## Parkinson's Research Antibody Sampler Kit 1 Kit (5 x 20 microliters) 3 Trask Lane | Date



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

Product Includes	Product #	Quantity	Mol. Wt	Isotype/Source	
DJ-1 (D29E5) XP <sup>®</sup> Rabbit mAb	5933	20 µl	22 kDa	Rabbit IgG	
LRRK2 (D18E12) Rabbit mAb	13046	20 µl	290 kDa	Rabbit IgG	
Parkin (Prk8) Mouse mAb	4211	20 µl	50 kDa	Mouse IgG2b	
PINK1 (D8G3) Rabbit mAb	6946	20 µl	60, 50 kDa	Rabbit IgG	
α-Synuclein (D37A6) Rabbit mAb	4179	20 µl	18 kDa	Rabbit IgG	
Anti-rabbit IgG, HRP-linked Antibody	7074	100 µl		Goat	
Anti-mouse IgG, HRP-linked Antibody	7076	100 µl		Horse	

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

Description	The Parkinson's Research Antibody Sampler Kit provides an economical means of detecting target proteins related to Parkinson's disease. The kit contains enough primary and secondary antibody to perform two western blots per primary.
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	Parkinson's disease (PD), the second most common neurodegenerative disease after Alzheimer's, is a progressive movement disorder characterized by rigidity, tremors, and postural instability. The pathological hallmark of PD is progressive loss of dopaminergic neurons in the substantia nigra of the ventral midbrain and the presence of intracellular Lewy bodies in surviving neurons of the brain stem (1). Research studies have shown that various genes and loci (α-synuclein/PARK1 and 4, parkin/PARK2, UCH-L1/PARK5, PINK1/PARK6, DJ-1/PARK7, LRRK2/PARK8, synphilin-1, and NR4A2) are genetically linked to PD (2).
	α-Synuclein, a 140 amino acid protein expressed abundantly in the brain, is a major component of aggregates found in Lewy bodies (3). Parkin is involved in protein degradation through the ubiquitin- proteasome pathway, and investigators have shown that mutations in Parkin cause early onset of PD (4). In the case of autosomal recessive juvenile Parkinsonism (AR-JP), deletions have been found on chromosome 6 in the Parkin gene (5). PTEN induced putative kinase 1 (PINK1) is a mitochondrial serine/threonine kinase involved in the normal function and integrity of mitochondria, as well as a reduction of cytochrome c release from mitochondria (6-8). PINK1 phosphorylates Parkin and promotes its translocation to mitochondria (7). Mutations of PINK1 are associated with loss of protective function, mitrochondrial dysfunction, aggregation of α-synuclein, and proteasome dysfunction (6,8). DJ-1 is involved in multiple cellular functions; it has been shown to cooperate with Ras to increase cell transformation, to regulate transcription of the androgen receptor, and may function as an indicator of oxidative stress, while loss-of-function mutations in DJ-1 (-/-) mice, resulting in reduced long-term depression (13). Leucine-rich repeat kinase 2 (LRRK2) contains amino-terminal leucine-rich repeats (LRR), a Ras-like small GTP binding protein-like (ROC) domain, an MLK protein kinase domain, and a carboxy-terminal WD40-repeat. At least 20 LRRK2 mutations have been linked to PD (14). The most prevalent mutation, G2019S, causes increased LRRK2 kinase activity, leading to progressive neurite loss and decreased neuronal survival (15).
Background References	<ol> <li>Fahn, S. (2003) Ann N Y Acad Sci 991, 1-14.</li> <li>Moore, D.J. et al. (2005) Annu Rev Neurosci 28, 57-87.</li> <li>Goldberg, M.S. and Lansbury, P.T. (2000) Nat Cell Biol 2, E115-9.</li> <li>Borrelli, E. (2005) Neuron 45, 479-81.</li> <li>Polymeropoulos, M.H. et al. (1997) Science 276, 2045-7.</li> <li>Liu, W. et al. (2009) PLoS One 4, e4597.</li> <li>Kim, Y. et al. (2005) J Biol Chem 280, 34025-32.</li> <li>Bonifati, V. et al. (2003) Science 299, 256-9.</li> </ol>

	<ol> <li>Nagakubo, D. et al. (1997) <i>Biochem Biophys Res Commun</i> 231, 509-13.</li> <li>Takahashi, K. et al. (2001) <i>J Biol Chem</i> 276, 37556-63.</li> <li>Mitsumoto, A. and Nakagawa, Y. (2001) <i>Free Radic Res</i> 35, 885-93.</li> <li>Goldberg, M.S. et al. (2005) <i>Neuron</i> 45, 489-96.</li> <li>Mata, I.F. et al. (2006) <i>Trends Neurosci</i> 29, 286-93.</li> <li>MacLeod, D. et al. (2006) <i>Neuron</i> 52, 587-93.</li> </ol>
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