

Code No. 28019

## Anti-Human LRRK2 Rabbit IgG Affinity Purify

Volume : 100 µg

- **Introduction** : Parkinson's disease is one of the most common neurodegenerative disease and characterized by progressive loss of dopaminergic neurons. Leucine-rich repeat kinase 2 (*LRRK2*) is one of the responsible gene of autosomal dominant Parkinson's disease. *LRRK2* gene codes about 280 kDa protein with 2,527 amino-acid residues and has Leucine-rich repeats, Roc (Ras of complex proteins), COR (C-terminal of Roc), MAPKKK (mitogen-activated protein kinase kinase kinase) and WD40 domains. In recent studies, it has been reported that LRRK2 protein has a kinase activity and the activity increases by a mutation of *LRRK2* gene. And the increase of kinase activity has been shown to induce neurologic toxicity. However, there are many unclear points about functions of LRRK2 protein, and its role in pathogenic mechanism of Parkinson's disease is still unknown.
- **Antigen** : Synthetic peptide of the C terminal part of Human LRRK2 (HIEVRKELAEKMRRTSVE)
- **Purification :** Purified with antigen peptide
- Form : Lyophilized product from 1% BSA in PBS containing 0.05% NaN<sub>3</sub>

## How to use : 1.0 mL deionized water will be added to the product (the conc. comes up 100 µg /mL)

- Stability : Lyophilized product, 5 years at 2 8 °C : Solution, 2 years at –20 °C
- **Application** : This antibody can be used for western blotting in concentration of  $1 3 \mu g$  /mL.
- Reference : 1. Hatano T, Kubo S, Imai S, Maeda M, Ishikawa K, Mizuno Y, Hattori N. Leucine-rich repeat kinase 2 associates with lipid rafts. Hum Mol Genet. 2007 Mar 15;16(6):678-90.
  - 2. Smith WW, Pei Z, Jiang H, Dawson VL, Dawson TM, Ross CA. Kinase activity of mutant LRRK2 mediates neuronal toxicity.Nat Neurosci. 2006 Oct;9(10):1231-3.
  - 3. Gloeckner CJ, Kinkl N, Schumacher A, Braun RJ, O'Neill E, Meitinger T, Kolch W, Prokisch H, Ueffing M. The Parkinson disease causing LRRK2 mutation I2020T is associated with increased kinase activity. Hum Mol Genet. 2006 Jan 15;15(2):223-32.