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Review

TAL1 (1p32) deletion in lymphoid malignancies

Lubomir Mitev, Liliya Grahlyova

Military Medical Academy, Department of Cytogenetics and Molecular Biology, Sofia, Bulgaria, cytogen.vma@abv.bg

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Abstract

Review on TAL1 deletion in lymphoid malignancies with data on clinics.

Keywords
TAL1; STIL; SIL; T-cell lymphoblastic leukemia; B-cell lymphoblastic leukemia; Follicular lymphoma; Diffuse large B-cell lymphoma; Multiple myeloma; Plasma cell leukemia; Hodgkin lymphoma; Anaplastic large cell lymphoma; Adult T-cell leukemia/lymphoma

Identity

Note
The deletion of the TAL 1 gene may be due to the deletion of the band 1p32 (del(1)(p32)) or to a submicroscopic interstitial deletion between the TAL1 and STIL genes (Note: STIL is better known as "SIL").
The pathogenesis of the two deletions is different. The 1p32 deletion is probably related to the inactivation of a tumor suppressor gene or genes localized in the band 1p32, while the SIL-TAL1 deletion to deregulation of the TAL1 gene. Del(1)(p32) is found in a variety of B or T lymphoid malignances and the SIL-TAL1 deletion is associated only with T-acute lymphoblastic leukemia (ALL).
Until now 39 cases with del(1)(p32) have been reported: 9 cases with acute lymphoblastic leukemia/lymphoblastic lymphoma, 2 cases with adult T-cell lymphoma/ leukemia (HTLV-1+), 8 cases with follicular lymphoma, 6 cases with diffuse large B-cell lymphoma, 3 cases with anaplastic large cell lymphoma, 2 cases with Hodgkin disease, 7 cases with multiple myeloma and 2 cases with plasma cell leukemia.
The 1p32 deletion can be detected by conventional cytogenetics, and the SIL-TAL1 deletion by fluorescence in situ hybridization, Southern blot analysis and real time PCR.

Clinics and pathology

Deletion of band 1p32

Disease: Acute lymphoblastic leukemia/lymphoblastic lymphoma (ALL)

Epidemiology

The deletion is found in 9 cases ALL - 0.08% of all ALL cases with an abnormal karyotype: 3 cases with T-ALL (Schoch et al, 1996; Jarosova et al, 2016), 5 cases with B-ALL (Kristofferson et al, 1985; Pui et al, 1990; Pui et al, 1992; Ivanov Ofverholm et al, 2016) and 1 case without data on the cell phenotype (Prigogina et al, 1988).
The patients with T-cell phenotype are males. The sex ratio of the cases with B-cell phenotype is M:F=1.5:1. The average age is 22.1 year (range 2-74).

Cytogenetics

In six cases del(1)(q32) are with complex predominantly di- or hyperdiploid karyotypes.
In two cases 1p32 deletion is accompanied with a second anomaly; one case with a T-cell phenotype has t(8;14)(q24;q11) and another with a B-cell phenotype del(12)(p12).
In three cases the deletion is presented as additional deleted chromosome 1.

Disease: Follicular lymphoma (FL)

Epidemiology

The 1p32 deletion is described in 8 cases - 0.53% of all FL cases with an abnormal karyotype) (Yunis et al, 1984; Gaunt et al, 1986; Offit et al, 1989; Goyns et al, 1993; Wiodarska et al, 1994; Horsman et al, 2001; Aamot et al, 2007).
The sex ratio is M:F=1:1.7 The average age is 51.2 year (range 43-62) (the age of 5 cases is reported).

Cytogenetics

The cases are presented predominantly with highly complex di- or hyperdiploid karyotypes. In 7 cases the 1p32 deletion is associated with t(14;18)(q32;q21).
In four cases the anomaly is accompanied with structural aberration of chromosome 6.

Disease: Diffuse large B-cell lymphoma (DBCL)

Epidemiology

Six cases are reported - 0.42% of all DBCL cases with an abnormal karyotype (Fukuhara et al, 1983; Ebrahim et al 1990; Weisenburger et al, 1996; Ichinohasama et al, 2000; Fan & Rizkalla, 2003; Kaneko et al, 2011).
The sex ratio is M:F=2:1. The average age is 51.5 year (range 36-65) (the age of 4 cases is reported).

Cytogenetics

The reported cases are predominantly with highly complex hyperdiploid or hypertetraploid karyotypes. In four cases the 1p32 deletion is associated with structural anomalies affecting 3q including the BCL6 locus 3q27.
In three cases are found T-lineage anomalies - 14q11 rearrangements in two cases and t(2;5)(p23;q35) in one case and in two the B-lineage anomalies - 14q32 rearrangements.

Disease: Multiple myeloma (MM)

Epidemiology

Seven cases are described - 0.37% of all MM cases with an abnormal karyotype (Lewis & MacKenzie, 1984; Dewalt et al, 1985; Seong et al, 1998; Weinlander et al, 1998; Lioveras et al, 2004; Wu et al, 2007). The sex ratio is M:F=0.8:1. Age is reported in two cases - 67 and 73 years.

Cytogenetics

All cases are with complex karyotypes. Three have hyperdiploid, one hypertetraploid, one pseudodiploid, and two hypodiploid karyotypes. In two cases an additional 1p32 deletion is found.
In four cases the anomaly is associated with other structural rearrangements of chromosome 1.
Only in one case 14q32 rearrangement is described.

Prognosis

1p32 deletion is major negative prognostic factor for progression free survival and overall survival in the cases with MM (Hebraud B et al, 2014).

Disease: Plasma cell leukemia (PCL)

Cytogenetics

Two cases (males; one 70 year old) are reported - 1.32% of all PCL cases with an abnormal karyotype (Lewis & MacKenzie, 1984; Colovic et al, 2008). Both cases have a complex karyotype (one hypodiploid and another hyperdiplod with 14q32 rearrangement).

Disease: Hodgkin disease (HD)

Cytogenetics

Two cases are reported (males, 52 and 76 years old) - 3.8% of all HD cases with an abnormal karyotype (Schlegelberger et al, 1994; Busson-Le Coniat et al, 1996).
One is with an additional 1p32 deletion and another with a highly complex hyperdiploid karyotype carrying additional structural anomalies of chromosome 1.
Both cases are with deletions of 6q.

Disease: Anaplastic large cell lymphoma (ALCL)

Epidemiology

Three cases are reported (two males and one female; 24, 40 and 52 years old) - 2.0% of all ALCL cases with an abnormal karyotype (Ebrahim et al, 1990; Falzetti et al, 1999; Colleoni et al, 2000). Two of them are with a T-cell phenotype.

Cytogenetics

The three cases are with a complex karyotype.
In one t(2;5)(p23;q35) is found and is associated with an additional 1p32 deletion and in another two copies of add(2)(p23).
In all three cases the 1p32 deletion is accompanied with structural anomalies of chromosome 8, two of them with i(8)(q10).

Disease: Adult T-cell lymphoma/leukemia (HTLV+) (ATCL)

Cytogenetics

Two cases with a complex karyotype are reported (males; 58 and 68 years old) - 0.75% of all ATCL cases with an abnormal karyotype (Sadamory et al, 1986; Sadamory et al, 1991).
In one the 1p32 deletion is presented as an additional anomaly. Both cases have structural aberrations involving chromosome 4.

SIL-TAL1 deletion

Disease: T- Acute lymphoblastic leukemia/lymphoblastic lymphoma (T-ALL)

Phenotype/cell stem origin

Is restricted to TCR of the alpha, beta and TCR delta lineage with a deletion of one or both alleles of the TCR delta gene (Breit et al, 1993a). TAL1 expression appeared to reflect the cortical stage of thymocyte development (late double positive stage) (Ferrando AA et al, 1992).

Etiology

SIL-TAL1 deletion is mediated via illegitimate V(D)J recombination processes of T-cell receptor (TCR) gene (Aplan PD et al, 1990b).

Epidemiology

SIL-TAL1 deletion is observed in 3-26 % of cases with T-ALL (Aplan et al, 1990b; Brown et al, 1990). The anomaly is more frequent in males (D'Angio M et al, 2014).

Clinics

SIL-TAL1 deletion is associated with higher initial WBC count, T-lineage immunophenotype with CD2 expression, predominant cortical T-phenotype, low incidence in adult patients and higher frequency of extramedullary relapse (Bash RO et al, 1993; Stock W et al, 1995; Mansur MB et al, 2009; D'Angio M et al, 2014). Increased risk from developing of tumor lysis syndrome and disseminated intravascular coagulation were also reported (Wang D et al, 2013).

Cytogenetics

SIL-TAL1 deletion represents a submicroscopic deletion of 90 Kb that affected all coding SIL exons. As a result of the deletion the first coding exons of TAL1 gene is juxtaposed to the promotor of SIL gene, causing its abnormal expression (Chen Q et al, 1990b; Aplan PD et al, 1992b). The breakpoint in SIL gene remains constant while several breakpoints of the TAL1 gene have been identified, which leads to formation of two main ($TAL1^{d1}$ and $TAL1^{d2}$ in 95% of the cases) and several rare types of SIL-TAL1 deletions (Breit et al, 1993a). Almost half of the cases with SIL-TAL1 deletion have normal karyotypes. The rest of the cases are presented with hyperdiploid and more frequently with pseudodiploid karyotypes associated with the structural anomalies del(6q) (7 cases), t(11;14)(p13;q11.2)/ t(11;14)(p15;q11.2) (3 cases) and t(8;14)(q24;q11.2) (1 case) (Wang Q et al, 2014; Cocce MC et al, 2015).

Prognosis

The data on the prognosis of the cases with SIL-TAL1 deletion are controversial. Mansur MB et al. (2009) reported negative impact on the patients, while no difference in survival and overall outcome were seen by D'Angio M et al. (2014).

Genetics

Using chromatin immunoprecipitation sequencing and chromosome conformation capture techniques, several scientific groups presented looping models for TAL1 expression in human and murine cell lines and described multiple interactions between TAL1 and their cis-acting regulatory elements (1a and 1b TAL1 promotors, enhancers and CTCF bound elements) (Zhou Y et al, 2013; Lai F et al, 2013; Patel B et al, 2013). Zhou Y et al. (2013) find in TAL1 expressing cell lines that the regulatory hubs which control transcription bring the TAL/SIL common breakpoint regions (TAL^{d}) into close proximity. The authors suggest that the physical proximity between these regions in the committed lymphoid cells may predispose to SIL-TAL1 deletion. However, the question remains: what is the reason that led to the rearrangement of the located in close proximity breakpoint regions? It should be noted that TAL1 gene is not expressed in the dividing double positive thymocytes, but SIL gene is expressed. In addition, the deletion primarily affects the SIL gene (all coding exons are deleted), so it can be assumed that the consequence of SIL-TAL1 deletion is not only deregulation of the TAL1 gene, but also inactivation of the SIL gene. These considerations suggest that the generation of SIL-TAL1 deletion is possibly due to a defective inactivation of the SIL gene which is intended to block the G2/M transition (through impairing the spindle assembly) and is a part of the complex mechanisms that induced the apoptosis during thymocyte negative selection. In this regard, future studies of looping patterns searching to discover the possible interactions leading to suppression of the SIL gene will elucidate the mechanisms of the formation of the SIL-TAL1 deletion as well as the role of the SIL gene in the regulation of the thymocyte apoptosis.

Genes involved and proteins

STIL

Location 1p33

STIL (or SIL: SCL interrupting locus) gene extended over 70 Kb and contained 18 exons.

The gene encodes a large (150 kDa) cytosolic protein implicated in regulation of the mitotic spindle checkpoint. It is required for the procentriole assembly and the regulation of the centriole

duplication. SIL mRNA expression is higher in rapidly proliferating cells and decreased rapidly during terminal differentiation. It is a positive regulator of the sonic hedgehog pathway and plays an important role in embryonic development. It is over expressed in multiple types of cancer and its expression correlates with the expression of mitotic checkpoint.

TAL1

Location 1p33

TAL1 (T-cell acute leukemia 1) is a member of the class II helix-loop-helix (bHLH) family of transcription factors. The gene extended over 16 Kb and contained 6 exons. Have two isoforms: a long TAL1 (L) and short TAL1 (S). After heterodimerization with members of the class I bHLH proteins known as E proteins (TCF3 (E2A), TCF12 (HEB), BHLHE22 (E2-2)), it binds E-box motif and forms complex with other transcription factors, including LMO, GATA1, RUNX1 and LDB1. TALl1 is expressed in hematopoietic stem cells, progenitor cells and erythro-megakaryocyte lineage. It is required for the specification of the haemangioblasts and the blood cell lineages and also plays a key role for the maturation of the megakaryocytes and erythroblasts (Porcher C et al, 1996; Porcher C et al, 2017; Gering M et al, 1998; Schlaeger TM et al, 2005). TAL1 is transcriptionally silenced during normal lymphocyte development including at the stage of CD4+ CD8+ double-positive thymocytes (the stage of maturation arrest of TAL1 positive T-ALL) (Tremblay M et al, 2010; Seita J et al, 2012). The transcriptional targets of the TAL1 in the normal hematopoietic cells are KIT, CDKN1A, DDIT4, KLF1, EPB42, GYPA, UBE2H and MEF2C (Lecuyer E et al, 2002; Lacombe J et al, 2010; Benyoucef A et al, 2015; Kassouf MT et al, 2010; Xu Z et al, 2003). As part of the highly interconnected auto-regulated circuit, it controls the transcription factors LMO2, RUNX2, MYB and GATA2. Except through a SIL-TAL1 deletion, TAL1 deregulation occurs also as a result of the chromosome translocations t(1;14)(p32;q11) and t(1;7)(p32;q34) and through its ectopic expression (60% of the cases) (Ferrando AA et al, 2002). Recently another mechanism oduces a binding motif for MYB transcription factor (Mansour MR et al, 2014). Deregulation of TAL1 inhibits E-proteins heterodimerization leading to block of T-cell differentiation. However, the oncogenic role of TAL1 in T-cell transformation is more complex and is linked to their influence on the function of the core regulatory circuitry (Sanda T et al, 2012) as well as on multiple downstream targets including ARID5B, NKX3-1, MYCN, CDKN2A, ALDH1A2 and MIR223 (Leong WZ et al, 2017; Kusy S et al, 2010; Astolfi A et al, 2014; Hansson A et al, 2003; Ono Y et al, 1998; Mansour MR et al, 2013).

References

Aamot HV, Torlakovic EE, Eide MB, Holte H, Heim S. Non-Hodgkin lymphoma with t(14;18): clonal evolution patterns and cytogenetic-pathologic-clinical correlations. J Cancer Res Clin Oncol. 2007 Jul;133(7):455-70

Aplan PD, Lombardi DP, Reaman GH, Sather HN, Hammond GD, Kirsch IR. Involvement of the putative hematopoietic transcription factor SCL in T-cell acute lymphoblastic leukemia. Blood. 1992 Mar 1;79(5):1327-33

Astolfi A, Vendemini F, Urbini M, Melchionda F, Masetti R, Franzoni M, Libri V, Serravalle S, Togni M, Paone G, Montemurro L, Bressanin D, Chiarini F, Martelli AM, Tonelli R, Pession A. MYCN is a novel oncogenic target in pediatric T-cell acute lymphoblastic leukemia. Oncotarget. 2014 Jan 15;5(1):120-30

Bash RO, Crist WM, Shuster JJ, Link MP, Amylon M, Pullen J, Carroll AJ, Buchanan GR, Smith RG, Baer R. Clinical features and outcome of T-cell acute lymphoblastic leukemia in childhood with respect to alterations at the TAL1 locus: a Pediatric Oncology Group study. Blood. 1993 Apr 15;81(8):2110-7

Benyoucef A, Calvo J, Renou L, Arcangeli ML, van den Heuvel A, Amsellem S, Mehrpour M, Larghero J, Soler E, Naguibneva I, Pflumio F. The SCL/TAL1 Transcription Factor Represses the Stress Protein DDiT4/REDD1 in Human Hematopoietic Stem/Progenitor Cells. Stem Cells. 2015 Jul;33(7):2268-79

Breit TM1, Mol EJ, Wolvers-Tettero IL, Ludwig WD, van Wering ER, van Dongen JJ.. Site-specific deletions involving the tal-1 and sil genes are restricted to cells of the T cell receptor alpha/beta lineage: T cell receptor delta gene deletion mechanism affects multiple genes. J Exp Med. 1993 Apr 1;177(4):965-77.

Brown L, Cheng JT, Chen Q, Siciliano MJ, Crist W, Buchanan G, Baer R.. Site-specific recombination of the tal-1 gene is a common occurrence in human T cell leukemia. EMBO J. 1990 Oct;9(10):3343-51.

Busson-Le Coniat M1, Salomon-Nguyen F, Dastugue N, Maarek O, Lafage-Pochitaloff M, Mozziconacci MJ, Baranger L, Brizard F, Radford I, Jeanpierre M, Bernard OA, Berger R.. Fluorescence in situ hybridization analysis of chromosome 1 abnormalities in hematopoietic disorders: rearrangements of DNA satellite II and new recurrent translocations. Leukemia. 1999 Dec;13(12):1975-81

Chen Q, Yang CY, Tsan JT, Xia Y, Ragab AH, Peiper SC, Carroll A, Baer R.. Coding sequences of the tal-1 gene are disrupted by chromosome translocation in human T cell leukemia. J Exp Med. 1990 Nov 1;172(5):1403-8.

Coccé M C, Alonso C N, Rossi J G, Bernasconi A R, Rampazzi M A, Felice M S, Rubio P L, Eberle S E, Medina A, Gallego M S.. Cytogenetic and Molecular Findings in Children with Acute Lymphoblastic Leukemia: Experience of a Single Institution in Argentina. Mol Syndromol. 2015 Oct; 6(4): 193-203. Published online 2015 Oct 7. doi: 10.1159/000441046

Colleoni GW, Bridge JA, Garicochea B, Liu J, Filippa DA, Ladanyi M.. ATIC-ALK: A novel variant ALK gene fusion in anaplastic large cell lymphoma resulting from the recurrent cryptic chromosomal inversion, inv(2)(p23q35). Am J Pathol. 2000 Mar;156(3):781-9.

Colovi? M, Jankovi? G, Suvajdzi? N, Mili? N, Dordevi? V, Jankovi? S.. Thirty patients with primary plasma cell leukemia: a single center experience. Med Oncol. 2008;25(2):154-60. doi: 10.1007/s12032-007-9011-5. Epub 2007 Oct 10.

D'Angi M, Valsecchi MG, Testi AM, Conter V, Nunes V, Parasole R, Colombini A, Santoro N, Varotto S, Caniglia M, Silvestri D, Consarino C, Levati L, Magrin E, Locatelli F, Basso G, Foà R, Biondi A, Cazzaniga G.. Clinical features and outcome of SIL/TAL1-positive T-cell acute lymphoblastic leukemia in children and adolescents: a 10-year experience of the AIEOP group. Haematologica. 2015 Jan;100(1):e10-3. doi:10.3324/haematol. 2014.112151.Epub 2014 Oct 10.

Dewald GW, Kyle RA, Hicks GA, Greipp PR.. The clinical significance of cytogenetic studies in 100 patients with multiple myeloma, plasma cell leukemia, or amyloidosis. Blood. 1985 Aug;66(2):380-90.

Ebrahim SA, Ladanyi M, Desai SB, Offit K, Jhanwar SC, Filippa DA, Lieberman PH, Chaganti RS.. Immunohistochemical, molecular, and cytogenetic analysis of a consecutive series of 20 peripheral T-cell lymphomas and lymphomas of uncertain lineage, including 12 Ki-1 positive lymphomas. Genes Chromosomes Cancer. 1990 May;2(1):27-35.

Falzetti D, Crescenzi B, Matteuci C, Falini B, Martelli MF, Van Den Berghe H, Mecucci C.. Genomic instability and recurrent breakpoints are main cytogenetic findings in Hodgkin's disease. Haematologica. 1999 Apr;84(4):298-305.

Fan YS and Rizkalla K.. Comprehensive cytogenetic analysis including multicolor spectral karyotyping and interphase fluorescence in situ hybridization in lymphoma diagnosis. a summary of 154 cases. Cancer Genet Cytogenet. 2003 May;143(1):73-9

Ferrando AA, Neuberg DS, Staunton J, Loh ML, Huard C, Raimondi SC, Behm FG, Pui CH, Downing JR, Gilliland DG, Lander ES, Golub TR, Look AT.. Gene expression signatures define novel oncogenic pathways in T cell acute lymphoblastic leukemia. Cancer Cell. 2002 Feb;1(1):75-87.

Fukuhara S, Nasu K, Kita K, Ueshima Y, Oguma S, Yamabe H, Nishigori M, Uchino H.. Cytogenetic approaches to the clarification of pathogenesis in lymphoid malignancies: clinicopathologic characterization of 14q+ marker-positive non-T-cell malignancies. Jpn J Clin Oncol. 1983 Sep;13(3):461-75.

Gaunt KL, Callaghan J, Roberts DF.. Karyotype abnormalities in non-Hodgkin lymphomas. Ann Genet. 1986;29(2):82-7.

Gering M, Rodaway A R, Göttgens B, Patient R K, Green A R.. The SCL gene specifies haemangioblast development from early mesoderm. EMBO J. 1998 Jul 15; 17(14): 4029-4045.doi: 10.1093/emboj/17.14.4029

Goyns MH, Hammond DW, Harrison CJ, Menasce LP, Ross FM, Hancock BW.. Structural abnormalities of the X chromosome in non-Hodgkin's lymphoma. Leukemia. 1993 Jun;7(6):848-52.

Hansson A, Manetopoulos C, Jönsson JI, Axelson H.. The basic helix-loop-helix transcription factor TAL1/SCL inhibits the expression of the p16INK4A and pTalpha genes. Biochem Biophys Res Commun. 2003 Dec 26;312(4):1073-81.

Hebraud B, Leleu X, Lauwers-Cances V, Roussel M, Caillot D, Marit G, Karlin L, Hulin C, Gentil C, Guilhot F, Garderet L, Lamy T, Brechignac S, Pegourie B, Jaubert J, Dib M, Stoppa AM, Sebban C, Fohrer C, Fontan J, Fruchart C, Macro M, Orsini-Piocelle F, Lepeu G, Sohn C, Corre J, Facon T, Moreau P, Attal M, Avet-Loiseau H.. Deletion of the 1p32 region is a major independent prognostic factor in young patients with myeloma: the IFM experience on 1195 patients. Leukemia. 2014 Mar;28(3):675-9. doi: 10.1038/leu.2013.225. Epub 2013 Jul 29.

Horsman DE, Connors JM, Pantzar T, Gascoyne RD.. Analysis of secondary chromosomal alterations in 165 cases of follicular lymphoma with t(14;18). Genes Chromosomes Cancer. 2001 Apr;30(4):375-82.

Ichinohasama R, Miura I, Takahashi N, Sugawara T, Tamate E, Endoh K, Endoh F, Naganuma H, DeCoteau JF, Griffin JD, Kadin ME, Ooya K. Ph-negative non-Hodgkin's lymphoma occurring in chronic phase of Ph-positive chronic myelogenous leukemia is defined as a genetically different neoplasm from extramedullary localized blast crisis: report of two cases and review of the literature. Leukemia. 2000 Jan;14(1):169-82.

Ivanov fverholm I, Tran AN, Olsson L, Zachariadis V, Heyman M, Rudd E, Syk Lundberg E, Nordenskjöld M, Johansson B, Nordgren A, Barbany G.. Detailed gene dose analysis reveals recurrent focal gene deletions in pediatric B-cell precursor acute lymphoblastic leukemia. Leuk Lymphoma. 2016 Sep;57(9):2161-70. doi: 10.3109/10428194.2015.1136740. Epub 2016 Apr 19.

Jarosova M, Volejnikova J, Porizkova I, Holzerova M, Pospisilova D, Novak Z, Vrbkova J, Mihal V.. Chromosomal aberrations in childhood acute lymphoblastic leukemia: 15-year single center experience. Cancer Genet. 2016 Jul-Aug;209(7-8):340-7. doi: 10.1016/j.cancergen.2016.06.004. Epub 2016 Jun 11.

Kaneko H, Shimura K, Horiike S, Kuroda J, Matsumoto Y, Yokota S, Nishida K, Ohkawara Y, Taniwaki M.. Cytogenetic analysis of de novo CD5-positive diffuse large B-cell lymphoma. Asia Pac J Clin Oncol. 2011 Dec;7(4):346-50. doi: 10.1111/j.1743-7563.2011.01432.x

Kassouf MT, Hughes JR, Taylor S, McGowan SJ, Soneji S, Green AL, Vyas P, Porcher C.. Genome-wide identification of TAL1's functional targets: insights into its mechanisms of action in primary erythroid cells. Genome Res. 2010 Aug;20(8):1064-83. doi: 10.1101/gr.104935.110. Epub 2010 Jun 21.

Kristoffersson U, Olsson H, Akerman M, Mitelman F.. Cytogenetic studies in non-Hodgkin lymphomas--results from fine-needle aspiration samples. Hereditas. 1985;103(1):63-76.

Kusy S, Gerby B, Goardon N, Gault N, Ferri F, Gérard D, Armstrong F, Ballerini P, Cayuela JM, Baruchel A, Pflumio F, Roméo PH. NKX3.1 is a direct TAL1 target gene that mediates proliferation of TAL1-expressing human T cell acute lymphoblastic leukemia. J Exp Med. 2010 Sep 27;207(10):2141-56. doi: 10.1084/jem.20100745. Epub 2010 Sep 20.

Lécuyer E, Herblot S, Saint-Denis M, Martin R, Begley CG, Porcher C, Orkin SH, Hoang T.. The SCL complex regulates c-kit expression in hematopoietic cells through functional interaction with Sp1. Blood. 2002 Oct 1;100(7):2430-40.

Lacombe J, Herblot S, Rojas-Sutterlin S, Haman A, Barakat S, Iscove NN, Sauvageau G, Hoang T.. Scl regulates the quiescence and the long-term competence of

hematopoietic stem cells. Blood. 2010 Jan 28;115(4):792-803. doi: 10.1182/blood-2009-01-201384. Epub 2009 Oct 22

Lai F, Orom UA, Cesaroni M, Beringer M, Taatjes DJ, Blobel GA, Shiekhattar R.. Activating RNAs associate with Mediator to enhance chromatin architecture and transcription. Nature. 2013 Feb 28;494(7438):497-501. doi: 10.1038/nature11884. Epub 2013 Feb 17.

Leong WZ, Tan SH, Ngoc PCT, Amanda S, Yam AWY, Liau WS, Gong Z, Lawton LN, Tenen DG, Sanda T.. ARID5B as a critical downstream target of the TAL1 complex that activates the oncogenic transcriptional program and promotes T-cell leukemogenesis. Genes Dev. 2017 Dec 1;31(23-24):2343-2360. doi: 10.1101/gad.302646.117. Epub 2018 Jan 11.

Lewis JP and MacKenzie MR.. Non-random chromosomal aberrations associated with multiple myeloma. Hematol Oncol. 1984 Oct-Dec;2(4):307-17.

Lloveras E, Granada I, Zamora L, Espinet B, Florensa L, Besses C, Xandri M, Pérez-Vila ME, Millà F, Woessner S, Solé F.. Cytogenetic and fluorescence in situ hybridization studies in 60 patients with multiple myeloma and plasma cell leukemia. Cancer Genet Cytogenet. 2004 Jan 1;148(1):71-6.

Mansour MR, Abraham BJ, Anders L, Berezovskaya A, Gutierrez A, Durbin AD, Etchin J, Lawton L, Sallan SE, Silverman LB, Loh ML, Hunger SP, Sanda T, Young RA, Look AT.. Oncogene regulation. An oncogenic super-enhancer formed through somatic mutation of a noncoding intergenic element. Science. 2014 Dec 12;346(6215):1373-7. doi: 10.1126/science.1259037. Epub 2014 Nov 13.

Mansur MB, Emerenciano M, Brewer L, Sant'Ana M, Mendonça N, Thuler LC, Koifman S, Pombo-de-Oliveira MS.. SIL-TAL1 fusion gene negative impact in T-cell acute lymphoblastic leukemia outcome. Leuk Lymphoma. 2009 Aug;50(8):1318-25. doi: 10.1080/10428190903040014.

Mitelman F, Johansson B and Mertens F (Eds.). Mitelman Database of Chromosome Aberrations and Gene Fusions in Cancer (2019). http://cgap.nci.nih.gov/Chromosomes/Mitelman

Offit K, Jhanwar S, Ebrahim SA, Filippa D, Clarkson BD, Chaganti RS.. t(3;22)(q27;q11): a novel translocation associated with diffuse non-Hodgkin's lymphoma. Blood. 1989 Nov 1;74(6):1876-9.

Ono Y, Fukuhara N, Yoshie O.. TAL1 and LIM-only proteins synergistically induce retinaldehyde dehydrogenase 2 expression in T-cell acute lymphoblastic leukemia by acting as cofactors for GATA3. Mol Cell Biol. 1998 Dec;18(12):6939-50.

Patel B, Kang Y, Cui K, Litt M, Riberio MS, Deng C, Salz T, Casada S, Fu X, Qiu Y, Zhao K, Huang S.. Aberrant TAL1 activation is mediated by an interchromosomal interaction in human T-cell acute lymphoblastic leukemia. Leukemia. 2014 Feb;28(2):349-61. doi: 10.1038/leu.2013.158. Epub 2013 May 23.

Porcher C, Chagraoui H, Kristiansen MS.. SCL/TAL1: a multifaceted regulator from blood development to disease. Blood. 2017 Apr 13;129(15):2051-2060. doi: 10.1182/blood-2016-12-754051. Epub 2017 Feb 8.

Porcher C, Swat W, Rockwell K, Fujiwara Y, Alt FW, Orkin SH.. The T cell leukemia oncoprotein SCL/tal-1 is essential for development of all hematopoietic lineages. Cell. 1996 Jul 12;86(1):47-57.

Prigogina EL, Puchkova GP, Mayakova SA.. Nonrandom chromosomal abnormalities in acute lymphoblastic leukemia of childhood. Cancer Genet Cytogenet. 1988 Jun;32(2):183-203.

Pui CH, Carroll AJ, Raimondi SC, Schell MJ, Head DR, Shuster JJ, Crist WM, Borowitz MJ, Link MP, Behm FG, et al.. Isochromosomes in childhood acute lymphoblastic leukemia: a collaborative study of 83 cases. Blood. 1992 May 1;79(9):2384-91.

Sadamori N, Isobe M, Shimizu S, Yamamori T, Itoyama T, Ikeda S, Yamada Y, Ichimaru M.. Relationship between chromosomal breakpoint and molecular rearrangement of T-cell antigen receptors in adult T-cell leukaemia. Acta Haematol. 1991;86(1):14-9

Sanda T, Lawton LN, Barrasa MI, Fan ZP, Kohlhammer H, Gutierrez A, Ma W, Tatarek J, Ahn Y, Kelliher MA, Jamieson CH, Staudt LM, Young RA, Look AT.. Core transcriptional regulatory circuit controlled by the TAL1 complex in human T cell acute lymphoblastic leukemia. Cancer Cell. 2012 Aug 14;22(2):209-21. doi: 10.1016/j.ccr.2012.06.007.

Schlaeger TM, Mikkola HK, Gekas C, Helgadottir HB, Orkin SH.. Tie2Cre-mediated gene ablation defines the stem-cell leukemia gene (SCL/tal1)-dependent window during hematopoietic stem-cell development. Blood. 2005 May 15;105(10):3871-4. Epub 2005 Jan 27.

Schlegelberger B, Weber-Matthiesen K, Himmler A, Bartels H, Sonnen R, Kuse R, Feller AC, Grote W.. Cytogenetic findings and results of combined immunophenotyping and karyotyping in Hodgkin's disease. Leukemia. 1994 Jan;8(1):72-80.

Schoch C, Rieder H, Stollmann-Gibbels B, Freund M, Tischler HJ, Silling-Engelhardt G, Fonatsch C.. 17p anomalies in lymphoid malignancies: diagnostic and prognostic implications. Leuk Lymphoma. 1995 Apr;17(3-4):271-9.

Seita J, Sahoo D, Rossi DJ, Bhattacharya D, Serwold T, Inlay MA, Ehrlich LI, Fathman JW, Dill DL, Weissman IL.. Gene Expression Commons: an open platform for absolute gene expression profiling. PLoS One. 2012;7(7):e40321. doi: 10.1371/journal.pone.0040321. Epub 2012 Jul 18.

Seong C, Delasalle K, Hayes K, Weber D, Dimopoulos M, Swantkowski J, Huh Y, Glassman A, Champlin R, Alexanian R.. Prognostic value of cytogenetics in multiple myeloma. Br J Haematol. 1998 Apr;101(1):189-94.

Stock W, Westbrook CA, Sher DA, Dodge R, Sobol RE, Wurster-Hill D, Davey FR, Larson RA, LeBeau MM, Aplan PD, Frankel SR, Stewart CC, Bloomfield CD.. Low incidence of TAL1 gene rearrangements in adult acute lymphoblastic leukemia: A cancer and leukemia group B study (8762) Clin Cancer Res. 1995 Apr;1(4):459-63.

Tremblay M, Tremblay CS, Herblot S, Aplan PD, Hébert J, Perreault C, Hoang T.. Modeling T-cell acute lymphoblastic leukemia induced by the SCL and LMO1 oncogenes. Genes Dev. 2010 Jun 1;24(11):1093-105. doi: 10.1101/gad.1897910.

Wang D, Zhu G, Wang |N, Zhou X, Yang Y, Zhou S, Xiong J, He J, Jiang L, Li C, Xu D, Huang L, Zhou J. SIL-TAL1 Rearrangement is Related with Poor Outcome: A Study from a Chinese Institution. PLoS One. 2013; 8(9): e73865.Published online 2013 Sep 9. doi: 10.1371/journal.pone.0073865

Wang Q, Wu LL, Dai HP, Ping NN, Wu CX, Pan JL, Cen JN, Qiu HY, Chen SN.. [Correlation between expression of

SIL-TAL1 fusion gene and deletion of 6q in T-cell acute lymphoblastic leukemia].

Weinländer G, Drach J, Raderer M, Okamoto I, Ackermann J, Stögermayer B, Fazeny B, Nowotny H, Marosi C.. Cytogenetic analysis and fluorescence in situ hybridization in a case of IgD multiple myeloma. Cancer Genet Cytogenet. 1998 Sep;105(2):172-6.

Weisenburger DD, Gordon BG, Vose JM, Bast MA, Chan WC, Greiner TC, Anderson JR, Sanger WG.. Occurrence of the t(2;5)(p23;q35) in non-Hodgkin's lymphoma. Blood. 1996 May 1;87(9):3860-8.

Wlodarska I, Stul M, De Wolf-Peeters C, Verhoef G, Mecucci C, Cassiman JJ, Van den Berghe H.. t(1;19) without detectable E2A rearrangements in two t(14;18)-positive lymphoma/leukemia cases. Genes Chromosomes Cancer. 1994 Jul;10(3):171-6.

Wu KL, Beverloo B, Lokhorst HM, Segeren CM, van der Holt B, Steijaert MM, Westveer PH, Poddighe PJ, Verhoef GE, Sonneveld P; Dutch-Belgian Haemato-Oncology Cooperative Study Group (HOVON); Dutch Working Party on Cancer Genetics and Cytogenetics (NWCGC).. Abnormalities of chromosome 1p/q are highly associated with chromosome 13/13q deletions and are an adverse

prognostic factor for the outcome of high-dose chemotherapy in patients with multiple myeloma. Br J Haematol. 2007 Feb;136(4):615-23.

Xu Z1, Huang S, Chang LS, Agulnick AD, Brandt SJ.. Identification of a TAL1 target gene reveals a positive role for the LIM domain-binding protein Ldb1 in erythroid gene expression and differentiation. Mol Cell Biol. 2003 Nov;23(21):7585-99.

Yunis JJ, Oken MM, Theologides A, Howe RB, Kaplan ME.. Recurrent chromosomal defects are found in most patients with non-Hodgkin's-lymphoma. Cancer Genet Cytogenet. 1984 Sep;13(1):17-28.

Zhou Y, Kurukuti S, Saffrey P, Vukovic M, Michie AM, Strogantsev R, West AG, Vetrie D.. Chromatin looping defines expression of TAL1, its flanking genes, and regulation in T-ALL. Blood. 2013 Dec 19;122(26):4199-209. doi: 10.1182/blood-2013-02-483875. Epub 2013 Nov 7.

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