

BACKGROUND

PLZF/ZBTB16 was initially identified as a fusion partner of retinoic acid receptor α (RAR α) in a variant chromosomal translocation $t(11;17)(q23;q21)$ that occurred in a subset of acute promyelocytic leukemia patients. PLZF/ZBTB16 is a transcriptional repressor of the POK (POZ and Krüppel) family of proteins. It contains one BTB (Broad complex, Tramtrack, and Bric à brac)/POZ (poxviruses and zinc finger and Krüppel) domain at the NH₂-terminal moiety and 9C₂H₂ Krüppel-type zinc fingers at the carboxyl-terminal end of the protein. The POZ/BTB domain mediates interactions with proteins such as transcriptional co-repressors entailing chromatin remodeling and transcriptional silencing. The Krüppel-type zinc fingers confer specificity of the repressor activity to particular promoters by interacting with corresponding response elements in regulatory regions of genes repressed by PLZF/ZBTB16. The hinge region of the protein contains a PEST domain with two consensus sites for CDK2-mediated phosphorylation that triggers ubiquitination and subsequent degradation of PLZF through the ubiquitin-proteasome pathway. The human PLZF/ZBTB16 gene maps to chromosome 11q22-q23 with seven exons distributed over a region of approximately 200 kb. Although additional alternative transcripts encoding distinct proteins have been reported, most recent NCBI and Ensembl databases contain 2 and 3 transcripts, respectively, that differ only in their 5' untranslated region and thus encode the same protein.¹

Regarding its function, a natural mutation (luxoid) in, and knock-out of, the mouse homologue *Zfp145/ZBTB16* unraveled a crucial role in limb and skeletal patterning and spermatogonial stem cell maintenance. PLZF/ZBTB16 has further been implicated in tumor suppression in melanoma and prostate cancer, ascribed to its ability to cause cell cycle arrest and induce apoptosis in certain cell systems.² The complex effects of PLZF/ZBTB16 have been associated with transcriptional repression of numerous genes such as members of the Hox family of transcription factors, kit, CRABPI, c-myc, CCNA2/Cyclin A, CDKN1B/p27/Kip1 and possibly others. Glucocorticoids (GCs) cause cell cycle arrest and apoptosis in lymphoid cells which is exploited to treat lymphoid malignancies. It was shown that PLZF/ZBTB16 is a glucocorticoid response gene in acute lymphoblastic leukemia. Thus, The suggested role of PLZF/ZBTB16 in cell cycle arrest and apoptosis induction in some systems, further supported a possible role of PLZF/ZBTB16 induction in the anti-leukemic glucocorticoid (GC) response.³ In addition, PLZF/ZBTB16 also plays a role in suppressing the proliferation of normal cultured human corneal endothelial cells. The expression of PLZF in HCECs is closely related to the formation of cell-cell contacts.⁴

References

1. McConnell, M.J. & Licht, J.D.: *Curr. Top. Microbiol. Immunol.* 313:31-48, 2007
2. Costoya, J.A. & Pandolfi, P.P.: *Curr. Opin. Hematol.* 8:212-7, 2001
3. Wasim, M. et al: *J. Steroid Biochem. Mol. Biol.* 120:218-27, 2010
4. Joko, T. et al: *Mol. Vis.* 13:649-58, 2007

TECHNICAL INFORMATION

Source:

PLZF/ZBTB16 Antibody is a mouse monoclonal antibody raised against purified recombinant human PLZF/ZBTB16 fragments expressed in *E. coli*.

Specificity and Sensitivity:

This antibody detects PLZF/ZBTB16 proteins without cross-reactivity with other related proteins.

Storage Buffer: PBS and 30% glycerol

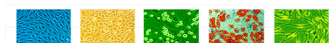
Storage:

Store at -20°C for at least one year. Store at 4°C for frequent use. Avoid repeated freeze-thaw cycles.

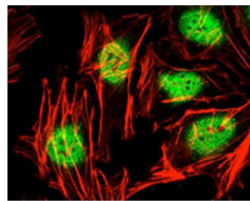
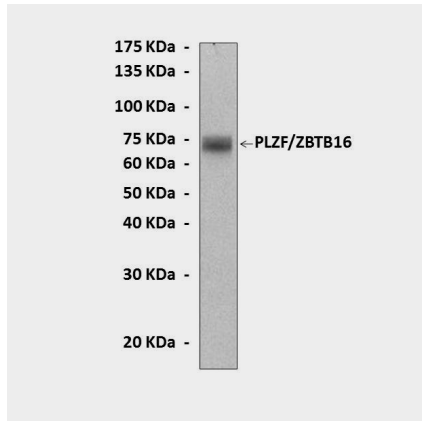
APPLICATIONS

Application:	*Dilution:
WB	1:1000
IP	n/d
IHC	n/d
ICC	1:50-200
FACS	n/d

**Optimal dilutions must be determined by end user.*



QUALITY CONTROL DATA



Top: Western Blot detection of PLZF/ZBTB16 proteins in HeLa cell lysate using PLZF/ZBTB16 Antibody.
Bottom: this antibody also stains HeLa cells in confocal immunofluorescent analysis (PLZF/ZBTB16 Antibody: Green; Actin filaments: Red).

