

Store at -20C
#8220**PhosphoPlus® ATF-2 (Thr71) Antibody Duet**

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Product Includes	Product #	Quantity	Mol. Wt	Isotype/Source
Phospho-ATF-2 (Thr71) (11G2) Rabbit mAb	5112	100 µl	70 kDa	Rabbit IgG
ATF-2 (20F1) Rabbit mAb	9226	100 µl	65 to 75 kDa	Rabbit IgG

Please visit cellsignal.com for individual component applications, species cross-reactivity, dilutions, protocols, and additional product information.

Description PhosphoPlus® Duets from Cell Signaling Technology (CST) provide a means to assess protein activation status. Each Duet contains an activation-state and total protein antibody to your target of interest. These antibodies have been selected from CST's product offering based upon superior performance in specified applications.

Storage Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA, 50% glycerol and less than 0.02% sodium azide. Store at -20°C. Do not aliquot the antibody.

Background The transcription factor ATF-2 (also called CRE-BP1) binds to both AP-1 and CRE DNA response elements and is a member of the ATF/CREB family of leucine zipper proteins (1). ATF-2 interacts with a variety of viral oncoproteins and cellular tumor suppressors and is a target of the SAPK/JNK and p38 MAP kinase signaling pathways (2-4). Various forms of cellular stress, including genotoxic agents, inflammatory cytokines, and UV irradiation, stimulate the transcriptional activity of ATF-2. Cellular stress activates ATF-2 by phosphorylation of Thr69 and Thr71 (2-4). Both SAPK and p38 MAPK have been shown to phosphorylate ATF-2 at these sites *in vitro* and in cells transfected with ATF-2. Mutations of these sites result in the loss of stress-induced transcription by ATF-2 (2-4). In addition, mutations at these sites reduce the ability of E1A and Rb to stimulate gene expression via ATF-2 (2).

Background References

1. Abdel-Hafiz, H.A. et al. (1992) *Mol Endocrinol* 6, 2079-89.
2. Gupta, S. et al. (1995) *Science* 267, 389-93.
3. van Dam, H. et al. (1995) *EMBO J* 14, 1798-811.
4. Livingstone, C. et al. (1995) *EMBO J* 14, 1785-97.

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