

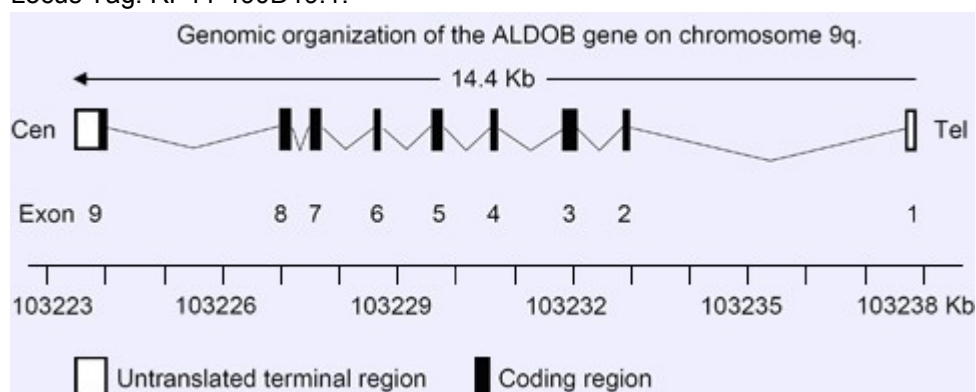
ALDOB (aldolase B, fructose-bisphosphate)

Identity

Other names	ALDB EC 4.1.2.13 OTTHUMP00000021803
HGNC (Hugo)	ALDOB
Location	9q31.1
Location_base_pair	Starts at 103222663 and ends at 103237883 bp from pter (according to hg18-Mar_2006) [Mapping]
Local_order	Telomeric to the PRG-3 (plasticity related gene 3), BAAT (bile acid Coenzyme A: amino acid N-acyltransferase), MRPL50 (mitochondrial ribosomal protein L50) and ZNF189 (zinc finger protein 189) genes. Centrimic to C9orf125 (chromosome 9 open reading frame 125), RNF20 (ring finger protein 20), PP3R2 (protein phosphatase 3 regulatory subunit B, beta isoform) and GRIN3A (glutamate receptor, ionotropic, N-methyl-D-aspartate 3) genes. Those genes are clustered within a genomic region at 102,830K-103,540K bp of chromosome 9q.

DNA/RNA

Note Locus Tag: RP11-490D19.1.



Genomic organization of ALDOB gene on chromosome 9q21.3 - q22.2. Exons are represented by boxes on the diagram.

Description ALDOB encompasses 14,448 base pairs of genomic DNA on the long arm of chromosome 9 in the telomere- to- centromere orientation. (NCBI Entrez Gene, NC-000009.10, 19-Nov-2008.)

The gene consists of 9 exons, with 115, 122, 212, 55, 161, 84, 193, 200, 526 base pairs, respectively.

Transcription ALDOB encodes a 1669 bp mRNA, the coding region is from 126bp to 1220bp of the mRNA.

Exon 1 and the 3' part of exon 9 of the ALDOB gene are non-coding.

Pseudogene None.

Protein

Note Names: aldolase B, Fructose-bisphosphate.
Other Names: aldolase 2, Liver-type aldolase.

Description The 1095 bps open reading frame of ALDOB encodes a 364 amino acids protein with a calculated molecular weight of 39.3kDa.

The functional Aldolase B is a homotetramer. According to the three-dimensional structures of aldolase B homotetramers, the active sites of each monomer locate at the center of the alpha/beta barrels, while the C terminus of the protein is involved in determining the isozyme-specific activity of aldolase. Four isozyme specific regions (ISR) of aldolase B were determined, the first three are expressed by exon 3 of the human aldolase gene, the fourth locates at the C-terminal region.

Expression There are three genetically distinct and tissue-specific isozymes of fructose-

biphosphate aldolase (EC-Number 4.1.2.13) class-I in mammals. The A isozyme (aldolase A) is expressed mainly in muscle, the B isozyme (aldolase B) in the liver, kidney, stomach and intestine, and the C isozyme (aldolase C) in the brain, heart and ovary. Aldolase B is the only expressed isoform in highly differentiated hepatocytes. The high level of gene expression results from cooperation between a liver-specific promoter and an intronic enhancer.

Localisation	Cytoplasm and perinuclear membrane of hepatocytes.
Function	All the three aldolase isozymes catalyze the reversible cleavage of fructose-1,6-(bis) phosphate (FBP) or fructose 1-phosphate (F1P) to dihydroxyacetone phosphate and either glyceraldehyde- 3-phosphate or glyceraldehyde, respectively. Aldolase B has equal activity toward substrate F1P and FBP, and is involved in the two opposite metabolic pathways, glycolysis and gluconeogenesis. Aldolase isozymes utilize covalent catalysis through a Schiff base in the active site of the enzyme, but exhibit distinct catalytic properties. The Schiff-base lysine is located in the central cavity of the barrel. The enzymatic active sites at aldolase B protein sequence are: Arg 55 and Lys146 for binding of c-1-phosphate group of the substrate; Lys 299, the Schiff base for dihydroxyacetone-p; Try 363 for enzymatic activity toward fructose 1,6- biphosphate site; Asp33, Glu187 and Lys229 residues for catalytic function.
Homology	<p>The three human aldolase isozymes are similar in sequence with 66% identity between human A and B, 68% identity between B and C, and 78% identity between A and C. Aldolase molecules have seven major conserved common sequence (CCS-1 to -7), that are the constituents forming a basal alpha/beta barrel structure, are conserved in all aldolase molecules beyond isozyme groups. All isozymes have strictly conserved residues in the active site consisting of Asp33, Arg42, Lys107, Lys146, Glu187, Ser271, Arg303, and Lys229.</p> <p>The identities of aldolase B between human and other animal species are shown bellow. Protein sequences of the mammalian aldolase B are highly conserved.</p> <p>[Pongo abelii] aldolase B, fructose-bisphosphate (364/364, 100%) [Pan troglodytes] aldolase B, fructose-bisphosphate (363/364, 99% identity) [Rattus norvegicus] Aldob, aldolase B fructose-bisphosphate (349/364, 95% identity) [Mus musculus] Aldob, aldolase B, fructose-bisphosphate (349/364, 95% identity) [Bos taurus] aldolase B, fructose-bisphosphate (334/364, 91% identity) [Canis lupus familiaris] aldolase B, fructose-bisphosphate (334/364, 91% identity) [Ovis aries] aldolase B (333/364, 91% identity) [Macaca mulatta] aldolase B (333/364, 91% identity) [Gallus gallus] aldolase B, fructose-bisphosphate (294/364, 80% identity) [Danio rerio] aldob, aldolase b, fructose-bisphosphate (277/364, 76% identity) [Salmo salar] aldolase B (266/365, 72% identity)</p>

Mutations

Mutations	Predicted effects	References
Missense mutations		
c.2T>C	p.M1T	ALI <i>et al.</i> 1993
c.170G>C	p.R57P	DAVIT-SPRAUL <i>et al.</i> 2008
c.343T>C	p.C135R	BROOKS and TOLAN 1994
c.441T>C	p.W148R	ALI and COX 1995
c.448G>C	p.A150P	CROSS <i>et al.</i> 1988
c.524C>A	p.A175D	CROSS <i>et al.</i> 1988
c.532T>C	p.C178R	SANTER <i>et al.</i> 2005
c.770T>C	p.L257P	ALI <i>et al.</i> 1994a
c.839C>A	p.A280P	DAVIT-SPRAUL <i>et al.</i> 2008
c.851T>C	p.L284P	SANTER <i>et al.</i> 2005
c.910C>T	p.R304W	SANTAMARIA <i>et al.</i> 1996
c.932T>C	p.L311P	DAVIT-SPRAUL <i>et al.</i> 2008
c.1005C>G	p.N335K	CROSS <i>et al.</i> 1990b
c.1013C>T	p.A338V	COX 1994
g.10236G>T	p.V222F	ESPOSITO <i>et al.</i> 2004
g.10258T>C	p.L229P	ESPOSITO <i>et al.</i> 2004
g.6846T>C	p.I74T	ESPOSITO <i>et al.</i> 2004
Nonsense mutations		
c.10C>T	p.R4X	ALI <i>et al.</i> 1994b
c.178C>T	p.R60X	ALI <i>et al.</i> 1994b
c.444G>A	p.W148X	DAVIT-SPRAUL <i>et al.</i> 2008
c.522C>G	p.Y174X	GRUCHOTA <i>et al.</i> 2006
c.612T>A	p.Y204X	ALI <i>et al.</i> 1993
c.612T>G	p.Y204X	SANTER <i>et al.</i> 2005
c.720C>A	p.C240X	KAJIHARA <i>et al.</i> 1990
g.8187C>T	p.Q111X	ESPOSITO <i>et al.</i> 2004
Deletions		
c.62delA	p.Q21del	SANTAMARIA <i>et al.</i> 1996
c.146delT	p.V49GfsX27	DAVIT-SPRAUL <i>et al.</i> 2008
c.250delC	frameshift	GRUCHOTA <i>et al.</i> 2006
c.324G>A	p.K108K, 4aa eliminated in exon 3	SANCHEZ-GUTIERREZ <i>et al.</i> 2002
c.345-372del28bp	frameshift	SANTER <i>et al.</i> 2005
c.357delAAAC	p.N120KfsX30	DAZZO and TOLAN 1990
c.360-363delCAAA	p.N120K121del	DAZZO and TOLAN 1990
c.479-482delAACA	frameshift	CHI <i>et al.</i> 2007
c.841-842delAC	frameshift	SANTER <i>et al.</i> 2005
c.865delC	L289del	CROSS <i>et al.</i> 1990a
c.953-994del42bp	p.A318-332del	DAVIT-SPRAUL <i>et al.</i> 2008
c.1044-1049delTTCTGinsACACT	frameshift	SANTER <i>et al.</i> 2005
g.7516-9165del	p.L109-S160del	CROSS and COX 1990
g.9912-10836del	p.N181-G267del	CROSS and COX 1990
IVS2-1GdelGGTA	p.G38T39del	CROSS and COX 1990
IVS8-1GdelGGCTAACinsG	p.A334N335del	BROOKS <i>et al.</i> 1991
Insertions		
c.313-314ins12bp	wrong protein conformation	GRUCHOTA <i>et al.</i> 2006
c.689-690insTGCTAA	p.K230MfsX136	DAVIT-SPRAUL <i>et al.</i> 2008
Mutations in the splicing region		
c.112+1G>A	deduced splicing defect	DAVIT-SPRAUL <i>et al.</i> 2008
c.113-1G>A	loss splice site	SANTER <i>et al.</i> 2005
c.325-1G>C	deduced splicing defect	ESPOSITO <i>et al.</i> 2004
c.625-1G>A	deduced splicing defect	ALI <i>et al.</i> 1994
c.625-2A>G	deduced splicing defect	ESPOSITO <i>et al.</i> 2004
c.799+2T>A	loss splice site	SANTER <i>et al.</i> 2005
g.922-925delgGTA	splicing defect	GRUCHOTA <i>et al.</i> 2006
IVS5+1G>C	splicing mutation	ALI <i>et al.</i> 1996

Types of mutation related to Hereditary fructose intolerance (HFI).
 c. means cDNA coding region mutations, g. means genome mutations and p. refers to protein change after nucleotide mutation. IVS (intervening sequence) refers to introns.

Germinal Recessively inherited mutations in the ALDOB gene, that caused catalytic deficiency of aldolase B, have been found in hereditary fructose intolerance (HFI). Many types of mutation in human ALDOB gene were reported, including missense mutations, nonsense mutations, deletions, insertions and mutation at the splicing regions (list in the diagram above). The mutations bring about reduced enzyme activity and affect structural stability. Mutants that retained tetrameric structure but with altered kinetic properties would reduce its catalytic activity. Mutants with homotetramers dissociated into subunits would have more severe impaired enzymatic activity. The three most common sites are: p.A150P (64%), p.A175D (16%) and p.N335K (5%).

Somatic Human cancer result from the genetic mutation of ALDOB was not reported so far.

Implicated in

Entity	Hereditary fructose intolerance (HFI)
Disease	An autosomal recessive disease that results in the inability to metabolize fructose and related sugars. When fructose, sucrose, or sorbitol was taken from the diet, affected patients suffer from vomiting, abdominal pain, hypoglycemia. Continued ingestion of noxious sugars leads to hepatic and renal injury, which eventually leads to liver cirrhosis and growth retardation.
Prognosis	Complete exclusion of fructose, sucrose, and sorbitol from the diet results in dramatic recovery if liver and kidney damage is not irreversible.
Oncogenesis	Not found
Entity	Hepatocellular cellular carcinoma (HCC)
Note	Aldolase B is the only expressed isoenzymes of aldolase in highly differentiated hepatocytes. The mRNA of aldolase B was downexpressed in HCC patients detected by northern blot or RT-PCR, and it was also undetectable or expressed at very low levels in the hepatocellular carcinoma (HA22T, SKHep, HCC36, PLC/PLZ/5 and Hep3B) and hepatoblastoma (HepG2) cell lines.
Disease	Hepatocellular carcinoma (HCC) is an aggressive malignancy with a poor prognosis. Down-regulation of ALDOB was detected in patients of HCC and is associated with advanced disease, ETR and poor prognosis. A dramatic down-regulation of ALDOB was found in 116 of 203 HCCs (57%), while 43% of HCCs maintained the expression. The ALDOB down-regulation correlated with high-grade (grade II-IV) HCC ($p < 0.0001$), portal vein invasion ((stage IIIB-IV) ($p < 1 \times 10^{-6}$), early tumor recurrence (ETR) ($p < 0.001$)) and a lower 5-year survival ($p = 0.000001$).
Prognosis	In stage II HCC which had no vascular invasion, the ALDOB down-regulation was associated with ETR ($p < 0.05$) and a lower 5-year survival ($p = 0.015$), and ALDOB down-regulation in stage II HCC is a predictive marker of ETR and an unfavorable outcome.

External links

	Nomenclature
HGNC (Hugo)	ALDOB 417
Entrez_Gene (NCBI)	ALDOB 229 aldolase B, fructose-bisphosphate
	Cards
Atlas	ALDOBID44287ch9q31
GeneCards (Weizmann)	ALDOB
Ensembl (Hinxton)	ENSG00000136872 [Gene_View] ALDOB [Vega]
AceView (NCBI)	ALDOB
GenAtlas (Paris)	ALDOB
euGene (Indiana)	229
SOURCE (Stanford)	NM_000035
	Genomic and cartography
GoldenPath (UCSC)	ALDOB - 9q31.1 chr9:103222663-103237883 - 9q21.3-q22.2 [Description] (hg18-Mar_2006)

[Ensembl](#) [ALDOB - 9q21.3-q22.2 \[CytoView\]](#)
 Mapping of homologs : [NCBI](#)
[OMIM](#) [229600](#)

Gene and transcription

Gene : [Genbank](#) [AK026411](#) [AK290795](#) [AV645373](#) [AV656265](#) [AW242415](#)
 (Entrez)
 Reference sequence (RefSeq transcript) : [SRS](#) [NM_000035](#)
 Reference transcript : [Entrez](#) [NM_000035](#)
 RefSeq genomic : [SRS](#) [AC_000052](#) [AC_000141](#) [NC_000009](#) [NT_008470](#) [NW_001839236](#) [NW_924539](#)
 RefSeq genomic : [Entrez](#) [AC_000052](#) [AC_000141](#) [NC_000009](#) [NT_008470](#) [NW_001839236](#) [NW_924539](#)
 Consensus coding sequences : [CCDS](#) [ALDOB](#)
[NCBI](#)
 Cluster EST : [Unigene](#) [Hs.530274](#) [SRS] [Hs.530274](#) [NCBI]
 Alternative Splicing : [Fast-db \(Paris\)](#) [12123](#)

Protein : pattern, domain, 3D structure

Protein : [UniProt/SwissProt](#) [P05062](#) (SRS) [P05062](#) (Expasy) [P05062](#) (Uniprot)
 With graphics : [InterPro](#) [P05062](#)
 Splice isoforms : [VarSplice](#) [FASTA](#) [P05062](#)(VarSplice FASTA)
 Domains pattern : [Prosites \(SRS\)](#) [ALDOLASE_CLASS_I](#) (PS00158)
 Domain pattern : [Prosites \(Expasy\)](#) [ALDOLASE_CLASS_I](#) (PS00158)
 Domains : [Interpro \(SRS\)](#) [Aldolase_I](#) [Aldolase_TIM](#)
 Domains : [Interpro \(EBI\)](#) [Aldolase_I](#) [Aldolase_TIM](#)
 Related proteins : [CluSTr](#) [P05062](#)
 Domain families : [Pfam SRS](#) [Glycolytic](#) (PF00274)
 Domain families : [Pfam Sanger](#) [Glycolytic](#) (PF00274)
 Domain families : [Pfam NCBI](#) [pfam00274](#)
 Domain structure : [Prodom \(Prabi Lyon\)](#) [Aldolase_I](#) (PD001128)
[Blocks \(Seattle\)](#) [P05062](#)
 Crystal structure of protein : [PDB SRS](#) [1QO5](#) [1XDL](#) [1XDM](#)
 Crystal structure of protein : [PDBSum](#) [1QO5](#) [1XDL](#) [1XDM](#)
 Crystal structure of protein : [IMB](#) [1QO5](#) [1XDL](#) [1XDM](#)
 Crystal structure of protein : [PDB RSDB](#) [1QO5](#) [1XDL](#) [1XDM](#)

HPRD	01972
	Protein Interaction databases
DIP (DOE-UCLA)	P05062
IntAct (EBI)	P05062
	Polymorphism : SNP, mutations, diseases
Single Nucleotide Polymorphism (SNP) :	ALDOB
dbSNP NCBI	
SNP : GeneSNP Utah	ALDOB
SNP : HGBase	ALDOB
Genetic variants :	ALDOB
HAPMAP	
Somatic Mutations in Cancer :	ALDOB
COSMIC	
Mutations and Diseases :	ALDOB
HGMD	
Hereditary diseases :	229600
OMIM	
Hereditary diseases :	229600
GENETests	
Diseases : Genetic Association	ALDOB
	General knowledge
Homologs :	ALDOB
HomoloGene	
Homology/Alignments :	ALDOB
Family Browser	
UCSC	
Phylogenetic Trees/Animal Genes :	ALDOB
TreeFam	
Catalytic activity :	4.1.2.13 [Enzyme-ExPASy] 4.1.2.13 [Enzyme-SRS] 4.1.2.13 [IntEnz-EBI] 4.1.2.13 [BRENDA] 4.1.2.13 [KEGG]
Enzyme	
Chemical/Protein Interactions :	229
	fructose-bisphosphate aldolase activity nucleus nucleolus microtubule organizing center glycolysis NADH oxidation cytoskeletal protein
Keywords Ontology : AmiGO	binding metabolic process lyase activity fructose 1,6-bisphosphate metabolic process positive regulation of ATPase activity centriolar satellite identical protein binding ATPase binding fructose binding vacuolar proton-transporting V-type ATPase complex assembly
Keywords Ontology : EGO-EBI	fructose-bisphosphate aldolase activity nucleus nucleolus microtubule organizing center glycolysis NADH oxidation cytoskeletal protein binding metabolic process lyase activity fructose 1,6-bisphosphate metabolic process positive regulation of ATPase activity centriolar satellite identical protein binding ATPase binding fructose binding vacuolar proton-transporting V-type ATPase complex assembly
Pathways :	Glycolysis Pathway [Genes]
BIOCARTA	
Pathways : KEGG	Glycolysis / Gluconeogenesis Pentose phosphate pathway Fructose and mannose metabolism Carbon fixation
	Other databases
Other database	ProteinAtlas
	Probes
Probes : Imagenes	ALDOB Related clones (RZPD - Berlin)
	Literature

[PubMed](#) [40 Pubmed reference\(s\) in Entrez](#)
[PubGene](#) [ALDOB](#)

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