

<b>Product name:</b>	FoxE3 Rabbit Polyclonal Antibody
<b>Cat number:</b>	ABN11085
<b>Conjugate:</b>	Unconjugated
<b>Size:</b>	100µL
<b>Clone:</b>	Polyclonal
<b>Concentration:</b>	1mg/ml
<b>Host:</b>	Rabbit
<b>Isotype:</b>	IgG
<b>Immunogen:</b>	The antiserum was produced against synthesized peptide derived from human FOXE3. AA range:81-130
<b>Reactivity:</b>	Human,Mouse,Rat
<b>Applications:</b>	WB 1:500-1:2000,IHC 1:100-1:300,ICC/IF 1:50-1:200,ELISA 1:10000-1:20000
<b>Molecular Weight:</b>	33kDa
<b>Purification:</b>	Affinity purification
<b>Form:</b>	Liquid
<b>Buffer:</b>	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% New type preservative N.
<b>Storage:</b>	Store at 4°C short term. Aliquot and store at -20°C for 12 months. Avoid freeze/thaw cycles.

**Background:**

This intronless gene belongs to the forkhead family of transcription factors, which is characterized by a distinct forkhead domain. The protein encoded functions as a lens-specific transcription factor and plays an important role in vertebrate lens formation. Mutations in this gene are associated with anterior segment mesenchymal dysgenesis and congenital primary aphakia. [provided by RefSeq, Dec 2009],disease:Defects in FOXE3 are a cause of anterior segment mesenchymal dysgenesis (ASMD) [MIM:107250]; also known as anterior segment ocular dysgenesis (ASOD). ASMD includes all malformations involving the first (corneal endothelium and trabecular meshwork), second (corneal stroma) and third (iris stroma) mesenchymal waves of neural crest. The ASMD phenotype is characterized by corneal opacities with or without iris adhesions in 100%, cataracts of varying severity in 100% and optic-nerve abnormalities in 20% of affected individuals.,disease:Defects in FOXE3 are a cause of congenital primary aphakia (CPA) [MIM:610256]. Human aphakia is a rare congenital eye disorder in which the lens is missing. It has been histologically subdivided into primary and secondary forms, in accordance with the severity of defects of the ocular tissues, whose development requires the initial presence of a lens. CPA results from an early developmental arrest, around the 4th-5th week of gestation in humans, that prevents the formation of any lens structure and leads to severe secondary ocular defects, including a complete aplasia of the anterior segment of the eye. In contrast, in secondary aphakic eyes, lens induction has occurred, and the lens vesicle has developed to some degree but finally has progressively resorbed perinatally, leading, therefore, to less-severe ocular defects.,similarity:Contains 1 fork-head DNA-binding domain.,