Phospho-Ubiquitin (pS65) Rabbit Polyclonal Antibody

Cat. # AB141

Background: Ubiquitin, a small regulatory protein, plays a critical role in various cellular processes critical for maintaining homeostasis. Among its pivotal roles, ubiquitin governs protein degradation via the ubiquitin-proteasome system, ensuring the removal of misfolded or damaged proteins. Additionally, ubiquitin participates in the selective clearance of damaged mitochondria through a process known as mitophagy, which is vital for preserving cellular health and functionality.

The phosphorylation of ubiquitin at serine 65 is a key post-translational modification involved in the regulation of mitophagy. Through this modification, phosphorylated ubiquitin facilitates the clearance of dysfunctional mitochondria, thereby contributing to the maintenance of cellular homeostasis. Recent findings suggest a correlation between elevated phospho-ubiquitin levels and the accumulation of damage mitochondria observed in Parkinson's Disease (PD) progression. This association underscores the potential utility of phospho-ubiquitin as a biomarker for PD diagnosis and prognosis. The development of specific phospho-ubiquitin antibodies enables researchers to investigate the role of phospho-ubiquitin in mitophagy regulation and its implications in cellular health and disease pathogenesis.

Application: Western blotting. For all applications, optimal conditions should be determined by the end user.

Product Information

Immunogen:	Phospho-Ubiquitin (pS65)
Purification:	Polyclonal antibodies are produced by immunizing rabbits with a synthetic peptide surrounding pS65 of ubiquitin. Antibodies were recovered by affinity purification and subjected to an additional immunodepletion step with control peptides.
Specificity:	Antibody detects ubiquitin only when phosphorylated at serine 65.
Supplied as:	Liquid, phosphate-buffered saline with 50% glycerol
MW:	8.5 kDa (monomer)
Quantity:	100µl
Storage:	Store at -20°C. Avoid repeated freeze/thaw cycles.

References

1. Chin, L.-S., & Li, L. (2016). Ubiquitin phosphorylation in parkinson's disease: Implications for pathogenesis and treatment. Translational Neurodegeneration, 5(1). https://doi.org/10.1186/s40035-015-0049-6

2. Heo, J.-M., Ordureau, A., Paulo, J. A., Rinehart, J., & Harper, J. W. (2015). The PINK1-parkin mitochondrial ubiquitylation pathway drives a program of OPTN/NDP52 recruitment and TBK1 activation to promote mitophagy. *Molecular Cell*, *60*(1), 7–20. https://doi.org/10.1016/j.molcel.2015.08.016

3. Hou, X., Chen, T. H., Koga, S., Bredenberg, J. M., Faroqi, A. H., Delenclos, M., Bu, G., Wszolek, Z. K., Carr, J. A., Ross, O. A., McLean, P. J., Murray, M. E., Dickson, D. W., Fiesel, F. C., & Springer, W. (2023). Alpha-synuclein-associated changes in pink1-prkn-mediated mitophagy are disease context dependent. *Brain Pathology*, *33*(5). https://doi.org/10.1111/bpa.13175

4. Ordureau, A., Sarraf, S. A., Duda, D. M., Heo, J.-M., Jedrychowski, M. P., Sviderskiy, V. O., Olszewski, J. L., Koerber, J. T., Xie, T., Beausoleil, S. A., Wells, J. A., Gygi, S. P., Schulman, B. A., & Harper, J. W. (2014). Quantitative proteomics reveal a feedforward mechanism for mitochondrial parkin translocation and ubiquitin chain synthesis. *Molecular Cell*, *56*(3), 360–375. https://doi.org/10.1016/j.molcel.2014.09.007

5. Watzlawik, J. O., Hou, X., Richardson, T., Lewicki, S. L., Siuda, J., Wszolek, Z. K., Cook, C. N., Petrucelli, L., DeTure, M., Dickson, D. W., Antico, O., Muqit, M. M., Fishman, J. B., Pirani, K., Kumaran, R., Polinski, N. K., Fiesel, F. C., & Springer, W. (2024). *Development and Characterization of Phospho-Ubiquitin Antibodies to Monitor PINK1-PRKN Signaling in Cells and Tissue*. https://doi.org/10.1101/2024.01.15.575715