

**NRF2 (D9J1B) Rat mAb**

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|                               |                         |                                   |                            |                                     |                               |                                 |
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| <b>Applications:</b><br>IF-IC | <b>Reactivity:</b><br>M | <b>Sensitivity:</b><br>Endogenous | <b>MW (kDa):</b><br>97-100 | <b>Source/Isotype:</b><br>Rat IgG2b | <b>UniProt ID:</b><br>#Q60795 | <b>Entrez-Gene Id:</b><br>18024 |
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**Product Usage Information****Application**

Immunofluorescence (Immunocytochemistry)

**Dilution**

1:50 - 1:200

**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.*

**Specificity/Sensitivity**

NRF2 (D9J1B) Rat mAb recognizes endogenous levels of total NRF2 protein.

**Source / Purification**

Monoclonal antibody is produced by immunizing animals with recombinant protein surrounding Ala200 of mouse NRF2 protein.

**Background**

The nuclear factor-like 2 (NRF2) transcriptional activator binds antioxidant response elements (ARE) of target gene promoter regions to regulate expression of oxidative stress response genes. Under basal conditions, the NRF2 inhibitor INrf2 (also called KEAP1) binds and retains NRF2 in the cytoplasm where it can be targeted for ubiquitin-mediated degradation (1). Small amounts of constitutive nuclear NRF2 maintain cellular homeostasis through regulation of basal expression of antioxidant response genes. Following oxidative or electrophilic stress, KEAP1 releases NRF2, thereby allowing the activator to translocate to the nucleus and bind to ARE-containing genes (2). The coordinated action of NRF2 and other transcription factors mediates the response to oxidative stress (3). Altered expression of NRF2 is associated with chronic obstructive pulmonary disease (COPD) (4). NRF2 activity in lung cancer cell lines directly correlates with cell proliferation rates, and inhibition of NRF2 expression by siRNA enhances anti-cancer drug-induced apoptosis (5).

**Background References**

1. Cullinan, S.B. et al. (2004) *Mol Cell Biol* 24, 8477-86.
2. Nguyen, T. et al. (2005) *J Biol Chem* 280, 32485-92.
3. Jaiswal, A.K. (2004) *Free Radic Biol Med* 36, 1199-207.
4. Suzuki, M. et al. (2008) *Am J Respir Cell Mol Biol* 39, 673-82.
5. Homma, S. et al. (2009) *Clin Cancer Res* 15, 3423-32.

**Species Reactivity**

Species reactivity is determined by testing in at least one approved application (e.g., western blot).

**Applications Key**

**IF-IC:** Immunofluorescence (Immunocytochemistry)

**Cross-Reactivity Key**

**M:** Mouse

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