

## PKHD1 Conjugated Antibody

Catalog No: #C48398



Package Size: #C48398-AF350 100ul #C48398-AF405 100ul #C48398-AF488 100ul  
 #C48398-AF555 100ul #C48398-AF594 100ul #C48398-AF647 100ul  
 #C48398-AF680 100ul #C48398-AF750 100ul #C48398-Biotin 100ul

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## Description

Product Name	PKHD1 Conjugated Antibody
Host Species	Mouse
Clonality	Monoclonal
Species Reactivity	Hu
Immunogen Description	Recombinant protein
Conjugates	Biotin AF350 AF405 AF488 AF555 AF594 AF647 AF680 AF750
Other Names	ARPKD antibody FCYT antibody Fibrocystin antibody FPC antibody PKHD1 antibody PKHD1_HUMAN antibody Polycystic kidney and hepatic disease 1 protein antibody Polyductin antibody TIGM1 antibody Tigmin antibody
Accession No.	Swiss-Prot#:P08F94
Uniprot	P08F94
GeneID	5314;
Excitation Emission	AF350: 346nm/442nm AF405: 401nm/421nm AF488: 493nm/519nm AF555: 555nm/565nm AF594: 591nm/614nm AF647: 651nm/667nm AF680: 679nm/702nm AF750: 749nm/775nm
Calculated MW	445 kDa
Formulation	0.01M Sodium Phosphate, 0.25M NaCl, pH 7.6, 5mg/ml Bovine Serum Albumin, 0.02% Sodium Azide
Storage	Store at 4°C in dark for 6 months

## Application Details

## Suggested Dilution:

AF350 conjugated: most applications: 1: 50 - 1: 250  
 AF405 conjugated: most applications: 1: 50 - 1: 250  
 AF488 conjugated: most applications: 1: 50 - 1: 250  
 AF555 conjugated: most applications: 1: 50 - 1: 250  
 AF594 conjugated: most applications: 1: 50 - 1: 250  
 AF647 conjugated: most applications: 1: 50 - 1: 250  
 AF680 conjugated: most applications: 1: 50 - 1: 250  
 AF750 conjugated: most applications: 1: 50 - 1: 250

## Background

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May be required for correct bipolar cell division through the regulation of centrosome duplication and mitotic spindle assembly. May be a receptor protein that acts in collecting-duct and biliary differentiation. Defects in PKHD1 are the cause of polycystic kidney disease autosomal recessive (ARPKD). ARPKD is a severe form of polycystic kidney disease affecting the kidneys and the hepatic biliary tract. The clinical spectrum is widely variable, with most cases presenting during infancy. The fetal phenotypic features classically include enlarged and echogenic kidneys, as well as oligohydramnios secondary to a poor urine output. Up to 50% of the affected neonates die shortly after birth, as a result of severe pulmonary hypoplasia and secondary respiratory insufficiency. In the subset that survives the perinatal period, morbidity and mortality are mainly related to severe systemic hypertension, renal insufficiency, and portal hypertension due to portal-tract fibrosis.

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Note: This product is for in vitro research use only