UGT1A1 Rabbit mAb

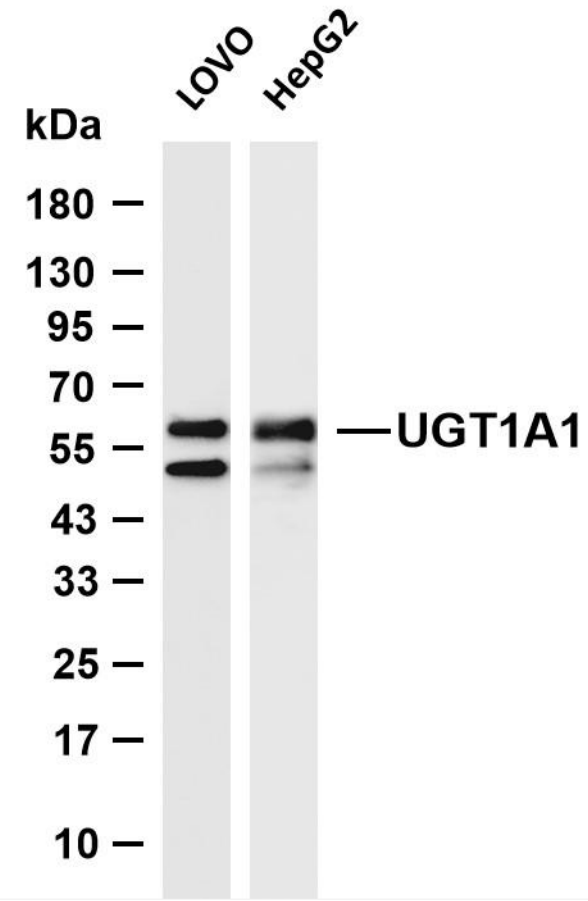
Catalog No	YP-rAb-17216
Isotype	IgG
Reactivity	Human
Applications	WB,IHC,IF,ELISA
Gene Name	UGT1A1 GNT1 UGT1
Protein Name	UDP-glucuronosyltransferase 1A1
Purification Process	Protein A
Specificity	Endogenous
Formulation	PBS, 50% glycerol, 0.05% Proclin 300, 0.05%BSA
Source	Monoclonal, Rabbit,IgG
Dilution	IHC 1:200-1:1000; WB 1:500-1:2000; IF 1:200-1:1000; ELISA 1:5000-1:20000; Note: For IHC, we suggest antigen retrieval with TE buffer pH 9.0
Concentration	0.5 mg/ml
Purity	≥90%
Storage Stability	-15° C to -25° C/1 year(Do not lower than -25° C)
Synonyms	UD11
Observed Band	60kD
Calculated Molecular Weight	60kD
Cell Pathway	Endoplasmic reticulum membrane ; Single-pass membrane protein . Cytoplasm, perinuclear region .
Tissue Specificity	[Isoform 1]: Expressed in liver, colon and small intestine. Not expressed in kidney, esophagus and skin. ; [Isoform 2]: Expressed in liver, colon, small intestine and kidney. Not expressed in esophagus and skin.
Function	Alternative products:A number of isoforms are produced. The different isozymes have a different N-terminal domain and a common C-terminal domain of 245 residues,Alternative products:A number of isoforms may be produced. Isoforms have a different N-terminal domain and a common C-terminal domain of 245 residues,Catalytic activity:UDP-glucuronate + acceptor = UDP + acceptor beta-D-glucuronoside.,Caution:The sequence shown here is derived from an Ensembl automatic analysis pipeline and should be considered as preliminary data.,Disease:Defects in UGT1A1 are the cause of Crigler-Najjar syndrome type I (CN-I) [MIM:218800]. CN-I patients have severe hyperbilirubinemia and usually die of kernicterus (bilirubin accumulation in the basal ganglia and brainstem nuclei) within the first year of life. CN-I inheritance is autosomal recessive.,Disease:Defects in UGT1A1 are the cause of Crigler-Najjar syndrome

type II (CN-II) [MIM:606785]. CN-II patients have less severe hyperbilirubinemia and usually survive into adulthood without neurologic damage. Phenobarbital, which induces the partially deficient glucuronyl transferase, can diminish the jaundice. CN-II inheritance is autosomal dominant.,Disease:Defects in UGT1A1 are the cause of Gilbert syndrome [MIM:143500]. Gilbert syndrome occurs as a consequence of reduced bilirubin transferase activity and is often detected in young adults with vague nonspecific complaints.,Disease:Defects in UGT1A1 may be a cause of transient familial neonatal hyperbilirubinemia [MIM:237900]. The defects is characterized by excessive concentration of bilirubin in the blood, which may lead to jaundice. Breast milk jaundice is a common problem in nursing infants. It has been ascribed to various breast milk substances, but the component or combination of components that is responsible remains unclear. Defects of UGT1A1 are an underlying cause of the prolonged unconjugated hyperbilirubinemia associated with breast milk. One or more components in the milk may trigger the jaundice in infants who have such mutations. Mutations are identical to those detected in patients with Gilbert syndrome [MIM:143500], a risk factor of neonatal non-physiologic hyperbilirubinemia and a genetic factor in fasting hyperbilirubinemia.,Disease:The Gilbert syndrome is shown to occur as a consequence of reduced bilirubin transferase activity. The disorder, is most often detected in young adults with vague nonspecific complaints. A more severe inheritable deficiency in bilirubin activity exist in Crigler-Najjar (CN): patients with type I (recessive trait) have severe hyperbilirubinemia and usually die of kernicterus (bilirubin accumulation in the basal ganglia and brainstem nuclei) within the first year of life. Patients with type II (dominant trait) have less severe hyperbilirubinemia and usually survive into adulthood without neurologic damage. Phenobarbital, which induces the partially deficient glucuronyl transferase, can diminish the jaundice.,Function:UDPGT is of major importance in the conjugation and subsequent elimination of potentially toxic xenobiotics and endogenous compounds.,Function:UDPGT is of major importance in the conjugation and subsequent elimination of potentially toxic xenobiotics and endogenous compounds. This isoform glucuronidates bilirubin IX-alpha to form both the IX-alpha-C8 and IX-alpha-C12 monoconjugates and diconjugate.,Function:UDPGT is of major importance in the conjugation and subsequent elimination of potentially toxic xenobiotics and endogenous compounds. This isoform has specificity for phenols.,induction:By phenobarbital.,online information:Glucuronosyltransferase entry,polymorphism:Polymorphisms in the UGT1A6 gene define four common haplotypes: UGT1A6*1, UGT1A6*2, UGT1A6*3 and UGT1A6*4. Liver tissue samples that were homozygous for UGT1A6*2 exhibited a high rate of glucuronidation relative to tissues with other genotypes. Biochemical kinetic studies indicate that the UGT1A6*2 allozyme, expressed homozygously, had almost two-fold greater activity toward p-nitrophenol than UGT1A6*1 and when expressed heterozygously (UGT1A6*1/*2) it is associated with low enzyme activity. Common genetic variation in UGT1A6 confers functionally significant differences in biochemical phenotype. This genetic variation might impact clinical efficacy or toxicity of drugs metabolized by UGT1A6.,polymorphism:There are four common allelic UGT1A7 variants which exhibit significant differences in catalytic activity towards 3-, 7-, and 9-hydroxy-benzo(a)pyrene. UGT1A7*3 exhibits a 5.8-fold lower relative Vmax compared to UGT1A7*1, whereas UGT1A7*2 and UGT1A7*4 have a 2.6- and 2.8-fold lower relative Vmax than UGT1A7*1, respectively, suggesting that these mutations confer slow glucuronidation phenotype.,similarity:Belongs to the UDP-glycosyltransferase family.,subunit:Part a large chaperone multiprotein complex comprising CABP1, DNAJB11, HSP90B1, HSPA5, HYOU, PDIA2, PDIA4, PPIB, SDF2L1, UGT1A1 and very small amounts of ERP29, but not, or at very low levels, CALR nor CANX.,tissue specificity:Colon specific.,tissue specificity:Expressed in liver. Not expressed in skin or kidney.,tissue specificity:Expressed in skin, kidney and liver.,tissue specificity:Liver and colon.,tissue specificity:Liver and gastric tissue.,tissue specificity:Liver.,

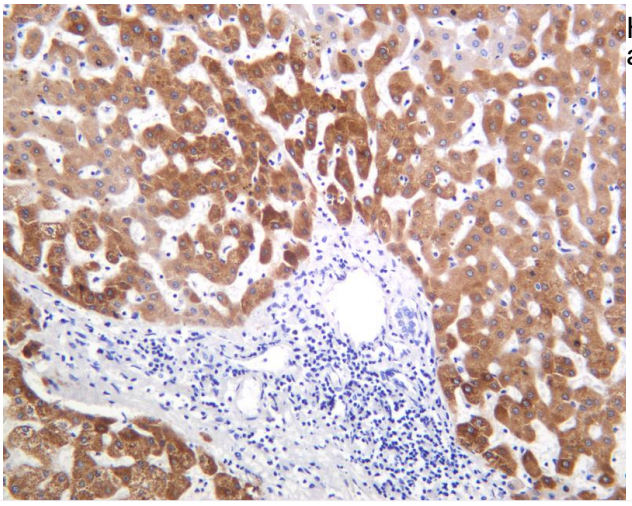
Background

This gene encodes a UDP-glucuronosyltransferase, an enzyme of the glucuronidation pathway that transforms small lipophilic molecules, such as steroids, bilirubin, hormones, and drugs, into water-soluble, excretable metabolites. This gene is part of a complex locus that encodes several UDP-glucuronosyltransferases. The locus includes thirteen unique alternate first exons followed by four common exons. Four of the alternate first exons are considered pseudogenes. Each of the remaining nine 5' exons may be spliced to the four common exons, resulting in nine proteins with different N-termini and identical C-termini. Each first exon encodes the substrate binding site, and is

	regulated by its own promoter. The preferred substrate of this enzyme is bilirubin, although it also has moderate activity with simple phenols, flavones, and C18 steroids. Mutations in this gene result in Crigler-Najjar syndromes types I and II and in Gilbert syndrome. [provided by RefSeq, Jul 2008],
matters needing attention	Avoid repeated freezing and thawing!
Usage suggestions	This product can be used in immunological reaction related experiments. For more information, please consult technical personnel.



Various whole cell lysates were separated by 4-20% SDS-PAGE, and the membrane was blotted with anti-UGT1A1 antibody. The HRP-conjugated Goat anti-Rabbit IgG (H + L) antibody was used to detect the antibody. Lane 1: LOVO Lane 2: HepG2 Predicted band size: 60kDa Observed band size: 60kDa



Human liver was stained with anti-UGT1A1 Rabbit antibody