

Product name:	FoxC1/2 Rabbit Polyclonal Antibody
Cat number:	ABN11076
Conjugate:	Unconjugated
Size:	100µL
Clone:	Polyclonal
Concentration:	1mg/ml
Host:	Rabbit
Isotype:	IgG
Immunogen:	The antiserum was produced against synthesized peptide derived from human FOXC1/2. AA range:151-200
Reactivity:	Human,Mouse,Rat
Applications:	WB 1:500-1:2000,IHC 1:100-1:300,ICC/IF 1:200-1:1000,ELISA 1:20000-1:40000
Molecular Weight:	57kDa
Purification:	Affinity purification
Form:	Liquid
Buffer:	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% New type preservative N.
Storage:	Store at 4°C short term. Aliquot and store at -20°C for 12 months. Avoid freeze/thaw cycles.

Background:

This gene belongs to the forkhead family of transcription factors which is characterized by a distinct DNA-binding forkhead domain. The specific function of this gene has not yet been determined; however, it has been shown to play a role in the regulation of embryonic and ocular development. Mutations in this gene cause various glaucoma phenotypes including primary congenital glaucoma, autosomal dominant iridogoniodysgenesis anomaly, and Axenfeld-Rieger anomaly. [provided by RefSeq, Jul 2008],disease:Defects in FOXC1 are a cause of Axenfeld-Rieger syndrome (ARS) [MIM:601090]; also known as Axenfeld syndrome or Axenfeld anomaly. It is characterized by posterior corneal embryotoxon, prominent Schwalbe line and iris adhesion to the Schwalbe line. Other features may be hypertelorism (wide spacing of the eyes), hypoplasia of the malar bones, congenital absence of some teeth and mental retardation. When associated with tooth anomalies, the disorder is known as Rieger syndrome. Glaucoma is a progressive blinding condition that occurs in approximately half of patients with Axenfeld-Rieger malformations.,disease:Defects in FOXC1 are a cause of Peters anomaly [MIM:604229]. Peters anomaly consists of a central corneal leukoma, absence of the posterior corneal stroma and Descemet membrane, and a variable degree of iris and lenticular attachments to the central aspect of the posterior cornea.,disease:Defects in FOXC1 are the cause of iridogoniodysgenesis anomaly (IGDA) [MIM:601631]. IGDA is an autosomal dominant phenotype characterized by iris hypoplasia, goniodysgenesis, and juvenile glaucoma.,function:Binding of FREAC-3 and FREAC-4 to their cognate sites results in bending of the DNA at an angle of 80-90 degrees.,similarity:Contains 1 fork-head DNA-binding domain.,subunit:Monomer.,tissue specificity:Expressed in all tissues and cell lines examined.,