

Store at  
-20C  
#51274**Keratin 8/18 (C51) Mouse mAb (BSA and Azide Free)**

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**For Research Use Only. Not for Use in Diagnostic Procedures.**

Applications:	Reactivity:	Sensitivity:	MW (kDa):	Source/Isotype:	UniProt ID:	Entrez-Gene Id:
W, IHC-P, IF-IC, FC-FP	H Mk	Endogenous	46 Keratin 18, 55 Keratin 8.	Mouse IgG1	#P05787, #P05783	3856, 3875

### Product Usage Information

This product is the carrier free version of product #4546. All data were generated using the same antibody clone in the standard formulation which contains BSA and glycerol.

This formulation is ideal for use with technologies requiring specialized or custom antibody labeling, including fluorophores, metals, lanthanides, and oligonucleotides. It is not recommended for ChIP, ChIP-seq, CUT&RUN, or CUT&Tag assays. If you require a carrier-free formulation for chromatin profiling, please contact us. Optimal dilutions/concentrations should be determined by the end user.

BSA and Azide Free antibodies are quality control tested by size exclusion chromatography (SEC) to determine antibody integrity.

### Formulation

Supplied in 1X PBS (10 mM Na<sub>2</sub>HPO<sub>4</sub>, 3 mM KCl, 2 mM KH<sub>2</sub>PO<sub>4</sub>, and 140 mM NaCl (pH 7.8)). BSA and Azide Free.

For standard formulation of this product see product #4546.

### Storage

Store at -20°C. *This product will freeze at -20°C so it is recommended to aliquot into single-use vials to avoid multiple freeze/thaw cycles.* A slight precipitate may be present and can be dissolved by gently vortexing. This will not interfere with antibody performance.

### Specificity/Sensitivity

Keratin 8/18 (C51) Mouse mAb (BSA and Azide Free) detects endogenous levels of total keratins 8 and 18. The antibody does not cross-react with other keratins.

### Source / Purification

Monoclonal antibody (isotype: IgG1) is produced by immunizing a BALB/c mouse with a cytoskeleton preparation from HeLa cells.

### Background

Keratins (cytokeratins) are intermediate filament proteins that are mainly expressed in epithelial cells. Keratin heterodimers composed of an acidic keratin (or type I keratin, keratins K9-K28) and a basic keratin (or type II keratin, keratins K1-K8 and K71-K80) assemble to form filaments. Keratin isoforms demonstrate tissue- and differentiation-specific profiles that make them useful as research and clinical biomarkers (1,2).

Dysregulation/mutations in keratin genes can lead to a variety of disorders affecting the skin, hair, nails, and other epithelial tissues (3). While expression of keratins can be variable, immunohistochemical staining of keratins is widely used to help in the identification and classification of epithelial tumors, and may also provide prognostic information.

Keratins 8 and 18 (K8/K18) are expressed in simple epithelia of normal tissue, as well as in adenocarcinomas of the breast, lung, ovary, and gastrointestinal tract. Keratin 17 is expressed in basal keratinocytes of stratified epithelia, hair follicles, and sebaceous glands. Onset of keratin 17 expression coincides with the definition of major epithelial lineages during skin development (4). Keratin 14 (K14) is expressed in basal cells of stratified epithelia, and in basal-like subtypes of breast cancer and squamous cell carcinomas. Keratin 19 (K19) is expressed in glandular epithelia, including the liver, gallbladder, and pancreas, as well as in adenocarcinomas of the breast, thyroid, and bile duct. Keratin 20 (K20) is expressed in gastrointestinal epithelium, urothelium, and Merkel cells in the skin, as well as in colorectal carcinomas and some urothelial carcinomas. Keratin 5/6 (K5/6) is expressed in basal cells of stratified epithelia, including the skin, prostate, and breast, as well as in basal-like breast cancers, squamous cell carcinomas, and some lung carcinomas. Keratin 7 (K7) is expressed in glandular epithelia, such as those in the lung, breast, and female reproductive tract, as well as in adenocarcinomas of the lung, breast, and ovary (5,6).

Keratins, particularly K8, K18, and K19, serve as biomarkers for identification of circulating tumor cells (CTCs) (5).

Post-translational modifications, including phosphorylation, acetylation, ubiquitylation, sumoylation,

glycosylation, and transamidation, have been shown to affect the functions of keratins in normal and disease states (6). Understanding the molecular mechanisms underlying these PTMs may provide insights into cancer pathogenesis.

## Background References

1. Chang, L. and Goldman, R.D. (2004) *Nat Rev Mol Cell Biol* 5, 601-13.
2. Schweizer, J. et al. (2006) *J Cell Biol* 174, 169-74.
3. Sarma, A. (2022) *Int J Biol Macromol* 219, 395-413.
4. McGowan, K.M. and Coulombe, P.A. (1998) *J Cell Biol* 143, 469-86.
5. Werner, S. et al. (2020) *Mol Aspects Med* 72, 100817.
6. Dmello, C. et al. (2019) *J Biosci* 44, 33.

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## Species Reactivity

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

## Applications Key

**W:** Western Blotting **IHC-P:** Immunohistochemistry (Paraffin) **IF-IC:** Immunofluorescence (Immunocytochemistry) **FC-FP:** Flow Cytometry (Fixed/Permeabilized)

## Cross-Reactivity Key

**H:** Human **Mk:** Monkey

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