1/20000

dilution

Predicted band size: 53 kD Observed band size:53 kD



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