



# Structural Analysis and Molecular Mechanics of Pxr1 Gene which is Responsible For Zellweger Syndrome

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## Article Info

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## Abstract

Zellweger syndrome is an autosomal recessive disorder caused by mutations in genes that encode peroxins, proteins required for the normal assembly of peroxisomes. It causes a defect in the peroxisomes, which affects the body severely. It affects both males and females and is present at birth. The target gene coding protein in Zellweger syndrome had been identified. Blast P is used for the similarities identification of pxr1 protein and the Functional identification of pxr1 is viewed through AMIGO. Secondary structural analyses of pxr1 protein were carried through tools like, TARGET P, GLOBPROT, and DOMAIN LINKER PREDICTION. From the molecular mechanics study, the electrostatic properties have been identified.

## INTRODUCTION

Zellweger syndrome, also called cerebrohepato-renal syndrome, is a rare congenital disorder, characterized by the reduction or absence of functional peroxisomes in the cells of an individual<sup>1</sup>. It is one of a group of four related diseases called peroxisome biogenesis disorders (PBD), which are part of a larger group of diseases known as the leukodystrophies<sup>2</sup>. These are inherited conditions that damage the white matter of the brain and also affect how the body metabolizes particular substances in the blood and organ tissues. They are caused by defects in any one of 12 genes, termed PEX genes that are required for the normal formation and function of peroxisomes (cell structures that break down toxic substances in the liver, kidneys, eyes, and brain, and synthesize fatty materials that are necessary for cell function). Peroxisomes are required for normal brain development and the function and formation of myelin, the whitish material that coats nerve fibers<sup>3</sup>.

Zellweger syndrome is an inherited peroxisomal metabolic disorder. Peroxisomes are found in almost all body cells and are responsible for many important cellular processes. Zellweger syndrome causes a defect in the peroxisomes, which affects the body severely (Fig – 1). Zellweger syndrome is estimated to occur in 1 of every 50,000 to 100,000 births. It affects both males and females and is present at birth<sup>4</sup>. Peroxisomes are usually abundant in a person's kidney and liver.

Peroxisomes are cell structures that break down toxic substances and synthesize lipids (fatty acids. Oils, and

waxes) that are necessary for cell function. Peroxisomes are required for normal brain development and function and the formation of myelin, the whitish substance that coats nerve fibers<sup>5</sup>. They are also required for normal eye, liver, kidney, and bone functions. Zellweger spectrum disorders result from dysfunctional lipid metabolism, including the over-accumulation of very long-chain fatty acids and phytanic acid, and defects of bile acids and plasmalogens--specialized lipids found in cell membranes and myelin sheaths of nerve fibers. Symptoms of these disorders include an enlarged liver; characteristic facial features such as a high forehead, underdeveloped eyebrow ridges, and wide-set eyes; and neurological abnormalities such as mental retardation and seizures<sup>6</sup>. Infants with Zellweger syndrome also lack muscle tone, sometimes to the point of being unable to move, and may not be able to suck or swallow. Some babies will be born with glaucoma, retinal degeneration, and impaired hearing. Jaundice and gastrointestinal bleeding also may occur.

## AIM AND OBJECTIVES

To find out the mutational genes involved in Zellweger syndrome. To predict 3D Structure of the Protein. To visualize molecular mechanics of the protein.

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## METHODOLOGY

Zellweger syndrome is the most severe of three PBD disorders that have overlapping features; the other disorders being neonatal adrenoleukodystrophy and Infantile Refsum Disease, the mildest disorder<sup>7</sup>. The National Center for Biotechnology Information (NCBI) provides a comprehensive website for biologists that includes biology-related databases, and tools for viewing and analyzing the data inherent in the databases. Basic Local Alignment Search Tool, or BLAST, is an algorithm for comparing primary biological sequence information, such as the amino-acid sequences of different proteins or the nucleotides of DNA sequences. . AmiGO is the official web-based set tools for searching and browsing the Gene Ontology database, which consists of a controlled vocabulary of terms covering biological concepts, and a

large number of genes or gene products whose attributes have been annotated using Gene Ontology terms. ClustalW starts by finding the score of the Multiple alignment between each pair of sequences, using a scoring function that is appropriate for proteins. Target to predict the subcellular location of eukaryotic proteins. GlobPlot is a web service that allows the user to plot the tendency within the query protein for order/globularity and disorder. Domain Linker Prediction-SVM is a domain linker predictor. It is composed of three loops-length dependent SVM predictors of domain linkers (SVM-All, SVM-Long and SVM-Short), and SVM-Joint, which combines the results of SVM-Short and SVM-Long into a single consolidated prediction. DIPOLE MOMENT SERVER used to predict Electrostatic properties of the target proteins.

Figure: 1



## RESULTS AND DISCUSSION

### 1. NCBI-SEQUENCE RETREIVAL

PROTEIN

>gi|223590138|sp|A5DRH5.2|PXR1\_PICGU RecName: Full=Protein PXR1; AltName: Full=PinX1-related protein 1

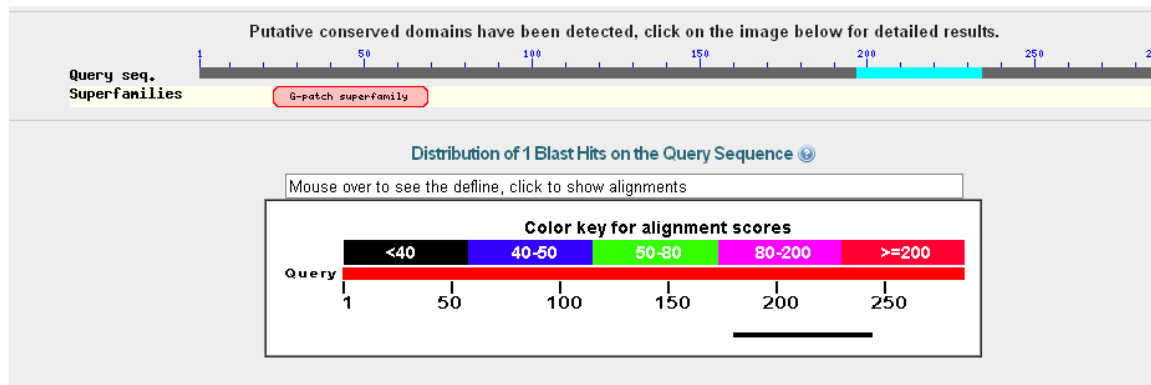
MGLAGTKVKQRFGLDPRNTSWSNDKSRFGHRYLESMGWAPGKGLGLVEHATTTTHVKVSVKDDTVGLGL

AKRSGTDDLETSSGLDDFQRLGRLNGRGREVDLEQKRKDNIIINGKWMHFIKGEVLCSTWDRKSKS  
 HMLKTALEDESEVNFKSSKRRRQSGSEPSRDSTSHAKRMRGDESKKSTRDQSKQERKEKKIKTEKKEKKE  
 KKEKKEKKEKKEKKEKKEKKEKDYGNRASPVPRKHDQISNVGRLSARAKYIKQKRASVMDAKAK  
 NUCLEOTIDE

>gj|359750250:9857-12145 *Torulaspora delbrueckii* CBS 1146 chromosome 7, complete genome  
 TAAAAGAGACAAACGAGGGACAACAACAGAACTGACAGTGATGATCTCACACGTGCCGCAGAGCCCTCG  
 TAGGACGTTAGGCTTGCCTGAGTATGAATAGATGCGAAACCAGTCGAAGAAGGCTCGCTTTTGGAAATAC  
 TTGATGTTGAAGCGGGTGCCTGACTGATCGTAGTCATAGTACTTTGCAACCCACTAACAACCTTCAGATTG  
 GCCAGAAAGTTGTTGATTAGTCGTTGCAGAAGCATGAGTTGAACTTGGATTGAGGATAAAGTCTCACCA  
 TAGTTTATGGTCGAGGCCACAGAGCTTAGGGATTCTGTAGGAGCTATGTGGCTTCCGGGTATCGAAAGGG  
 TGGTAGTTCGATGGGATCTAACTTTTGTAAACGTAGCAGTAGTGCCTTCTGAGGAAGATGTTACCGAAAC  
 ACCTGTGTCAGTTGACGTCTCGGGTAATTGTGATACCTCACTGCTTTGAGCACAAAGCTTCACACTCTACC  
 TGACTTTGAATCCCTATAGATCCACTAGAGGCTGGAGATGGGGTCAACGTTACTGGGCTTCTTGGAAACGA  
 AGGTAGCGGAATTTTGTGTGGAGTCCGTGCTATCAGACCTTGAAGCAGGTTACGAAAGGTGCAGAATCAGA  
 TGATACCAAAGCGTAGAATTAGGTGGTACCAATGTGCAACTAGATAGTCCGGAGCAGCTACGGATAGAA  
 ACTACCACCGTAGAAGTAGTTCCATAACGGATGTATAGCTGGGTAGTACCGATGTAGAAGTACTTGCCA  
 CTA AAAATGTAGAACCAGATGCAATCGAAGTAGAAGGTAAGTGGTTCGAGCTAGATGCAGAACCAGA  
 TAGTGCTGAAGAAGCGGAGCTAGATGGTGTGAAGACGCAGAGCTAGATGGTGCAGATGATATCCATGAA  
 GCTGGTTGCTTTGTGGTGTAGCTGGCACTGAAGTAGTTGAGCTAGATGCAGAGCTAGATGGTGTGAAG  
 ACGCAGAACTAGATGGTGGTGTATGTCCATGAAGCTGGTTGCTTTGTGGTGTAGCTGGCACTGAAGT  
 GGTGAGCTAGATGCAGAGCTAGATGGTGTGAAGACGCAGAGCTAGATGGTGCAGATGATATCCATGAA  
 GCTGGTTGCTTTGTGGTGTAGCTGGCACTGAAGTGGTTCGAGCTAGATGCAGGTTAGATGGTGGCGAAG  
 ACGTAGAACTAGATGGTGGGACAATGTGCAACTAGATGATGTGGAGGATGCAGCGCTAGATCTAGAGTT  
 AGATGAAACCAAAGACTTGGAGCTAGTTGGTTGTAATGTTTTGTAGTAGAACCAGGAACAGATGACGAT  
 GAAGAAGCGGAGCTATGCGCAGAGCTAATAGAAGCGGAGTTAGATGGCACTGACGATATCCAACCTGGCTG  
 GTTCCCTTGTAGTGCTAGCTGGCACTGAAGTAGTTGAGCTAGATGCTGAGCTAGAAGATGTGCAACTAGG  
 ACTGCTTGTGCTTATTGGGACTGATGTGGCAGAGCTAGAAGCTGACGGTACCGAAGAAGTAGATTTAGAT  
 GGTGCTGAAGTCGTCAAAATAGAAACAGAGCTAGCTGGCTCCTTTGTGGTACTAGCTGGCACTGAAGACG  
 TGGAGCTTGTGGAGCTGGTAGAGCTTGTGGAGCTGGTAGAGCTTGTGGAGCTGGTAGAGCACTTAGCATT  
 ATCCAGTACCATAGCGAAAGTCAGCATTAGAATTGCCATTGTTGTCTGCGTTACAACCAGTCATCAAAA  
 TCAAAAAGCAGTTGCACCCCATTTCCAAGTTTCCAGTACTGAGCGGCATCACCTTGCAGATACTCGTATT  
 GAATTTGGAATGATGGCATAGCAACTTTACATTCATCGCCATTATACTTTTGACCATAAACTTGAATTTT  
 CGTCGTGAAATCTGTCGGGTTATCAATTATATAGGTATTCTCATTCTTACCCCAAAGTTGTTTAGTACCC  
 ATTGGACCATTGATGCCAATGATTTTTAAGGAATAGAGATACTTCATATCAATCTGTTGATCACCCCTTGA  
 CATTATTGAAAGTTCATAAGTATTGTCACCGACCCAATCGACTGACAATAGATCCATGTAATATGGCAT  
 GGTGTTGGTATTTTTTGCCTGACATTTGAAGTTCAAATTTGGACACATATTAGTAACTCCAGACACAGCT  
 GACGATGTGCGGCGAACTAACGCTGAATCACCATTATTAGAATAAAAATCAGTCTCGTAAGTATTATCGG  
 AGGCTGCTGAAGCAACCCCTAACAAACATAAGAGCAGATAGGAAAGCAT

The above results show the FASTA format sequence of PXR1.

## 2. BLAST P



Sequences producing significant alignments:						
Accession	Description	Max score	Total score	Query coverage	E value	Max ident
3KZ4_A	Chain A, Crystal Structure Of The Rotavirus Double Layered	33.5	33.5	22%	0.16	34%

The above result shows the similarity search of PXR1.

### 3. FUNCTIONAL IDENTIFICATION-AMIGO

rel ↓	Symbol, full name	Species
<input type="checkbox"/>	<b>PXR1</b> Essential protein involved in rRNA and snoRNA maturation	10 associations <b>gene</b> from <i>Saccharomyces cerevisiae</i>
<input type="checkbox"/>	<b>Pex5</b> ★ peroxisomal biogenesis factor 5 Query matches synonym <b>Pxr1</b> <a href="#">View gene product details</a>	32 associations <b>protein</b> from <i>Mus musculus</i>
<input type="checkbox"/>	<b>PEX5</b> Peroxisomal targeting signal 1 receptor Query matches synonym <b>PXR1</b>	14 associations <b>protein</b> from <i>Bos taurus</i>
<input type="checkbox"/>	<b>PEX5</b> ★ Peroxisomal targeting signal 1 receptor Query matches synonym <b>PXR1</b>	15 associations <b>protein</b> from <i>Homo sapiens</i>
<input type="checkbox"/>	<b>PEX5</b> Peroxisomal targeting signal 1 receptor Query matches synonym <b>PXR1</b>	7 associations <b>protein</b> from <i>Cavia porcellus</i>
<input type="checkbox"/>	<b>F1:PXR1</b> Guanylate cyclase	6 associations <b>protein</b> from <i>Canis lupus familiaris</i>

The above result shows the functional identification of PXR1.

### 4. PHYLOGENETIC ANALYSIS

CLUSTAL 2.1 multiple sequence alignment

```

sp|O70525|PEX5_CAVPO      MAMRELVEGECGGANPLMKLAGHFTQDKALRQEGLRPGWPWPAGAPASETV 50
sp|P50542|PEX5_HUMAN     MAMRELVEAECGGANPLMKLAGHFTQDKALRQEGLRPGWPWPAGAPASEAA 50
sp|O09012-2|PEX5_MOUSE  MRELVEGECGGANPLMKLATHFTQDKALRQEGLRPGWPWPAGASAAETV 50
gi|285812468|tpg|DAA08368.1|MGLAATRQKRFGLDP--RNTAWSNDTSRFHQFLEKFGWKPGMGLGLSP 48
      *.:  : *.* :. :. :. *  ***  .:

sp|O70525|PEX5_CAVPO      SKPLGVASEDELVAEFLQDQNAPLVSRAPQTFKMDLLAEMQEIEQSNFR 100
sp|P50542|PEX5_HUMAN     SKPLGVASEDELVAEFLQDQNAPLVSRAPQTFKMDLLAEMQQIEQSNFR 100
sp|O09012-2|PEX5_MOUSE  SKPLGVGTEDELVSEFLQDQNA TLVSRAPQTFKMDLLAEMQEIEQSNFR 100
gi|285812468|tpg|DAA08368.1|  MN-----SNTSHIKVSIKDDNVGLGAKLKRKDKKDEFD----- 81
      :  .: : **.* *.: : * *.:

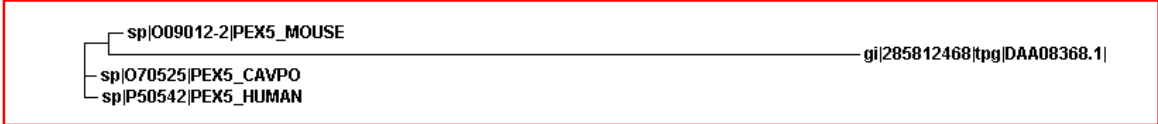
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sp|P50542|PEX5_HUMAN     QAPQRAPGVADLALSENWAQEFLAAGDAVDVTQDYNETDWSQEFISEVD 150
sp|O09012-2|PEX5_MOUSE  QAPQRAPGVADLALSENWAQEFLAAGDAVDVAQDYNETDWSQEFIAEVD 150
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```

```

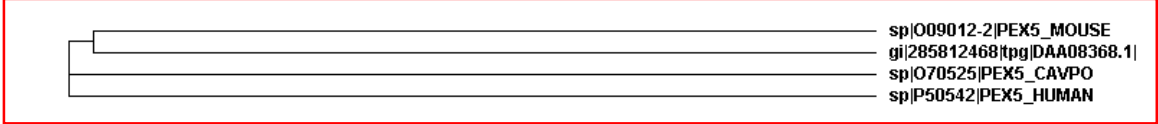
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sp|P50542|PEX5_HUMAN      PLSVSPARWAEELYEQSEKWLWLGEPEGTAT-DRWYDEYHPEEDLQHTAS 199
sp|O09012-2|PEX5_MOUSE    PLSVSPARWAEELYEQSEKWLWLDQEGSSTADRWYDEYHPEEDLQHTAS 200
gi|285812468|tpg|DAA08368.1|  RLNGKESKISEELDTRKQKIIDG-----KWGIHFVKGEVLAETWD 137
                                * . . . . .
                                * . . . . *
                                * . . . . *
                                * . . . . *
                                * . . . . *
sp|O70525|PEX5_CAVPO      DFVAKVDDPKLANSEFLKFVRQIGEGQVSLVAGSGRAQAEQWAAEFIQ 250
sp|P50542|PEX5_HUMAN      DFVAKVDDPKLANSEFLKFVRQIGEGQVSLVAGSGRAQAEQWAAEFIQ 249
sp|O09012-2|PEX5_MOUSE    DFVSKVDDPKLANSE----- 215
gi|285812468|tpg|DAA08368.1|  PKTHKLRN----- 145
                                * . . . .
                                * . . . .
sp|O70525|PEX5_CAVPO      QQGTSDAWVDQFTRPVN-TSALDMEFERAKSAIESDVDFWDLQAELEEM 299
sp|P50542|PEX5_HUMAN      QQGTSDAWVDQFTRPVN-TSALDMEFERAKSAIESDVDFWDLQAELEEM 298
sp|O09012-2|PEX5_MOUSE    --GTSEAWVDQFTRPGNKIAALQVEFERAKSAIESDVDFWDLQAELEEM 263
gi|285812468|tpg|DAA08368.1|  -----YSNAK-----KRKREGDDS 159
                                . . . . .
                                * . . . .
                                * . . . .
                                * . . . .
                                * . . . .
sp|O70525|PEX5_CAVPO      AKRDAEAHPWLSYDDLTASADYDGYQFEENPLRDHPQPFEEGLLRLEE 349
sp|P50542|PEX5_HUMAN      AKRDAEAHPWLSYDDLTASADYDGYQFEENPLRDHPQPFEEGLLRLEE 348
sp|O09012-2|PEX5_MOUSE    AKRDAEAHPWLSYDDLTASADYDGYQFEENPLRDHPQPFEEGLLRLEE 313
gi|285812468|tpg|DAA08368.1|  EDEDDDDK----EDKDSDKKKHKKHKKHKKDKKKDKKDKKEHKKHKKEE 204
                                * . . . .
                                * . . . . *
                                * . . . . *
                                * . . . . *
                                * . . . . *
sp|O70525|PEX5_CAVPO      GDLPNAVLLFEAAVQQDPKHMEAWQYLGTTQAENEQELLAISALRRCLEL 399
sp|P50542|PEX5_HUMAN      GDLPNAVLLFEAAVQQDPKHMEAWQYLGTTQAENEQELLAISALRRCLEL 398
sp|O09012-2|PEX5_MOUSE    GDLPNAVLLFEAAVQQDPKHMEAWQYLGTTQAENEQELLAISALRRCLEL 363
gi|285812468|tpg|DAA08368.1|  KRLK-----KEKRAEKTKETKTSKL 225
                                *
                                * . . . .
                                * . . . .
                                * . . . .
                                * . . . .
sp|O70525|PEX5_CAVPO      KPDNRTALMALAVSFTNESLQRQACETLRDWLRCTPAYAHLVTPAEEGAG 449
sp|P50542|PEX5_HUMAN      KPDNQTALMALAVSFTNESLQRQACETLRDWLRCTPAYAHLVTPAEEGAG 448
sp|O09012-2|PEX5_MOUSE    KPDNRTALMALAVSFTNESLQRQACETLRDWLRCTPAYAHLVTPAEEGAG 413
gi|285812468|tpg|DAA08368.1|  KSSESASNIPDAVNTR-----LSVRSKWIKQK----- 252
                                * . . . . *
                                * . . . .
                                * . . . .
                                * . . . .
                                * . . . .
sp|O70525|PEX5_CAVPO      GAGLGSSKRILGSLSDSLFLEVKELFLAAVRLDPTSIDPDVQCGLGVLF 499
sp|P50542|PEX5_HUMAN      GAGLGPSKRILGSLSDSLFLEVKELFLAAVRLDPTSIDPDVQCGLGVLF 498
sp|O09012-2|PEX5_MOUSE    GA--GPSKRILGSLSDSLFLEVKDLFLAAVRLDPTSIDPDVQCGLGVLF 461
gi|285812468|tpg|DAA08368.1|  -----RAALMDSKALNEIFMITND----- 271
                                * . . . .
                                * . . . .
                                * . . . .
                                * . . . .
                                * . . . .
sp|O70525|PEX5_CAVPO      NLSGEYDKAVDCFTAALSVRPNDYLLWNKLGATLANGNQSEEAVAAAYRRA 549
sp|P50542|PEX5_HUMAN      NLSGEYDKAVDCFTAALSVRPNDYLLWNKLGATLANGNQSEEAVAAAYRRA 548
sp|O09012-2|PEX5_MOUSE    NLSGEYDKAVDCFTAALSVRPNDYLLWNKLGATLANGNQSEEAVAAAYRRA 511
gi|285812468|tpg|DAA08368.1|  -----
                                * . . . .
                                * . . . .
                                * . . . .
                                * . . . .
                                * . . . .
sp|O70525|PEX5_CAVPO      LELQPGYIRSRYNLIGISCINLGAHREAVEHFLEALNMQRKSRGPRGEGGA 599
sp|P50542|PEX5_HUMAN      LELQPGYIRSRYNLIGISCINLGAHREAVEHFLEALNMQRKSRGPRGEGGA 598
sp|O09012-2|PEX5_MOUSE    LELQPGYIRSRYNLIGISCINLGAHREAVEHFLEALNMQRKSRGPRGEGGA 561
gi|285812468|tpg|DAA08368.1|  -----
                                * . . . .
                                * . . . .
                                * . . . .
                                * . . . .
                                * . . . .
sp|O70525|PEX5_CAVPO      MSENIWSTLRLALSMLGQSDAYRAADARDLSALLALFGLSQ 640
sp|P50542|PEX5_HUMAN      MSENIWSTLRLALSMLGQSDAYGAADARDLSTLLTMFGLPQ 639
sp|O09012-2|PEX5_MOUSE    MSENIWSTLRLALSMLGQSDAYGAADARDLSALLAMFGLPQ 602
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```

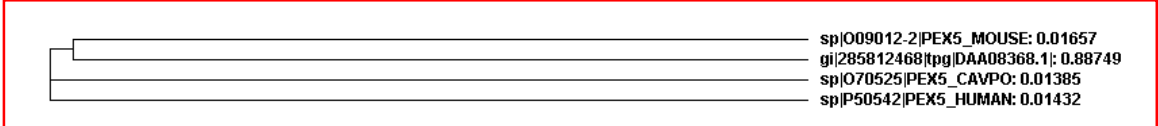
**GUIDE TREE**



**CLADOGRAM TREE**



**DISTANCE TREE**



The above result shows the phylogenetic analysis of PXR1.

**5.STRUCTURAL ANALYSIS**

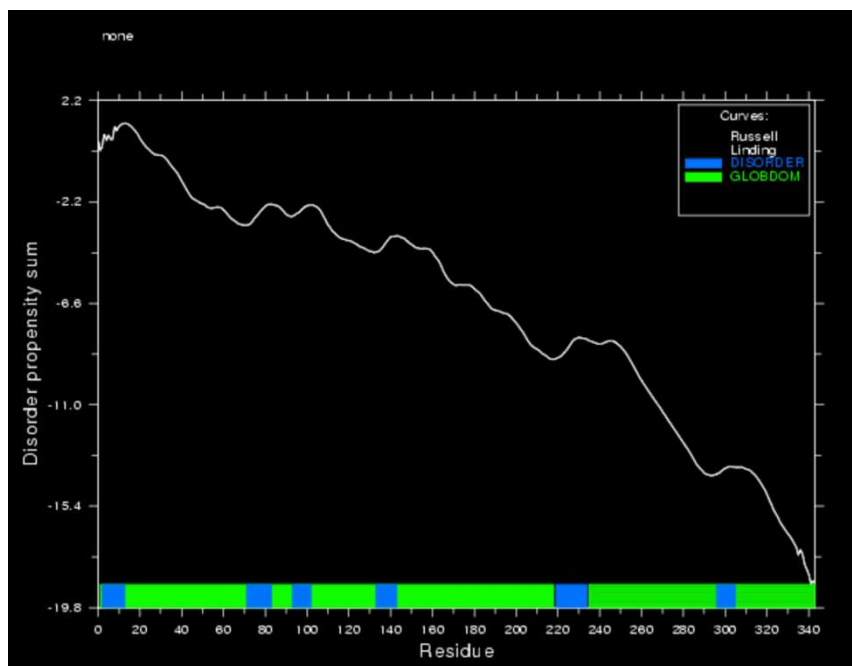
**A.TARGETP**

### targetp v1.1 prediction results #####  
 Number of query sequences: 1  
 Cleavage site predictions not included.  
 Using NON-PLANT networks.

Name	Len	mTP	SP	other	Loc	RC
gi_223590138_sp_A5DR	286	0.440	0.038	0.614	_	5
cutoff		0.000	0.000	0.000		

The above result shows the transmembrane reigns in the PXR1.

**B.GLOBPLOT**



**Disordered by Russell/Linding definition**

```
>none_Disorder 2-13, 71-83, 93-102, 133-143, 219-234, 296-305
gISPADRHPR PICgrecnam efillprtein praltnamef llpinrelat edprteinmg lagtkvkqrf GLDPRNTSWS
NDKsrfgghry leSMGWAPGK GLglvehatt thvkvsvkdd tvglgaklak rSGTDDLETD SSGlddfqri lgrlgrgre
vdealeqkrk dniingkwgm hfikgevlcs twdrkskshM lktaledeSe vnfksskRR QSGSEPSRDS TSHAKRMRGD
eskkstrdqs kqerkekKik tekkekkekK ekkekkekK kkekkekKek kerdyGNRAS PVEPRkhdqi snvgrlsara
kyikqkrasv mDakalneif misk
```

**Potential globular domains (GlobDoms) by Russell/Linding definition**

```
>none_GlobDoms 1-218, 235-344
GISPADRHPR PICGRECNAM EFLLRTEIN PRALTNAMF LLPINRELAT EDPRTINMG LAGTKVKQRF GLDPRNTSWS
NDKSRFGHRY LESMGWAPGK GLGLVEHATT THVKVSVKDD TVGLGAKLAK RSGTDDLETD SSGLDLDFQRI LGRINGRGRE
VDEALBQKRK DNIINGKWGM HFIKGEVLCs TWDRKSKSHM LKTALEDESE VNFKSSKRRr qsgsepsrds tshakMRGD
ESKKSTRDQS KQERKEKKIK TEKKEKKEK EKKEKKEK KKEKKEKKEK KERDYGNRAS PVEPRKHDQI SNVGRLSARA
KYIKQKRASV MDAKALNEIF MISK
```

**JOB-ID:** none\_121427BWYRtgoLCBQAADgiisEAAAAL  
**Parameters:** propensities=Russell/Linding smooth=10 dy/dx\_smooth=10  
**Disorder frames:** peak-frame=5 join-frame=4  
**Globularity frames:** peak-frame=74 join-frame=15  
**Name:** None  
**Description:** None  
**Plot title/ID:** None  
**Sequence length:** 344  
**Download Results** [smoothed raw dydx](#)

The above result shows the disorders regions of PXR1 protein.

**C.DOMAIN LINKER PREDICTION**

**Candidate Region:**

SVM-All

Rank	Peak Value	Peak Position	Region	Sequence
1	2.009	165	148 - 247	EDESEVNFKSSKRRRQSGSEPSRDSTSHAKRMRGDE SKKSTRDQSKQERKEKKIKTEKKEKKEKKEKKEKKEKKE KKEKKEKKEKKEKDYGNRASVPEPR
2	0.305	75	73 - 79	RSGTDDL

SVM-Long

Rank	Peak Value	Peak Position	Region	Sequence
1	1.406	245	205 - 247	KKEKKEKKEKKEKKEKKEKKEKKEKKEKKEKDYGNR ASPVEPR
2	1.192	167	156 - 190	KSSKRRRQSGSEPSRDSTSHAKRMRGDESKKSTRD

SVM-Short

Rank	Peak Value	Peak Position	Region	Sequence
1	1.494	191	178 - 200	RMRGDESKKSTRDQSKQERKEK
2	1.148	110	107 - 112	LEQKRK





- Campodarve I, Zellweger C, Irfan A, Drexler B, Mueller C. Patients with Acute Coronary Syndrome and Normal High-sensitivity Troponin. Department of Internal Medicine, Division of Cardiology, University Hospital, Basel, Switzerland; Paris Descartes University, Cardiology Department, Cochin Hospital, APHP, Paris, France.
- Ezgu E, Eminoglu T, Okur I, Gunduz M, Tumer L, Hasanoglu A, Dalgic B. An infantile case of Zellweger syndrome presented with Kabuki-like phenotype. Gazi University Faculty of Medicine, Department of Pediatric Metabolism, Division of Genetics and Molecular Diagnosis, Ankara, Turkey. fezgu@gazi.edu.tr
  - Thoms S, Grønberg S, Rabenau J, Ohlenbusch A, Rosewich H, Gärtner J. Characterization of two common 5' polymorphisms in PEX1 and correlation to survival in PEX1 peroxisome biogenesis disorder patients. Department of Pediatrics and Pediatric Neurology, University Medical Center, University of Göttingen, Robert Koch Str, 40, 37099 Göttingen, Germany. sven.thoms@med.uni-goettingen.de
  - Cho SY, Chang YP, Park JY, Park HD, Sohn YB, Park SW, Kim SH, Ji S, Kim SJ, Choi EW, Kim CH, Ko AR, Paik KH, Jin DK. Two novel PEX1 mutations in a patient with Zellweger syndrome: the first Korean case confirmed by biochemical, and molecular evidence. Department of Pediatrics, Samsung Medical Center, Sungkyunkwan University School of Medicine, 50 Irwon-dong, Gangnam-gu, Seoul, Korea.
  - Nakayama M, Sato H, Okuda T, Fujisawa N, Kono N, Arai H, Suzuki E, Umeda M, Ishikawa HO, Matsuno K. *Drosophila* carrying pex3 or pex16 mutations are models of Zellweger syndrome that reflect its symptoms associated with the absence of peroxisomes. Genome and Drug Research Center, Tokyo University of Science, Noda, Chiba, Japan
  - Mast FD, Li J, Virk MK, Hughes SC, Simmonds AJ, Rachubinski RA. A *Drosophila* model for the Zellweger spectrum of peroxisome biogenesis disorders. Department of Cell Biology, University of Alberta, Edmonton, AB T6G 2H7, Canada.
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