

v-Maf (C-20): sc-478

BACKGROUND

Members of the Maf family of basic region/leucine zipper (bZIP) transcription factors affect transcription in either a positive or negative fashion, depending on their particular protein partner and the context of the target promoter. v-Maf (the avian viral homolog of Maf, also designated c-Maf, for cell, or Maf2) and the closely related family members Neural retina leucine zipper (Nrl), L-Maf, and Krml1/MafB (Maf-1) all bind to T-MARE sites and have been implicated in a wide variety of developmental and physiologic roles. The three small Maf family proteins (MafF, MafG, and MafK) are components of NF-E2 that function as heterodimers with the large tissue-restricted subunit of NF-E2 called p45, and they are implicated in the transcriptional regulation of many erythroid-specific genes. MafB is expressed in a wide variety of tissues and encodes a protein containing a typical bZip motif in its carboxy-terminal region. As a transcriptional activator, MafB plays a pivotal role in regulating lineage-specific gene expression during hematopoiesis by repressing Ets-1-mediated transcription of key erythroid-specific genes in myeloid cells. c-Maf interacts with the c-Myb DNA binding domain and forms Myb-Maf complexes, which in turn mediate the cooperative interactions between c-Myb and Ets-1 during early myeloid cell differentiation.

REFERENCES

1. Kerppola, T.K. et al. 1994. A conserved region adjacent to the basic domain is required for recognition of an extended DNA binding site by Maf/Nrl family proteins. *Oncogene* 9: 3149-3158.
2. Igarashi, K., et al. 1995. Conditional expression of the ubiquitous transcription factor MafK induces erythroleukemia cell differentiation. *Proc. Natl. Acad. Sci. USA* 92: 7445-7449.
3. Kataoka, K., et al. 1995. Small Maf proteins heterodimerize with Fos and may act as competitive repressors of the NF-E2 transcription factor. *Mol. Cell. Biol.* 15: 2180-2190.
4. Johnsen, O., et al. 1996. Small Maf proteins interact with the human transcription factor TCF11/Nrf1/LCR-F1. *Nucleic Acids Res.* 24: 4289-4297.
5. Hedge, S.P., et al. 1998. c-Maf interacts with c-Myb to regulate transcription of an early myeloid gene during differentiation. *Mol. Cell. Biol.* 18: 2729-2737.

CHROMOSOMAL LOCATION

Genetic locus: MAF (human) mapping to 16q23.1; Maf (mouse) mapping to 8 E1.

SOURCE

v-Maf (C-20) is an affinity purified rabbit polyclonal antibody raised against a peptide mapping at the C-terminus of v-Maf of chicken origin.

STORAGE

Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

RESEARCH USE

For research use only, not for use in diagnostic procedures.

PRODUCT

Each vial contains 200 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Blocking peptide available for competition studies, sc-478 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

Available as TransCruz reagent for Gel Supershift and ChIP applications, sc-478 X, 200 µg/0.1 ml.

APPLICATIONS

v-Maf (C-20) is recommended for detection of v-Maf of avian, and to a lesser extent, c-Maf of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1–2 µg per 100–500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

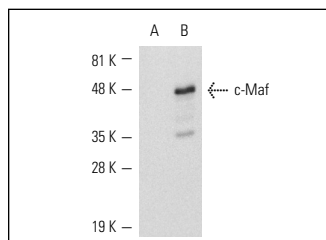
v-Maf (C-20) is also recommended for detection of v-Maf of avian origin and, to a lesser extent, c-Maf in additional species, including avian.

v-Maf (C-20) X TransCruz antibody is recommended for Gel Supershift and ChIP applications.

Molecular Weight of v-Maf: 43 kDa.

Positive Controls: COS whole cell lysate.

DATA



v-Maf (C-20): sc-478. Western blot analysis of c-Maf expression in control (A) and c-Maf-transfected (B) COS cells.

SELECT PRODUCT CITATIONS

1. Hedge, S.P., et al. 1998. c-Maf interacts with c-Myb to regulate transcription of an early Myeloid gene during differentiation. *Mol. Cell. Biol.* 18: 2729-2737.
2. Cull, V.S., et al. 2003. Type I interferon differential therapy for erythroleukemia: specificity of STAT activation. *Blood* 101: 2727-2735.

PROTOCOLS

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