Atlas of Genetics and Cytogenetics in Oncology and Haematology

OPEN ACCESS JOURNAL AT INIST-CNRS

Gene Section
Mini Review

BCL7B (B-cell CLL/lymphoma 7B)

Nicola Potter

Department of Molecular Neuroscience, Institute of Neurology, University College London, National Hospital for Neurology and Neurosurgery, London, WC1N 3BG, UK (NP)

Published in Atlas Database: December 2008

Online updated version : http://AtlasGeneticsOncology.org/Genes/BCL7BID779ch7q11.html
DOI: 10.4267/2042/44600

Identity

HGNC (Hugo): BCL7B

Location: 7q11.23

Local order: BCL7B is flanked by BAZ1B and TBL2.

DNA/RNA

Note
Members of the BCL7 family have significant sequence similarity at their N-terminus.

Description

The gene spans 21.339 kb and includes 6 exons.

Transcription

The mRNA transcript is 1690 bp in length.

Protein

Description

The protein product of this gene is 202 aa and alternative splicing generates 3 isoforms.

Expression

Little is known about the expression pattern of this gene in human tissues. However, serial analysis of gene expression (SAGE) data suggests that gene transcripts are present at low levels in the brain and a variety of breast cells. Data is not available for other tissues. BCL7B has also been shown to be under-expressed in paediatric pilocytic astocytomas, suggesting that this gene may play a role in tumour development.

Function

Unknown.

Homology

BCL7B shares 90% sequence homology in the amino-terminal 51 amino acids with human BCL7A from the same gene family.

Implicated in

Paediatric pilocytic astrocytoma

Disease
The loss of BCL7B expression in paediatric pilocytic astrocytoma correlated with a small region of deletion at 7q11.23 in 86% of tumours investigated. The role of this gene in tumour development is still to be determined.

Burkitt lymphoma

Note
The BCL7A protein is encoded by a gene known to be directly involved in a three-way gene translocation in a Burkitt lymphoma cell line.

Williams Syndrome

Disease
BCL7B is located in a chromosomal region commonly deleted in Williams syndrome. The role of BCL7B loss in this syndrome is yet to be established. Furthermore, in rare cases, malignancies have presented in patients with Williams syndrome including non-Hodgkin lymphoma in a 29-year-old woman and an 8 year old boy and an astrocytoma in a 5-year-old child.

References

Semmekrot BA, Rotteveel JJ, Bakker-Niezen SH, Logt F. Occurrence of an astrocytoma in a patient with Williams syndrome. Pediatr Neurosci. 1985-1986;12(3):188-91

Felice PV, Ritter SD, Anto J. Occurrence of non-Hodgkin's lymphoma in Williams syndrome--case report. Angiology. 1994 Feb;45(2):167-70

Jadayel DM, Osborne LR, Coignet LJ, Zani VJ, Tsui LC, Scherer SW, Dyer MJ. The BCL7 gene family: deletion of BCL7B in Williams syndrome. Gene. 1998 Dec 11;224(1-2):35-44

Meng X, Lu X, Li Z, Green ED, Massa H, Trask BJ, Morris CA, Keating MT. Complete physical map of the common deletion region in Williams syndrome and identification and characterization of three novel genes. Hum Genet. 1998 Nov;103(5):590-9

Amenta S, Moschovi M, Sofocleous C, Kostaridou S, Mavrou A, Fryssira H. Non-Hodgkin lymphoma in a child with Williams syndrome. Cancer Genet Cytogenet. 2004 Oct 1;154(1):86-8

Potter N, Karakoula A, Phipps KP, Harkness W, Hayward R, Thompson DN, Jacques TS, Harding B, Thomas DG, Palmer RW, Rees J, Darling J, Warr TJ. Genomic deletions correlate with underexpression of novel candidate genes at six loci in pediatric pilocytic astrocytoma. Neoplasia. 2008 Aug;10(8):757-72

This article should be referenced as such:

Potter N. BCL7B (B-cell CLL/lymphoma 7B). Atlas Genet Cytogenet Oncol Haematol. 2009; 13(11):778-779.