Anti-PLZF/ZBTB16: Mouse PLZF/ZBTB16 Antibody

Product Sheet CP10261

Description

BACKGROUND PLZF/ZBTB16 was initially identified as a fusion partner of retinoic acid receptor ? (RAR?) 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

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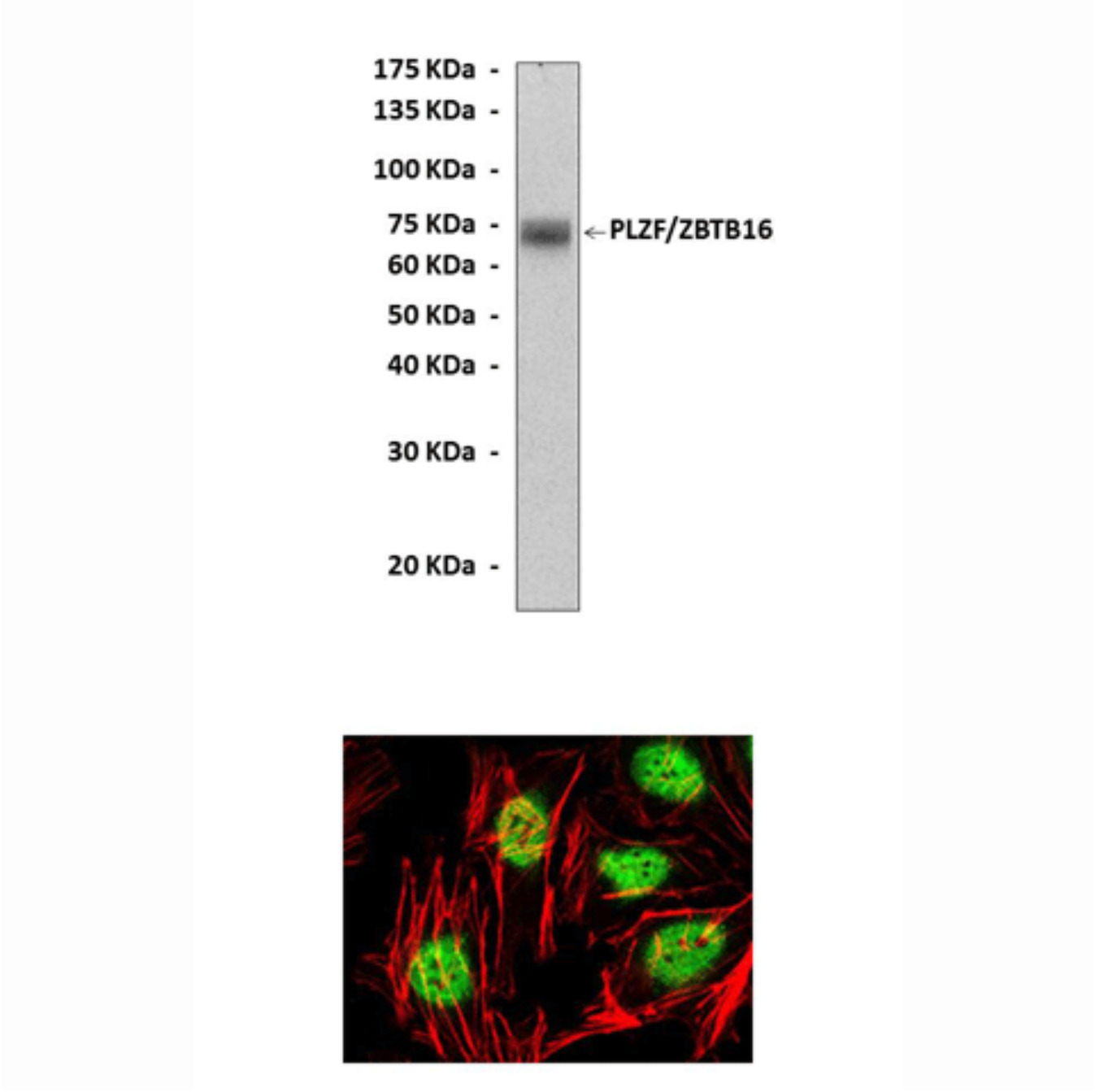
3. Wasim, M. et al: J. Steroid Biochem. Mol. Biol. 120:218-27, 2010
4. Joko, T. et al: Mol. Vis. 13:649-58, 2007



[1]

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).

Details

Cat.No.:	CP10261
Antigen:	Purified recombinant human PLZF/ZBTB16 fragments expressed in *E. coli*.
Isotype:	Mouse IgG1
Species & predicted species cross-reactivity ( ):	Human, Mouse, Rat
Applications & Suggested starting dilutions:*	WB 1:1000 IP n/d IHC n/d ICC 1:50 - 1:200 FACS n/d
Predicted Molecular Weight of protein:	74 kDa
Specificity/Sensitivity:	Detects PLZF/ZBTB16 proteins without cross-reactivity with other related proteins.
Storage:	Store at -20°C, 4°C for frequent use. Avoid repeated freeze-thaw cycles.

*Optimal working dilutions must be determined by end user.

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